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INFLUENCE OF CLIMATE ON HEALTH. (*Williams.*)
WATER. (*Stevenson.*)
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THE DISPOSAL OF REFUSE. (*Corfield and Parkes.*)
OFFENSIVE AND NOXIOUS BUSINESSES. (*Hime.*)
SLAUGHTER-HOUSES. (*Hope.*)

CONTENTS OF THE THIRD VOLUME.

[*Nearly ready.*]

SANITARY LAW.

HYGIENE
AND
PUBLIC HEALTH

VOL. II.

A TREATISE
ON
HYGIENE AND PUBLIC HEALTH

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IN THREE VOLUMES

VOL. II.



PHILADELPHIA
P. BLAKISTON, SON, & CO.
1012 WALNUT STREET
1898

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PREFACE

TO

THE SECOND VOLUME

IN the Preface to the First volume the Editors explained the intention of the work which they had undertaken at the request of the Publishers, and indicated the subject-matter which would be discussed in its pages. In presenting the Second volume they desire to repeat that it has been thought well to allow to each author freedom of expression of his own views, even when these are not in complete harmony with those of other contributors, or of the Editors themselves. The Second volume contains some matter of the greatest interest and importance to the student and practitioner of preventive medicine, but necessarily of a more speculative and controversial character than much that is contained in the First volume. The monographs on the 'Pathology and Etiology of Infectious Disease,' on the 'Natural History of Infectious Disease,' and on the 'Disposal of the Dead' afford instances of these observations, which will be apparent to the reader. It would have been improper to exclude such matter, and unfair to the authors to have attempted to reconcile all the statements and views advanced. The names of the contributors are sufficient guarantee of the value of their opinions, and that nothing is printed in these volumes which is not accepted by at least a large number of competent authorities.

As in the First volume, it has not been thought well to prevent some amount of repetition where this has been considered necessary to preserve the completeness of any article. Thus in the monograph on the 'Duties of the Medical Officer of Health' it has been advisable to make frequent reference to the statutory powers under which this officer proceeds; but a fuller discussion of the Law relating to the Public Health will be found in the Third volume.

CONTENTS

OF

THE SECOND VOLUME

THE PATHOLOGY AND ETIOLOGY OF IN- FECTIOUS DISEASES	PAGE <i>E. Klein</i> 1
THE NATURAL HISTORY OF INFECTIOUS DISEASES	<i>T. W. Thompson</i> 241
SMALL-POX AND VACCINATION	<i>John C. McVail</i> 388
VITAL STATISTICS	<i>Arthur Ransome</i> 465
MARINE HYGIENE	<i>Henry E. Armstrong</i> 511
MILITARY HYGIENE	<i>J. Lane Notter</i> 599
DISPOSAL OF THE DEAD	{ <i>Sir T. Spencer Wells and</i> <i>F. W. Lowndes</i> 671
MEDICAL OFFICER OF HEALTH	<i>Alfred Ashby</i> 781
INDEX	825

PLATES IN VOL. II.

PLATE

- I. TO XLII. ILLUSTRATING ARTICLE ON THE PATHOLOGY
AND ETIOLOGY OF INFECTIOUS DISEASES . *To follow p. 240***
- XLIII. MAP SHOWING THE AVERAGE DIPHTHERIA
DEATH-RATES IN ENGLISH AND WELSH
COUNTIES DURING THE PERIOD 1855 TO 1880 *To face p. 296***
- XLIV. CHART SHOWING THE ANNUAL DEATH-RATE
IN ENGLAND AND WALES OF ERYSIPELAS,
SCARLATINA, AND CERTAIN OTHER DISEASES
FROM 1855 TO 1880 " 356**
- XLV. DIAGRAM SHOWING ANNUAL DEATHS IN
LONDON FROM SMALL-POX, MEASLES, AND
WHOOPIING-COUGH PER 1,000 DEATHS FROM
ALL CAUSES; SMALL-POX AND MEASLES
FROM 1680 TO 1890, WHOOPING-COUGH FROM
1740 TO 1890 " 433**

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THE PATHOLOGY AND ETIOLOGY
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INTRODUCTION

FEW branches of medical science can be said to have received, within the last twenty-five years, greater attention, and to have developed more largely within a comparatively short period, than the knowledge of the nature of infectious diseases. Not only have many disorders, belonging to this class and for a long time suspected as belonging to this group, become well understood in their nature, their mode of origin and spread, but others, not previously suspected to be communicable diseases at all, have been shown to appertain to this class. To quote for each of these one instance: typhoid fever, though for a long time assumed to be a communicable disorder spreading from the sick to the healthy by the evacuations, had not, prior to the researches of Budd, Snow, Simon, Buchanan and others, been proved to be of this character, and though some authorities, like Murchison, pleaded for the possibility of a spontaneous origin, the weight of evidence was in favour of its being a specific disease, which, like all specific diseases, can only arise from a previous specific disorder of the same kind. By the researches of more modern investigators, notably Buchanan, Ballard, and Thorne Thorne, its mode of spread has become well understood, and by their investigations its nature—viz. that of a specific communicable disease—been definitely settled. As examples of diseases not previously suspected to belong to this group but now known to be so, we may quote tetanus, and epidemic or croupous pneumonia. Of both these diseases recent research has shown that they are of an infectious character, that in both there exists a specific virus which, finding entrance into a healthy individual, is capable of setting up the disorder. These examples we could easily multiply. Nay more, we could show, on the one hand, that as regards a number of disorders for whose specific nature there existed until quite recently very slender evidence, there are now definite and exact experimental grounds for asserting their specific nature, e.g. anthrax, tuberculosis, septicæmia, and erysipelas; on the other hand, by ascertaining the exact nature of some of these disorders, others formerly not distinguished from them have been shown to have a separate existence and to be different from them in their pathology, symptoms, course, and causation. Thus, for instance, malignant anthrax has been shown to be a perfectly separate disorder from symptomatic anthrax; woolsorters' disease (malignant pustule) to be a separate disease from some forms of ragsorters' disease; tuberculosis to be different from other chronic lung diseases, to which it bears a distant pathological semblance.

These results have been achieved by the isolation and study of the essence of these diseases—the *contagium*, which was shown to be a living microphyte,¹ *contagium vivum*, differing as regards morphology and biology in the different diseases to which it gives rise. It is no doubt an interesting study to trace the history and the development of the theory of the *contagium vivum*; but at the present day such a study is chiefly of an historical interest, and not an

¹ μικρός, small, φυτόν, plant.

integral part of the present work. It is interesting to go back to the time when the basis of a discussion as to whether infectious diseases can arise *de novo* was evidence such as the existence of epidemics arising from a case imported from outside (small-pox, the plague, cholera, &c.); or evidence that the isolation of the patient, and destruction of matter derived from such a patient, puts a stop to, or prevents the spread of that disorder; or when the similarity of the specific nature and spread of infectious diseases to the specific nature of fermentations, such as those specially studied and revealed by the earlier labours of M. Pasteur, was insisted on. At present the evidence that infectious diseases owe their origin to specific well-characterised micro-organisms, which, having entered, in however small a number, a susceptible human or animal body, multiply therein, and set up the whole chain of symptoms characterising the particular disease; and further, that the diseased tissues of such an infected individual harbour the specific microbes, so that a minute particle of such tissue is again capable of starting the disorder in a new body;—all this is no longer a matter of assumption and indirect evidence, but one of exact science. Just as a chemist is enabled to detect and to demonstrate with tolerable exactness the existence in special cases of a chemical poison—e.g. arsenic, phosphorus, strychnine, &c.—the material that has caused disease and death, so nowadays can the pathologist by microscopical, cultural, and animal experiments demonstrate the specific *materies morbi*; more than that, in cases where the physician, or surgeon and sanitarian, is unable by the clinical and other aspects of a case to give an opinion, the pathologist can by his methods state with exactness the nature of the disease in question. In illustration of this we may mention the case of tuberculosis, of anthrax, of cholera, and of hydrophobia. Numerous cases occur in the human subject where from a clinical point of view the physician does not feel justified in pronouncing whether or not the case is one of tuberculosis; the pathologist, by demonstrating the presence or absence of the tubercle bacilli, is enabled to settle the point without difficulty. The same holds good in certain surgical cases, where the diagnosis of tuberculosis of the skin or bones is only possible after the demonstration of the tubercle bacilli. In certain diseases affecting sheep and cattle, and wool and hide sorters, the exact and reliable diagnosis of malignant anthrax is furnished by the demonstration of the bacillus anthracis by the microscope, by culture, and by experiment on animals. In suspected cases of cholera, when the clinical aspect is not sufficiently characteristic, the demonstration of the choleraic comma bacilli in the evacuations, or in the contents of the intestines, furnishes an important piece of evidence. In cases of suspected rabies, the experimental method discovered by M. Pasteur—viz. that the spinal marrow of such an animal inoculated under the dura mater of a rabbit produces hydrophobia—furnishes positive proof. The differential diagnosis between tuberculosis and actinomycosis in cattle, possible by the microscopic test, furnishes another not less important illustration. The study of micro-organisms connected with specific diseases—i.e. the *contagia*, their nature, habits, and mode of growth, outside and within an infected individual, the manner in which they leave and in which they enter the human and animal organism, the nature of the changes which they induce in the material in which they grow and multiply, be that material the human or animal organism, or organic matter outside these—all this is now well understood.

The micro-organisms or microbes¹ that interest us belong to three distinct groups: (1) bacteria, (2) fungi, (8) monadinae. The first are those which as

¹ Microbe (*μικρός*, small, *βίος*, life) is the term also generally applied to these microscopic organisms.

regards infectious diseases hold the most prominent place, owing to their general and very wide distribution, and because most of the infectious disorders hitherto analysed owe their origin to one or another species of these microphytes; the second or fungi have, so far as research has hitherto gone, a more limited distribution as disease-producers; and the third group, or monadinæ and allied organisms, belonging to the animal kingdom, are at present only imperfectly investigated, although, as will be shown, they produce some well-marked disorders in man and animals.

SECTION A.

CHAPTER I

CHARACTERS OF BACTERIA

BACTERIA are microscopic organisms which contain no chlorophyll, which possess an investment of cellulose, and are therefore considered to belong to the vegetable kingdom, which multiply by simple division or fission, and are therefore called, after Naegeli, Schizomycetes (σχίζω, divide, and *μύκης*, fungus). Though they are considered as single-celled plants, their action is not quite in harmony with that generally attributed to plants, since, speaking in a general way, they do not possess in any conspicuous degree, like vegetable cells, the power of building up complex organic substances from simple inorganic material; but, on the contrary, on the plan of animal cells, they have in an eminent degree the character of destroying or breaking down higher or complex organic molecules into those of simpler combinations. This is true in a general way, although, just as is the case with animal cells, exceptions are not wanting, i.e. instances when by synthetic processes the reverse takes place.

GENERAL MORPHOLOGY

We will first consider the general morphological characters of bacteria. Bacteria are single cells of extremely minute size, 0.1 to $1\mu^1$ or more, consisting of a protoplasmic body—mycoprotein of Nencki, and an investing sheath of cellulose. The protoplasm while active and living is homogeneous, when dead often appears granular; it has great affinity for neutral and basic aniline dyes, and in this respect compares with the substance of nuclei in vegetable and animal cells. There is no evidence of the presence in their protoplasm of any differentiation comparable to the nucleus in the animal or vegetable cell, although in some individual cases such a structure has been mentioned, but this, owing to the methods of complex staining used for its demonstration, is not free from objection; in the fresh state, and after the usual methods of staining, no differentiation of a nucleus can be recognised in the active and living cells constituting the elements of any bacterial species. The protoplasm does, however, show in certain conditions and in certain species differentiation of denser and less dense parts, causing a segregation of the protoplasm into granules or particles; this occurs in conditions generally taken as indicating degeneration, but in several instances, to be mentioned later, is a character belonging to the life of the bacteria. Similarly, the appearance of fluid droplets in the protoplasm—vacuoles—can be noticed in certain

¹ 1μ = one micromillimetre = one twenty-five thousandth of an inch.

conditions. In some organisms granules of a special chemical nature have been noticed in the protoplasm (Cohn). The process of vacuolation is constantly noticed in conditions of fairly rapid multiplication; the bacterial element elongates more or less, generally in the middle of its protoplasm a vacuole appears, and hereby the protoplasm segregates into two polar masses. Such forms are very numerous met with in all species of bacilli, and under the most varied conditions of growth and multiplication; often these vacuoles have been, and still are, occasionally mistaken for spores (*see below*). The cellulose sheath invests closely the protoplasm; when the cells degenerate the protoplasm breaks down and disappears, and the sheath remains empty; during this process the sheath as a whole swells up into a jelly, faintly granular, and gradually loses altogether its outlines. In many species of bacteria during rapid multiplication the sheath gives rise to a gelatinous secretion surrounding the individuals, in fact forming a kind of general matrix—hyaline, viscous, and more or less faintly granular—for the new brood, and by this matrix a larger or smaller number of the new individuals remain aggregated together: it is not quite clear whether this matrix is derived from a gelatinous metamorphosis of the old sheaths, or whether it is a secretion or new formation by the new elements. When, in an individual, the protoplasm divides into two masses, each of the new offsprings forms its own investment, so that the old sheath, swelling up into a gelatinous mass, persists as a sort of binding matrix; in all those species in which, after continued division, new elements remain aggregated together as groups (*zooglæa*, *staphylococcus*), the gelatinous matrix has this origin (Cohn). In other cases the bacteria become invested outside the sheath by a gelatinous secretion, which as a narrower or broader hyaline-looking zone invests the elements like a capsule, e.g. *ascococcus Billrothi*, *bacillus cyanogenus*, *bacillus pneumoniæ* of Friedländer, and other capsulated cocci and bacilli of the same tribe. In these instances of 'capsulated' bacteria the capsules are probably special secretions and not the old swollen-up sheaths, since these capsules are wanting under certain different conditions of soil, while they are present and well marked in other conditions, the rapidity of multiplication having no influence whatever on the presence or absence of these capsules. These hyaline capsules around the sheath must not be confounded with the swollen-up gelatinous sheath of bacteria in certain phases of their life, notably in the degenerative phases.

The affinity of the protoplasm towards certain aniline dyes is greater than that of the sheath, and that of the latter greater than of the matrix and capsules above mentioned, so that on staining with an aniline dye, the protoplasm takes up the dye quicker and more intensely than the sheath or the matrix; and on withdrawing the dye, by washing in alcohol, or water, acids, or alkalies or other special methods (*see below*), the protoplasm retains it longest, the capsules and matrix least. For this reason it will be found that by either of these ways it is possible to bring out these three different structures as different tints of the same dye, and further, that, owing to the rapidity with which the matrix and capsules can be deprived of the dye, a second dye can be applied, to be taken up by them while the protoplasm and sheath have still retained the first dye. By these means various double, and even treble, stainings can be effected by successive applications of different dyes. Acids and alkalies, chloroform, and alcohol, though affecting the fresh protoplasm, have no visible effect on the sheath, and bacteria acted upon by these reagents remain, therefore, unaltered both as to shape and aspect; and these chemical reactions are often resorted to for the sake of differentiating bacteria—notably the spherical species—from similar forms of organic matter

not of bacterial nature. This is particularly important in the examination of animal tissues in the fresh state, in which tissue granules and rod-like forms, fat granules, &c., may easily give semblance to the presence of bacteria, but the test of acids and alkalies soon brings clearness into the matter. Other tests to be presently described, such as staining and cultivation, are of importance, and add certainty to the diagnosis.

SIZE OF BACTERIA

The different species of bacteria differ one from another very considerably in size. Although all are of microscopical size, yet, comparing the different families and the different species in one family, sufficient differences will be detected to enable us to consider them as characteristic. The smallest bacteria are some of those species which, owing to their spherical form, are called cocci or micrococci.¹ But also amongst the micrococci some are of considerable diameter, 1μ or even more; in the majority the individuals are considerably less, 0.5 , 0.4 , and even as small as 0.1μ . But, as will be presently mentioned of other species, so also amongst the cocci of the same species, there occur the greatest differences in size under various conditions and in the various phases of growth. In an active state of growth, differences amounting to two and three and more times in size are noticed amongst the individuals of the same species in almost all media, sometimes less in fluid than in solid media; for though the great majority of the individuals have the same size, there are always some that are much larger. The writer has specimens of various species of micrococci, notably those capable of forming chains or chaplets when growing in fluid media (streptococci), in which some of the cocci are several times the diameter of the smallest individuals (see fig. 1). These large cocci can be shown to divide into medium-sized cocci, and these again into the small normal individuals.

Those forms of bacteria which, owing to their being more or less rod-shaped, are known by the name of bacilli show a diameter greater in length than in thickness. And in the size of both the thickness and length, different species vary very considerably. But it is chiefly the diameter in thickness which is for diagnostic purposes of greater importance than that in length. It must be understood that where in a given species, growing in a particular medium, the great majority of the individuals possess a certain length, this will be taken as the typical length, and is quite sufficient to serve for diagnostic purposes; but in all species of bacilli, according to the phase of growth of the individuals, the length of the rods is subject to very great variations. In characterising the length of these individuals, it is therefore necessary to give the length of the shortest and the longest individuals. But whatever the length of the different individuals, their thickness is almost uniform, and therefore this would be of greater importance than the length. As a matter of fact, in not a few species of bacteria, with which we shall become acquainted in the course of this work, the individuals often vary between slight ovals of 0.5μ in length, and long cylinders of 5μ or more, and it would therefore not be quite correct to say that the average length of the cylindrical cells is about 3 to 4μ , though there may be in a particular species numerous individuals of that length; but notwithstanding these gross differences in the length of the cells, the thickness is uniform, though a few noteworthy exceptions are also here noticeable, as will be mentioned later on.

¹ The name 'micrococci' was first applied by Hallier to the granules contained in some of his 'spores,' which granules becoming free after bursting of the 'spores' germinate into various forms of microphytes. *Kókos* means a kernel.

Another important point, apart from the variations due to phase of growth, is the influence on size of the medium in which the bacteria are growing. This is in many instances considerable. One of the best cases is that of the bacillus anthracis, though many other species (micrococci and bacilli) behave in a similar manner. Bacilli of anthrax taken from the blood of an animal dead from virulent anthrax are a little more than half the thickness of the bacilli grown in neutral broth or neutral gelatine; the majority of the rods of the former are in suitably prepared specimens made up of short, almost cubical cells, while of the latter the majority of the rods are distinctly cylindrical. But also the bacilli of anthrax derived from different species of animals dead of anthrax show differences in thickness and length. The same can be said of other bacilli (bacillus of typhoid fever, of swine fever, of tubercle, cholera, and others), both as regards length and thickness when growing in different media.

From all these considerations it follows that by naming a certain size as characteristic of the individuals of a species it is necessary to name also the source from, or the medium in which, they have been growing, and the phase of growth or length of time they have been growing, and, further, in the case of bacilli, to give the length of the shortest as well as the longest single individuals, or when the great majority are of a uniform size to state this as the typical length.

SHAPE OF BACTERIA

The first scientific classification of bacteria based on difference in shape was given by F. Cohn, and has been and still is, regarded as the most practical one, although within recent years it has undergone some not unimportant modifications. Cohn divided bacteria according to shape into: (a) spherical or micrococci; (b) oval or bacterium proper, length only twice the thickness; (c) cylindrical or bacilli, length more than twice the thickness; (d) curved or vibrio, and (e) spiral or spirilla, to which may be added the folded and long spirals or spirochetæ. Recent research has shown that the second division—i.e. the oval species of bacterium proper—cannot stand as a separate species or rather group of species, since more accurate observation has shown that also amongst these some of the individuals attain the cylindrical shape. This group is therefore merged into the bacilli. Likewise, it has been shown that group (d) cannot be separated from group (e), since the individuals of the species of group (d) in continued growth attain the shape of spirals, and *vice versa*, that some of the spirillar forms are made up of individuals comparable to those of group (d). And lastly, the spirochetæ cannot stand as a separate group and must be merged with those of the spirilla. So that at present, according to shape, only three groups can be correctly distinguished, viz. (1) cocci; (2) bacilli; and (3) spirilla; the first comprises those species in which no matter what the phase or the medium is, the individuals are spherical or nearly spherical; to the second group belong all those species in which the individuals may attain the rod or cylindrical shape; and in the third the individuals are curved and may form spirals.

1. *The Cocci*.—A species in which the individuals are never cylindrical or rod-shaped cells, but are always more or less spherical, is called a micrococcus. But there are considerable differences amongst these and also amongst the individuals of the same species; for in almost all these species there are phases of growth, in which the majority of individuals are not exactly spherical, but are either crescentic or dumb-bell-shaped—diplococci.¹

¹ διπλοῦς, double.

Both these represent a phase in the division of the individuals; and here again the daughter cells may be in immediate contact or may be already well separated one from another, according to whether they are in the act of dividing or already divided; in the latter case, as much matrix will be found separating the new offsprings as in those that have completely divided: in this last case the individuals have the same spherical shape. In fig. 2, numerous dumb-bell forms are shown, in which the cocci are spherical; in fig. 1, dumb-bells are seen in which the cocci are crescentic; and in fig. 5, single cocci are shown, all of uniform spherical form.

Careful examination has proved that some species which in former years were considered as belonging to the group of micrococci are really bacilli, since under certain conditions of growth and, in certain phases, there are oval and even cylindrical individuals to be found amongst them, e.g. the pink organism, formerly spoken of as *Micrococcus prodigiosus*, has been shown to be really a bacillus; the organism, held by Friedländer and others to be one of the organism of croupous pneumonia, and called the pneumococcus, is really a bacillus, called now the bacillus of Friedländer, and many other species which at first sight appear in certain preparations to be cocci, and cocci only, but under other conditions of growth are shown to be bacilli; e.g. the organism which is shown in fig. 8 and which is the organism of grouse disease. This microbe appears in the grouse as cocci and diplococci, many spherical, some also oval, few rod-shaped; but numerous cylindrical forms exist in certain artificial cultures (broth) and in guinea-pigs and mice, inoculated with them.

A special form of cocci is represented by *sarcina*. Under this name are understood those species of micrococci which, owing to a rapid and repeated subdivision of the cocci in vertical lines, form groups of four cocci and multiples thereof. When a coccus divides into a dumb-bell or diplococcus, and each of these again divides in a direction vertical to the previous one, a group of 4 cocci is formed—*Micrococcus tetragonus* (fig. 4). In these groups each two cocci are crescentic with one more or less straightened side, or they are even square cut; by further division of each coccus, the group increases from 4 to 8, 16, 32, and more; but in these the cocci are always more or less subgrouped in fours—*Sarcina lutea* (fig. 3). Now, some species of cocci show under certain conditions of growth this tetragonal arrangement or grouping in fours in a conspicuous manner, others show it only imperfectly; but even those that show it conspicuously under certain conditions show it less or in a not at all pronounced degree under other conditions of growth.

When owing to repeated division the cocci remain aggregated in larger or smaller groups, which themselves gradually extend as reproduction proceeds, and are connected by streaks of cocci in a more or less irregular fashion, such cocci are called staphylococci (*staphylis*, a bunch of grapes), to distinguish them from a species of cocci which, after division, remain joined in a linear series. By this latter method of division chaplets or chains of cocci are formed which, according to the number of subdivisions, are shorter or longer; such a chain-forming coccus is called streptococcus (*streptos*, bent, twisted), because generally these chains, particularly when long, are more or less wavy and twisted. In these chains the cocci are often arranged as dumb-bells, this indicating the actual phase of division or one immediately after; in others, the cocci are of equal distance, indicating the phase before division of the individual cocci has commenced. Always, however, one meets with individual cocci in chains which are considerably larger than the rest; these are found either in the chain or, more

commonly, at one end; in the latter case they are sometimes club- or pear-shaped. Whether these larger cells represent a particularly active element or one that has undergone involution, is not easily to be made out. From what will be stated below of similar forms of bacilli, the writer is inclined to think that they are not involution forms, but indicate an altogether different phase, viz. atavism or a reversion to an antecedent form in the history of the evolution of the cocci.

2. *The Bacilli*.—All bacilli are rod-shaped or cylindrical cells, which differ one from another in the length and thickness of the cells. In the same species, as mentioned above, there occur the greatest differences as regards the length of the rods. While in a good many species, to be hereafter more minutely described, even the shortest or youngest elements are distinctly cylindrical, there are numerous species in which the youngest elements do not differ from spherical or crescentic cocci; these are then chiefly arranged as diplococci and represent a rapid mode of division of young elements; but there are present already in early stages elements which are distinctly cylindrical, and, as growth proceeds, these cylindrical elements increase in number and in length. We could name here a long series of species of pathogenic bacilli, which, when taken from the blood of an animal, or growing on gelatine or agar mixture, show in the earlier stages the majority of the elements as oval rods; some, like dumb-bells, constricted in the middle; but in broth cultures there are also noticeable numerous cylindrical elements in the early stages. After a few days' growth on solid media (gelatine, agar) numerous cylindrical elements are met with. Thus, the microbes known as connected with, and causing rabbits' and pigeons' septicæmia of Davaine and Koch, the organism of swine fever and of Wildseuche, of infectious fowl enteritis, of guinea-pigs' septicæmia, of fowl cholera, of the Middlesbrough (human) pneumonia, of grouse disease, the bacillus of Friedländer and several species allied to this, the bacillus pyocyaneus, the bacillus of veal and pork pie, and others when taken from the blood or tissues, or from early cultures on solid media (gelatine or agar mixture)—all these are like diplococci or slightly oval organisms; when examined, after a few days' growth on these media, they are in numerous individuals cylindrical, some four or five times longer than they are thick.

All these species taken from early cultures on gelatine or agar mixture, or from the blood and tissues, would in former years have been regarded as belonging to Cohn's group of bacteria proper. But also in species, about whose bacillary nature there never was any question, some individuals occur in all cultivations which do not differ from cocci and diplococci, being spherical or slightly oval cells, singly, or more commonly as dumb-bells; such is the case with the tubercle bacillus, with the bacillus of diphtheria, and with a variety of species of non-pathogenic bacilli. But here the question whether or not they are bacilli is at once answered by the occurrence in the same preparations of numerous, or even the majority of, cylindrical elements.

Now, the opinion has been repeatedly expressed by various observers (Naegeli, Lankester, Zopf, and others) that there exists no well-marked boundary between the different forms separated by Cohn as micrococci, as bacilli, and as spirilla, on account of the transitional forms between each of the two groups. As we have shown above, numerous species of bacilli exist, in each of which cocci, cylindrical forms and intermediate forms, do occur; but does this justify us in saying that between the *species* of micrococci and those of bacilli, intermediate species occur, and that there is no definite boundary between them? Clearly not. In those species that are micrococci,

the elements, under every condition of growth that has been tried, always appear as cocci, either singly or as diplococci, as staphylococci or as streptococci, as the case may be, and never as cylindrical rods: these are clearly and decidedly species of micrococci. In some of the species of bacilli, on the other hand, the youngest forms may not be larger than a spherical or cubical mass of protoplasm, may therefore not differ morphologically from a coccus, but have the power to elongate into rod- or cylindrical-shaped cells; in other species, again, even the youngest elements are as a rule, cylindrical, although also here amongst these well-pronounced bacillary forms under certain conditions some elements appear almost spherical or cubical, e.g. bacillus anthracis, bacillus of tubercle, bacillus of diphtheria, as will be shown later on; and the same holds good also for some of the species which are spirilla, for, as we shall presently show, the youngest forms also of these appear as mere spherical or oval masses of protoplasm, capable of elongating into more or less curved rods and cylinders, and these by continued growth elongate into wavy or spirally twisted chains or threads. But because in a certain state they are like cocci, we shall not put them down as micrococci, and because between the cocci forms and the spirillar forms intermediate shapes occur, we shall not consider them as indicating a change of species, as if micrococci were to change into spirilla; but since in their adult phase they grow into spirilla, we shall call such species spirilla.

An organism has been minutely described by Zopf as *Cladothrix dichotoma*: in this organism there is no doubt that cocci, straight rods, isolated and in chains, and shorter or longer spirilla do occur, but it is not at all permissible to consider this as evidence that between the three groups of bacteria, viz. micrococci, bacilli, and spirilla, there is no definite boundary line. The cocci-form of the *Cladothrix dichotoma* when sown on the different media will again give rise to *Cladothrix dichotoma*, and will never become or grow as micrococci; and if the bacillary forms of this microbe are watched in their growth, it will be seen that some of them again give rise to spirilla. This same phenomenon can be easily ascertained in some of the typical spirillar species, e.g. the spirillum of Finkler, of Koch, the spirillum of noma. In all cultures, after a few days' growth, forms show themselves which do not differ from cylindrical bacilli, in which a curve of the rods is very slightly or not at all indicated; they appear as single rods, as dumb-bell rods, or as slightly wavy chains or threads, yet on further growth they are capable of becoming curved and assuming the spirillar form.

We conclude then that if a certain species under all conditions of growth is and remains spherical or nearly so, we have to deal with a species of micrococcus; if, on the other hand, a certain microbe grows into cylindrical elements more or less straight, it is a species of bacillus; and lastly, if it grows into curved rods and more or less spirillar forms, it is a species of spirillum.

Many species of bacillus form chains by linear arrangement, that is, the offsprings of one element after division remain attached one to another end to end; these chains may be composed of few or many rods, according to the number of subdivisions that have taken place; and in this way short or long threads may be formed. These are spoken of as leptothrix. Some species of bacillus have this power of growing out into threads in a very high degree; these species are very numerous, and their type is represented by the bacillus subtilis of hay infusion, and by the bacillus anthracis. The threads are either more or less straight or wavy and often like coils, the individual threads being more or less twisted one round another. When

viewed in the fresh state they appear smooth and uniform, but after staining, they, like the shorter and shortest chains, are seen to be really only linear series of rods or cylinders. In such chains the individual cells are more or less of uniform length and thickness; but there are not wanting such chains and threads, in which successive or distinct elements differ considerably in length and also occasionally in thickness. Fig. 14 shows threads composed of rods of equal length and thickness; fig. 19 shows threads of the same species, in which the elements show a difference both in the length and thickness. Particularly the difference in the length of the rods is instructive, since it shows that this is merely a phase of development; the most recent divisions or the youngest forms being shorter, cubical, or even like cocci, the older forms more cylindrical. This is also very well pronounced in tubercle bacilli, as will be illustrated below. Occasionally in a chain the rod-shaped elements follow one another as dumb-bells, evidently indicating a phase of simultaneous division of the cylindrical elements of a chain.

When bacilli are stained with aniline dyes and then sufficiently washed, they all show certain well-marked appearances: (1) the sheath, (2) the protoplasmic contents. The sheath appears as a thin faintly stained investment. When bacilli are arranged in dumb-bells or double-rods, or in shorter or longer chains and filaments, there is always a thin septum passing between two serial rods; the whole chain or thread appears invested by a common continuous sheath from which pass transverse septa which separate the individuals crossways; it is also noticed that the protoplasm of the individual rods is as a rule in much closer contact with the sheath than with the transverse septa. The protoplasm is either uniformly stained, or, as is not uncommon, it shows at the ends of each rod much deeper staining than in the middle, that is to say, there is denser protoplasm at the ends of the rods than in the middle. In the short individuals this often gives a very characteristic appearance, inasmuch as each rod appears made up of three parts of equal size: two terminal stained granules and a middle clear unstained part. As just stated, this is not peculiar to any one species, but can be noticed in all species; it is particularly conspicuous in those in which the young elements are short, e.g. fowl cholera, fowl enteritis, septicæmia of rabbit, swine fever, &c., &c. (fig. 7). But also amongst the longer, i.e. cylindrical elements, the middle part of the rod very often appears unstained and clear, while the protoplasm at the end is denser and stained; the middle clear part is at the same time more or less well marked off with rounded outline, spherical or oval in shape, and represents a vacuole; occasionally the stained protoplasm is central while the unstained parts, the vacuoles, are terminal. Such vacuoles are very common in all species of bacilli; they (vacuoles) are, however, more frequently met with under conditions which imply want of sufficient nutritive material, as for instance when bacilli grow on solid media (gelatine, agar mixture, potato), and when, owing to the continued growth into the depth of the medium, the first formed or superficial layer becomes gradually removed from the nutritive material; in this superficial layer the vacuoles in the rods are very conspicuous; in preparations made of thread-forming bacilli under the above conditions of growth these appearances, i.e. the presence of vacuoles regularly disposed in the individual rods, are very striking (fig. 17).

But, as before stated, the presence of vacuoles in the rods is also found under other than the above conditions, in some species more numerous than in others, and more often where rapid growth takes place than where this is not the case. This vacuolation is not indicative of any degenerative change—any more than in the mycelial threads of fungi where it is well known

and typical—but seems, in some cases at any rate, to be due to the medium in which the bacilli grow containing comparatively less nutritive material: not only in bacilli, but also in the individuals composing a spirillum are these vacuoles to be observed. In cylindrical bacilli these vacuoles may be, and sometimes have been, mistaken for spores.

The ends of bacilli are generally rounded, occasionally straight, and less frequently more or less pointed or conical at one or both ends. In *Bacillus anthracis* the ends are generally more or less straight; in the *Bacillus* of diphtheria grown on gelatine many bacilli show one end pointed, the other rounded or straight and thick.

In many species one or both ends of the rods, or the free end of the rods forming the terminals in a chain, are swollen and thick, spherical, round, pear-shaped, or club-shaped; occasionally there are some elements, in the middle of a shorter or longer chain, swollen, spherical, or oval. Such forms are considered as involution forms, but there are good grounds for doubting this, and the reasons will be stated later on in connection with the evolution of bacteria. When in a chain of rods, i.e. in a thread, the individual rods become so changed, an organism results which is totally unlike the typical thin smooth thread, but appears more like a varicose thread in which the individuals are torula-like, spherical, or spindle-shaped cells, connected by thin bridges, the cells being three or more times as thick as the typical rods (fig. 70). In connection with this and the former appearance, another appearance deserves notice, viz. the segregation of the protoplasm in a chain or in individual rods as separate spherical or oval granules, whereby the rods and chains become transformed into varicose rods or fibres; in these the granules take and retain the dye easily, whereas the bridges between them are less stained; in the tubercle bacilli, leprosy bacilli, diphtheria bacilli, and others this appearance is sometimes very regular and characteristic (fig. 78).

3. *The Spirilla*.—The individuals of spirilla are rod-shaped or cylindrical, but more or less curved, representing the arc of a smaller or larger circle; they are also called comma-bacilli,¹ their ends are rounded, or not unfrequently slightly pointed. As regards the amount of curvature there exist the greatest varieties, not only amongst the different species, but also within the same species. Thus, for instance, in the cholera spirilla, in Finkler's spirilla, and in spirilla which were found in the intestinal mucus of apes affected with diarrhoea, one sees some elements of the shape of small semi-circles, while many others only just show an indication of a curvature. But the same species grown on different media shows remarkable differences as regards the curvature of the ends. When growing for several days the comma-bacilli form longer or shorter chains, which are wavy or more or less spiral; this depends obviously on the curvature of the individuals from which such a chain is derived. When growing in fluids, some species of comma-bacilli form well-pronounced uniform spirals; also in solids, some chains are more distinctly spiral than others, some being only slightly wavy, while others are uniformly spirally twisted—sometimes the turns of the spirals are very close, sometimes more open. The spirals are in some cases uniform, e.g. in relapsing fever, and it is difficult to show that they are made up of curved rod-like elements, while in others (cholera) many of the spirals are distinctly jointed. There exist very considerable differences in the thickness

¹ The name comma-bacilli was first applied by Koch to the cholera spirilla, owing to their likeness in shape to a German comma, which is simply a slightly curved rod; the word cholera bacilli for these microbes is reprehensible, because they are spirilla and not bacilli.

of the spirals in the different species, and in the thicker species the jointed character of the spirals is more easily demonstrated than in the thinner species. The individual comma-bacilli in stained and well-washed specimens show the same distinction into sheath and protoplasm as was mentioned of the bacilli, and also the presence of a vacuole in the middle of the individual comma-bacilli and the terminal easily stained collections of protoplasm. Though in some species of bacilli, e.g. bacillus of glanders, bacillus of diphtheria, there exist rods which are more or less curved, they do not form spirals, and their curved character is not permanent; but in the true spirilla, however slight the curvature of some elements—and in some species and under some media the curvature of some of the elements is very slight indeed—they nevertheless are capable of forming spirals. Thus the writer has seen even anthrax bacilli growing on alkaline gelatine assuming a very markedly curved shape, while Finkler's spirilla, or those found in noma and in Asiatic cholera, appear in some media only to show the very slightest curve; but from subcultures of the above anthrax bacilli in broth or gelatine the typical straight anthrax bacilli result, while of the above spirilla subcultures made in broth, in gelatine, &c., the typical spirilla will be the result. This shows that the first, though they may occasionally become curved rods, are not spirilla but bacilli, and the latter, though the individuals may occasionally appear almost straight, are not bacilli but spirilla.

MOTILITY

One of the most interesting phenomena shown by bacilli is the power of active locomotion possessed by some species. When examined under the microscope in a fluid medium all bacteria show the kind of oscillation known as Brownian molecular movement; but in some species there is an active locomotion, by which the individual bacteria are enabled to move actively and to change their place; this movement shows itself either by the bacteria darting with great rapidity across the field of the microscope in one or another direction, or spinning round with greater or lesser velocity, or briskly moving like a screw in one direction and then back again. Observing a single straight bacillus in its movement, either a darting or spinning movement in one direction is noticed; when two such bacilli are connected endwise, but bent one to another under an angle, then often, with a forward or backward movement of the one, a spinning movement of the other is noticed, the former not really actively moving but being simply propelled by the spinning movement of the latter bent under an angle. When comma-bacilli or spirilla move, the motion is always more or less spiral.

When longer chains or leptothrix of bacilli move, the movement is always more or less serpentine. The locomotion of bacilli is either rapid or slow; the latter may be a character of the species, that is to say, the individuals as a rule show only a relatively slow movement, e.g. typhoid bacilli generally move comparatively slowly, and the longer bacilli move in a serpentine manner. The mobile individuals do not continue to move indefinitely, since often an individual which has been spinning round or darting about gradually comes to rest and remains so for some time; besides this, all motile bacilli during the phase of division are at rest, and when they form groups, i.e. when they are in an active state of division, they do not move. But of such groups here and there an individual may be seen to separate itself from the margin and to move briskly away; on breaking up a group, crowds of motile bacilli sally forth. The writer has watched single bacilli of the human Middlesbrough pneumonia spinning round with great velocity without much

changing their place. One and the same bacillus was noticed to spin round for five minutes without any diminution in its velocity; then this gradually lessened, and ultimately, after further five minutes, the organism came to rest. When a drop of broth was added, the spinning-round commenced again with great vigour. Some mobile bacilli show motility under a certain condition and not under others; others again show it under all conditions. Thus, for instance, many individuals of the bacillus of the Middlesbrough pneumonia show active locomotion in specimens made of gelatine and agar cultures; made of broth cultures the motility of many individuals is observable only while the broth cultures are of recent date—24–48 hours old; later on only very few motile individuals are met with. The loss of motility may be and sometimes is due to chemical by-products in the cultivation (*see* bacillus of grouse disease and of pneumonia). Some species of motile bacilli when growing on a solid medium are capable by their locomotion of distributing themselves from a given point rapidly over and through the medium, e.g. certain species of bacilli known as proteus of Hauser, certain species of the potato bacilli, &c.; this phenomenon is spoken of as 'swarming'—thus, when a colony of such bacilli appears on gelatine, agar mixture, or potato, irregular streaks and lines and patches of the growth are soon seen extending in different directions, this being due to the swarming of the bacilli from the first colony and the establishment of new colonies by the former. There exist great differences in this respect between different species of motile bacteria, for while some species do not swarm at all and their colonies on solid media remain localised and more or less well defined, though they gradually enlarge (e.g. bacillus subtilis, bacillus fluorescens, some species of proteus, and many spirilla), other species possess this swarming propensity, and therefore the first colonies do not remain well defined, but gradually extend in lines and irregular streaks in different directions. But it is not correct to conclude that a bacillus is motile if its colonies do not remain defined, that is, if they extend in the shape of threads or irregular streaks on or through the medium, for there exist several well-studied species which do this (e.g. bacillus anthracis, bacillus filamentosus), though their bacilli are not motile, as will be more minutely described when speaking of the cultural characters of bacilli.

When certain bacilli show only slight motility it may be extremely difficult to distinguish this from Brownian molecular movement, but no locomotion can be ascribed to bacilli unless one or the other individual can be distinctly seen to show a darting or spinning movement. As mentioned above, the easiest and best way to see locomotion is to examine the fresh bacilli in a fluid, as sterile broth or sterile salt solution in the 'suspended drop.'

The motility of bacilli and spirilla is due to their possessing at one, and occasionally at both ends, or also over the general surface, fine flagella or cilia, the movement of which causes the motility of the microbe. Where two or more microbes are connected into a chain or thread, only the terminals have the flagella. Although the flagellum has been seen and photographed only in the larger bacilli and spirilla, there can be no doubt that all motile organisms do possess the flagellum, for without it motility would not be possible. Micrococci are not possessed of motility, but recently Ali-Cohen has isolated from drinking-water a species of micrococcus (*Micrococcus agilis*) which forms an exception, since this species is motile ('Centralbl. für Bact.' VI. 2).

Important methods of demonstrating (staining) the cilia have been published by Löffler ('Centralbl. f. Bacteriologie und Parasitenk.' VI. Bd. 8, 9). Löffler points out and gives photographs of bacilli, vibrios, and spirilla which

are possessed not of one, but a bundle of fine cilia at one end or over the whole body.

In most bacilli and spirilla which are possessed of motility this is intimately connected with a supply of oxygen. Though some species seem to obtain this readily even when in deep fluids (e.g. bacillus of hay, certain species of proteus), many others cease to move when the supply of oxygen becomes insufficient. Engelmann has made some very interesting experiments with certain motile bacilli, showing the direct influence of oxygen on their motility. When motile bacilli, owing to insufficient oxygen or after the consumption of the oxygen previously present, come to rest, by adding to them new oxygen in a drop of fresh fluid containing air, the motility is resumed. On removing the oxygen and adding carbon dioxide or hydrogen gas, ammonia, chloroform, or ether, the movement ceases, but on removing these gases and replacing them again by oxygen (or air) the movement is again resumed.

Motile bacilli and spirilla when growing in a fluid medium have a great tendency to seek the surface of the fluid, i.e. move towards the part where they can obtain oxygen, and here form more or less coherent pellicles, in which they are in a resting state, and in which a rapid multiplication goes on; but it is quite incorrect to assume that an organism which in a fluid medium forms a pellicle is a motile organism, since some species which form a pellicle are not motile, and some species of motile organisms do not form a pellicle.

CHAPTER II

CONDITIONS AND MODE OF MULTIPLICATION OF BACTERIA

BACTERIA when planted on a medium containing the necessary ingredients, and when the other conditions, as temperature, moisture, reaction, &c., are favourable, multiply with great rapidity. Substances containing nitrogenous compounds, potassium and sodium salts, phosphates and sulphates, are to be considered as necessary ingredients. In regard to these substances both qualitatively and quantitatively there exist, however, very great differences indeed. While some bacteria are capable of growing and multiplying on simple nitrogenous compounds (e.g. tartrate ammonium, ammonia, urea, uric acid), many others do not show any growth under such conditions. Many common or saprophytic bacteria living in the soil, in putrid or ammoniacal urine, thrive well when only simple nitrogenous material is available; e.g. such as is present in normal urine; while those bacteria like the disease-producing microbes, as also those generally present in dead organic matter of the living or non-living nature, thrive well only in the presence of albumen or albuminoid nitrogenous material. Various species of bacteria (micrococci and bacilli) occurring in ammoniacal urine, in soil, and other substances show active growth when planted in a fluid that contains nothing more complex than tartrate of ammonium (Pasteur's and Cohn's fluid); a great many species, amongst them all the pathogenic bacteria, do not show appreciable growth under these conditions. As regards the amount of the albuminous material, some species like those present in ordinary drinking-water, which contains only traces of albuminoid matter, are herein capable of multiplying, whereas others accustomed to live on more copious nitrogenous soil when transferred to such water gradually diminish in numbers, and ultimately, unless in the form of

spores, altogether die. The greater the amount of albuminous material present the greater will be found, *cæteris paribus*, the capability of multiplication. As regards the lowest limit of proteid required for active and good growth there exist great differences, as great as those previously named; for while the presence of albuminous material is to all bacteria a stimulation for multiplication, it is not, as stated above, absolutely essential to some. As a matter of fact there exist infinite gradations between species that require for good growth a comparatively large amount of highly differentiated albuminoid material—e.g. some pathogenic bacteria like tubercle bacilli—and those that can do well with very little of it, and even without it, but with only a little urea, or tartrate of ammonium—e.g. those in ammoniacal urine, those in soil, some of those in ordinary drinking water. More than that, there are at present some known species which can grow well on materials not containing any nitrogenous material at all; thus the bacillus amylobacter or clostridium butyricum can grow well on no higher organic material than starch (Prazmowski). Some organisms in the soil multiply most probably at the expense of the nitrogen absorbed from the air (*see below*), and Macfadyean shows that some species of bacilli, though accustomed to live on albuminous soil, can nevertheless do well on pure starch paste. Some bacteria in drinking water—e.g. *Micrococcus aquatilis* (Niessen)—are said to be able to exist even in pure distilled water. Now in these cases it must be assumed that the nitrogen necessary for the building up of the protoplasm of their own bodies is obtained by the bacteria by absorption of free nitrogen from the air. The protoplasm constituting the principal part of the body of the bacteria is, like all other protoplasm, essentially albumen, and therefore, in order to grow and multiply, the bacteria have naturally to build up protoplasm; and since this can only take place at the expense of nitrogen absorbed from the air (as in the cases just mentioned), or at the expense of simple nitrogenous compounds like salts of ammonia, urea, &c. (as in the cases of bacteria mentioned previously in putrid urine, water, and soil), it follows that some bacteria, at any rate, are capable of forming albumen by a process of synthesis, and in this respect, therefore, they compare with plants and not with animals. In the former this is the natural proceeding, while in the latter the protoplasm of the tissues is formed at the expense of the proteids taken in the first place with the food.

PROTEID MEDIUM

Most bacteria grow well in media containing 0·5 per cent. of proteid, less than is present in potato (1·5 per cent.); but there are some which show scanty or no growth on potato or in other media when only 0·5 per cent. proteid is offered. There does not seem any known reason why this should be so, but it is nevertheless a fact. Thus, one of the most highly differentiated microbes, like the glanders bacillus, grows well on potato, while, for instance, the diphtheria bacillus and the bacillus of fowl enteritis do not show any appreciable growth under the same conditions. This, however, does not apply to pathogenic microbes only, for some non-pathogenic microbes cannot grow on so small an amount of proteid as is present in the potato (1·5 per cent.).

SALTS

From the *presence of potassium salts and phosphates* in the bodies of bacteria the presence of these substances in the nutritive medium must be concluded as absolutely essential.

MOISTURE

An important influence on the growth and multiplication of bacteria is exerted by *moisture*, since their own bodies and the protoplasm constituting them contain water. Without a small amount of water no growth takes place. In fluid media the growth of all bacteria is, *ceteris paribus*, more rapid than on solids, and on these the growth varies, *ceteris paribus*, proportionately with the amount of moisture. It is easily shown that two solid media of the same kind, but differing from each other in the amount of water present, inoculated with the same species of bacteria will, after the same periods, and kept under otherwise the same conditions, show a remarkable difference of growth; the one that contains most water shows copious growth, while the other shows only comparatively little. For instance, on preparing two quantities of nutritive gelatine (meat juice peptone gelatine or broth peptone gelatine), one containing 97 per cent. water, the other 89 per cent., and inoculating these with various species all in double series, e.g. anthrax bacillus, staphylococcus, streptococcus pyogenes, bacillus subtilis, cholera spirillum, bacillus pneumoniæ, bacillus of fowl enteritis, bacillus of typhoid fever, or common saprophytic species, a notable difference will be noticed after two or three days' growth at 20° C.; those tubes containing gelatine with 89 per cent. water will show considerably less growth than those with 97 per cent. But it must be here mentioned that some bacteria are more sensitive to small amounts of moisture than others; for while some species show comparatively good growth when a small amount of free moisture is present, others show no growth unless the amount is considerable. Thus, for instance, many species like bacillus subtilis, and bacillus anthracis, various species of spirilla, grow well under the first conditions, while others, e.g. the tubercle bacillus, require for good growth a visible amount of moisture.

TEMPERATURE

Temperature plays an important rôle. Most bacteria show good growth between 18° C. and 38° C., and most between 30° C. and 37° C. Some show growth, but to a less extent, below 18° C., as low as 15° or even 14° C.; or to a higher extent above 38° C., as high as 42° and 48° C. Some few species are known that show good growth at a higher temperature, some even as high as 60–70° C., e.g. the bacillus thermophilus of Miquel, the micrococcus found in hot springs by Hoppe-Seyler. The most remarkable point about the bacillus thermophilus is that it ordinarily exists in the soil, and that nevertheless it shows very excellent growth and spore formation at temperatures even above 60° C.

Some species do not grow well at temperatures below 22° or 23° C., some not even below 30° C., or even 35° C.; to the first belong the diplococcus pneumoniæ of Fraenkel and Weichselbaum, a streptococcus occurring in the fluid of the mouth under normal conditions, a bacillus similar to but not identical with the bacillus of diphtheria, viz. the pseudo-diphtheria bacillus of Löffler; to the latter the tubercle bacillus, the leprosy bacillus, and to a lesser extent the glanders bacillus. Some species do not grow at temperatures above 22° or 23° C.; for instance, a micrococcus (streptococcus) which the writer isolated from certain infectious ulcerations of the teats of milch cows, a bacillus which he found in veal pie that had caused acute chemical poisoning (gastro-enteritis), and a bacillus which he found in dust taken from the plugging of the floor of dwelling-houses; these grow well at 20–22° C. in broth, gelatine, and agar mixture, but when exposed in these media to

25–30° C. no growth takes place. The bacillus prodigiosus, while growing well at temperatures below 25° C., does not grow at higher temperatures.

In ordinary drinking water, and in the air of dwelling-houses, there are many species of bacteria which grow well at 18–20° C., and at all degrees up to 38–40° C., but there are some that do not grow at temperatures below 25° C.; therefore testing for the presence and number of bacteria in water and in the air of dwelling houses by the simple gelatine plate method, in which the nutritive gelatine is kept at a temperature between 18°–22° C. (20° C. on the average), does not reveal all species present, since there are some which do not grow and multiply at these temperatures.

AIR

Pasteur was the first who showed a radical difference to exist between different bacterial species in their requirement of free access of air during their growth and multiplication. He distinguished those that can grow without free access of air as anaërobic, the others as aërobic. At present when the mode of growth of the different bacterial species can be more accurately studied (owing to greater perfection in the methods of cultivation), the bacteria are capable of being grouped as follows: (1) obligatory anaërobic; these are such species as do not grow except when free access of oxygen (air) is excluded; such are the malignant œdema bacillus of Koch, the clostridium or bacillus amylobacter of Prazmowski, the tetanus bacillus, the bacillus of symptomatic anthrax; (2) facultative anaërobic, those species which can grow in absence of free air, though they grow better if air be present; these and the following form the majority of the species hitherto known; (3) facultative aërobic are similar, and the boundary line is indistinct; speaking generally, these grow better without free air, though they grow also, but not so well, when free air is admitted, i.e. on the surface of the nutritive medium; and (4) obligatory aërobic, those that do not thrive and multiply without copious supply of air; as, for instance, the hay bacillus, various potato bacilli. In the study of the obligatory anaërobic bacteria it is, of course, necessary to see that the seeding is carried out so that they are deposited at once in the depth of the solid medium, or that if seeded on the surface this latter is then covered up by a new layer of medium—oil or paraffin, or a layer of the same medium, e.g. a new layer of gelatine or agar;—or that culture tubes are used from which the air is removed by the pump, or replaced by some indifferent gas (H), and then sealed; or that tubes are used which are completely filled with the medium and after inoculation are sealed. But in all cases in which anaërobic bacteria have been grown, it is sufficient to see that the seeding takes place in the depth of the gelatine, away from the surface. The growth, then, takes place only in the deeper layers (see the figures of cultures of malignant œdema bacillus of Koch and tetanus bacillus).

RAPIDITY OF GROWTH

The rapidity with which bacteria grow and multiply is subject to very great variations, and, *ceteris paribus*, constitutes definite and characteristic peculiarities; that is to say, some species under the same conditions of soil, temperature, &c., show a more rapid growth and multiplication than others, these bearing no relation to any known condition. Thus of the staphylococcus aureus and the streptococcus pyogenes, growing under exactly the same conditions, and on good nutritive media, the former shows incomparably greater rapidity in multiplication, and produces much more copious growth in a given time than the latter; or if the bacillus of swine erysipelas and the bacillus of

swine fever be taken, the latter is found to grow much more rapidly than the former; and, again, bacillus subtilis and Finkler's spirillum grow very much faster than bacillus anthracis and cholera spirillum respectively.

Comparative experiments which the writer has made with a number of microbes as to the rapidity of multiplication, by way of observing them directly under the microscope in a drop of solidified nutrient gelatine at 22° C. ('suspended solid drop') show as the average of several observations—

(a) The streptococcus pyogenes. Complete division of the cocci took place in thirty minutes.

(b) The staphylococcus aureus liquescens in twenty minutes.

(c) The streptococcus of erysipelas in forty-five minutes.

(d) An orange-coloured non-liquefying micrococcus in forty minutes.

(e) The bacillus anthracis in thirty minutes.

(f) The bacillus subtilis of hay infusion in twenty minutes.

(g) A filamentous bacillus liquefying gelatine, not mobile and isolated from sewage, in eighteen minutes.

(h) A mobile bacillus (bacillus fluorescens liquescens), rapidly liquefying gelatine and common in ordinary London drinking water, in eighteen minutes.

(i) A bacillus, non-mobile, non-liquefying, rapidly forming spores, and slightly filamentous, isolated from London sewage, in forty minutes.

(j) The bacillus of the Middlesbrough pneumonia in eighteen minutes.

(k) The bacillus of fowl enteritis in twenty-four minutes.

(l) The bacillus of typhoid fever in thirty minutes.

(m) The bacillus diphtheriæ in forty-five minutes.

In all these instances a single organism lying isolated was focussed and watched, and after a distinct division had been noticed the time was marked, and the interval it took for one of these to again completely divide was taken as the time for a division. In these observations, which do not claim more than approximate accuracy, it was remarked that the division of the two members of the dumb-bell cocci or dumb-bell rods does not proceed at the same rate, the difference being as much as a quarter to a third of the whole time. The above numbers indicate the average of three successive divisions, and therefore they only represent approximately the mean periods that these several microbes require for dividing under the above conditions. Buchner ('Centralbl. für Bact. und Parasit.' II. No. 1) calculated the time required for the cholera vibrio for a division at 37° C., and found it to amount to twenty minutes on an average.

Observations were made on the common staphylococcus pyogenes aureus, the bacillus of swine fever, the bacillus of grouse disease, the bacillus of fowl enteritis, and the bacillus of diphtheria, as to the amount of multiplication these several microbes undergo when a definite number of them is introduced into faintly alkaline beef broth (eight to ten cubic centimetres) and kept in the incubator at about 37° C. All these different organisms grow with great rapidity, and after twenty-four hours the broth is uniformly turbid, provided the number introduced at starting be comparatively large. By making gelatine plate cultivations with a given small quantity of the broth previously diluted to a definite degree, and then counting the number of colonies that make their appearance on incubation, it is easy to calculate the number of microbes present per cubic centimetre in the broth. In some experiments made with the staphylococcus pyogenes aureus it was found that on introducing 248 microbes per cubic centimetre, they increased in the first twenty-four hours to 20,000,000 per cubic centimetre; in another experiment 640,000 per cubic centimetre were counted after the first twenty-four hours' growth, 248,000,000

per cubic centimetre after the second twenty-four hours, i.e. after forty-eight hours' incubation, and 1,184,000,000 per cubic centimetre after the third twenty-four hours i.e. after seventy-two hours' incubation. From a number of experiments it was calculated that for each microbe introduced, the multiplication during the first twenty-four hours is 80,000-fold, during the second twenty-four hours 400-fold, and during the third twenty-four hours 5-fold.

The rapidity of the growth and multiplication of the bacillus of fowl cholera in the living blood was ascertained in an experiment made on a rabbit. Of the microbes 20,000 were subcutaneously injected into a rabbit. The animal died in about twenty hours. The bacilli in the heart's blood were then counted by the ordinary method of gelatine plate cultivation, and it was found that their number per cubic centimetre of heart's blood amounted to 14,150,000. The weight of the rabbit was 1,250 grammes, and taking eighty-three grammes ($\frac{1}{12}$) as the amount of blood present in the animal's body, and assuming that the bacilli were more or less uniformly distributed through the blood, it follows that the total blood contained about 1,200,000,000 of the bacilli. This would mean that each one of the 20,000 bacilli injected had given origin to a host of 60,000 bacilli in twenty hours.

The manner in which the individuals of the same species divide varies considerably; thus in the streptococcus scarlatinae and str. pyogenes the writer has observed that in gelatine some of the elements of a colony increase rapidly to five, six and more times the size of a typical coccus, grow, in fact, into a ball of great size, then a cleft appears by which the organism splits up into two demilunes, then each of these again divides under a right angle to the former line of division, so that the original ball is divided into four quarters, each of which separates gradually from its neighbour and becomes more or less spherical, and a further division into two and even into four cocci of the average size takes place. But the above mode of division does not take place everywhere in the preparation, for many of the typical cocci only slightly enlarge and then divide into two, thus forming a diplococcus; each of these divides again transversely, and thus a chain of four minute cocci is the result.

In broth cultures the writer has observed, as a rule, the latter mode of division, though also here occasionally an element is noticed in a chain which is much larger than the rest, and this larger element divides into two and four cocci successively. So also those large elements described above as occurring in the chains of streptococci show the successive fission into two and four cocci. And it is this which prompts him to say that these large elements found occasionally in the chains or in the diplococci are not involution forms, but are active elements which before successively dividing grow up to large size. In staphylococcus aureus liquescens, growing in gelatine, he has also observed some of these large elements, though on the whole they are not so numerous as in the streptococcus growing in the same kind of medium. The normal mode of division of a coccus is then (1) a slight enlargement and division into two by transverse fissure, or (2) a coccus enlarged to considerable size (four to six and more times) and then successively divided into two and four and further eight cocci of the normal size.

As regards bacilli, all observations hitherto recorded agree that a rod before dividing elongates sometimes more, sometimes less, and then a transverse indentation appears about midway, which ultimately becomes a fissure by which the originally single rod divides into two; according as the rod was short or long, the resulting offsprings are more coccus-like or more cylindrical. Now, in the observations which the writer has carried out as to the time of the division of the different microbes mentioned above, he

has repeatedly noticed that a single cylindrical bacillus not infrequently divides almost simultaneously into three and even four short rods. The writer has observed cylindrical bacilli in preparations of bacillus anthracis, made directly from the blood of guinea-pigs, which were uniform, and there was no indication in the fresh specimen that they were other than single elements. These elements he has seen to give origin almost simultaneously to as many as four short slightly rod-shaped elements; and these same elements were, on continued observation, seen to elongate and the terminals within several minutes seen to have increased almost to twice their length, then each of them again to have divided, one into three, the other into two distinct rods.

Observations were carried out on a filamentous bacillus isolated from sewage liquefying gelatine as a clear fluid; it was non-motile, rapidly growing into threads, and in the filaments copious spore formation took place; this bacillus resembled morphologically the bacillus anthracis, but it grows on the surface of gelatine more as a continuous membrane of threads arranged parallel and coming off at right angle from a central stalk; it grows much more rapidly than the bacillus anthracis.

Now, directly observing under the microscope the growth and multiplication of this bacillus in solidified gelatine, threads of bacilli are seen shooting out with considerable rapidity from a short cylindrical bacillus measuring 0.5μ , to 1μ , a thread more or less wavy is formed in the course of two hours and a half, which extends across the whole field of the microscope under a magnifying power of 500. On such a growing thread the simultaneous division after elongation of cylindrical elements into three and four rods is also distinctly and repeatedly noticed.

Also on the rods of the bacillus of diphtheria the same simultaneous fission of elementary cylindrical cells into two, three, and four elements was noticed. We conclude then that in the division of bacilli the elements increase in length and then by transverse fission divide into two, three, or four elements, and according to the length of the cell before division the elements resulting from the division differ in length.

SPORES

One of the most important and interesting phenomena in the life-history of bacteria is the power of some species to form *permanent seeds or spores*, by which the species can preserve itself and can withstand a variety of adverse circumstances. Various conditions in nature are often at play, in consequence of which weaker species are less liable to survive in the severe struggle for existence. There is first the adverse circumstance of competition, such as constantly obtains under the general conditions of growth in soil, in water, and in various organic materials exposed to contamination from air, water, and soil. Here numerous species find access and multiply, some more, others less easily, till all the available nutriment is exhausted. Some species, capable of forming spores, when this stage of the exhaustion of the nutriment has been reached, remain as spores and, till they are transferred by some means or other to new material, or till new nutriment is added, retain their power of again germinating and giving rise to a new crop of the same species, and this survival occurs even under severe adverse circumstances, e.g. the presence of various noxious chemicals, cold, heat, drying, &c.; but those species that do not form spores retain life only under exceptionally favourable conditions; as a rule, owing to the presence of acids or other chemicals, e.g. products of the growth of bacteria, and owing to drying, &c., they are easily deprived of life. This question of the formation of spores for the

above reasons plays a most prominent rôle as regards infectious diseases. A few illustrations will easily show this. Take, for instance, the bacillus anthracis. This organism, although present in enormous numbers in the blood and blood-vessels of animals dead of the disease, does not at any time form spores when kept away from the air, i.e. from a supply of oxygen; consequently in such an animal when left unopened all the bacilli, after having gone on increasing in numbers after death for some time, gradually degenerate and disappear, so that sometimes after five to eight days, in the case of small animals like mice and guinea-pigs, living anthrax bacilli are no longer to be found in the tissues, they having been suppressed by putrefactive organisms. The spleen of such an animal after this distance of time produces no infection with anthrax after inoculating it in comparatively large doses into a fresh guinea-pig or mouse; whereas if a trace of a droplet of the splenic blood of an animal dead of anthrax is used for inoculation of a guinea-pig or mouse, say within three days after death, virulent anthrax follows. Or if the blood and tissues of an animal dead of virulent anthrax are by some means or other thoroughly dried, such blood loses all virulent power, since by thorough drying the bacilli anthracis are killed. But let either the blood or the nasal or other discharges of an animal dead from anthrax be exposed to air for a sufficient time to allow the bacilli to form spores, then neither putrefaction, nor drying, nor chemical agencies such as acids and alkalis, will affect the power of these spores to germinate again into bacilli and to produce virulent anthrax when finding access to a suitable animal body. This is actually the case when cattle and sheep are sojourning on and feeding in a field, where months or even years previously an animal having died from anthrax, the blood and discharges of such an animal found access to the surface of the soil, that is where the bacilli anthracis find opportunity to multiply and to form spores. It is these spores which afterwards are picked up by the animals grazing in such a field. The same thing occurs in woolsorters' and hidesorters' disease, which is virulent anthrax in the human beings engaged in the sorting of wool or the handling of hides derived from animals—sheep, goats, and cattle respectively—which had succumbed to fatal anthrax. In these cases it is always spores of the bacillus anthracis which are the cause of infection of the human beings handling these articles. Or take another illustration as regards putrefactive organisms. In preserving various articles of food, these articles are often subjected to the temperature of boiling water for variable periods from a few minutes to a quarter of an hour or thereabouts. Although all micrococci and bacilli not bearing spores are killed by such exposure, the spores of some species are not killed by such a temperature, and therefore if such spores happen to be present before the exposure, the spores will remain unaffected by the exposure to that temperature, and afterwards will be able to germinate into bacilli, which by their multiplication cause a very undesirable change in those articles. Cohn found in tinned peas some species of bacilli whose spores withstood the temperature of boiling water for thirty minutes, and although these must be considered exceptionally resistant spores, nevertheless there exist not a few species of bacilli whose spores can successfully resist the exposure to 100° C. for two, three, and even five minutes; an exposure to 95° C. some resist for nearly an hour; in fact, the writer knows of spores of several species which withstand easily such an exposure, amongst them the spores of bacillus subtilis and bacillus anthracis. But no micrococcus or non-spore-bearing bacillus can withstand exposure to 70° to 75° even for a few minutes; many of them are killed at 60° C. or even 58° C. in five minutes.

The presence or absence of spores is then, for the various reasons indicated above, of great practical importance.

Observing bacilli, which do form spores (e.g. *bacillus subtilis*, various species of 'potato bacillus,' *bacillus mesentericus*, *bacillus anthracis*, and the *bacillus filamentosus* above mentioned), it is noticed that the first sign of the appearances of spores is indicated by the presence of a bright, glistening globule in the protoplasm of the bacillus; at the same time the bacillus is distinctly broader and paler in its substance as compared with the other bacilli. This globule gradually enlarges in diameter, becoming at the same time slightly oval; this continues till the thickness of the globule often exceeds the breadth of the bacillus, this latter being now markedly pale and transparent. The writer has watched in *bacillus anthracis* and *bacillus filamentosus* the spores from their first appearance as bright globules till they had reached their full thickness and length; this took about three hours, and he has also noticed that after sowing on the surface of solidified agar the blood of the heart or spleen of a guinea-pig dead of anthrax and keeping it under observation at the temperature of 20° C. spores would be noticed in a few of the bacillary filaments after twelve hours; in the case of the *bacillus subtilis*, various potato bacilli, and *bacillus filamentosus* growing in broth, copious spore formation was noticed in a superficial pellicle after sixteen hours. Koch first observed that spore formation in *bacillus anthracis* occurred after six hours. But not all bright granules that make their appearance in bacilli are spores; thus in the typhoid bacillus growing on potato, and in other species of bacilli growing on potato, there appear bright granules, either terminally or centrally, which are not spores; they do not show the reaction of spores, either to dyes, or on drying, or heating. Nor are all the bright granules that make their appearance in bacilli capable of forming spores to be at once taken as spores, since under certain conditions such granules do occur, but never reach the size of full spores; this is observed occasionally in anthrax bacilli when growing under conditions unfavourable for the formation and development of spores. The appearance of real spores in all bacillary species is very characteristic; the spores are of a bright, glistening aspect, are oval in shape, and generally thicker than the typical bacilli. The substance of these latter is at the same time pale and transparent and broader than the bacilli not containing spores; the threads of the bacilli appear beaded by the spores, the beads being the glistening oval thick spores, while the rest of the thread is pale and appears thinner. In the single bacilli the spores are placed either centrally or terminally; in the latter case, if the bacillus is of some length, it looks not unlike a spermatozoon, the spore corresponding to the head, the bacillus to the tail. Sometimes in motile bacilli short chains are noticed, in which, in one terminal element, a spore has already made its appearance, while the other bacillus is still possessed of motility; and here, on account of the motility, the resemblance to a spermatozoon is still more striking. Under the most favourable conditions almost every element constituting a bacillary thread or chain forms a spore, in other threads only here and there a cell contains a spore; in the first case the thread is regularly and densely beaded, in the latter the beads are relatively few and far between. The last phase is reached when the bacillus itself swells up into a gelatinous capsule enveloping the spore and ultimately altogether disappears; then the spore is free and has reached its full size and development. Examining in stained specimens spore-bearing bacilli, the spores appear unstained, whereas the rest of the bacillary substance takes readily the dye; under these conditions the spore looks like an oval clear space, not unlike the vacuoles above mentioned; but

the spore has a sharp outline of its own, the vacuole has not. It is, however, not easy to distinguish in a given specimen, stained after the ordinary methods, the spores which are not stained from vacuoles, and in these cases other methods of staining must be resorted to.

In order, then, to decide whether or not spores are present in a bacillary species, the morphological investigation, fresh aspect, special methods of staining, drying, and heating have to be resorted to.

The spore formation is associated with supply of oxygen in all bacteria that generally live well under access of air; in some species this is more pronounced than in others, for in some species, e.g. *bacillus anthracis* and *bacillus filamentosus*, spores are only formed, *cæteris paribus*, if oxygen has free access, and no spores are formed if there is no free supply of oxygen, e.g. deep in the fluid; while in other species, though spore formation is greatly enhanced by the free access of oxygen, it nevertheless takes place to a certain limited extent deep in the fluids. Thus, in the case of *bacillus subtilis* and various other bacilli a pellicle soon makes its appearance on the surface of the fluid (broth, &c.). This pellicle is made up of filaments and bacilli matted together, and in them copious spore formation is going on, but also in the depth there are a few spore-containing bacilli to be noticed. When such a pellicle is broken up by shaking, it in most instances falls to the bottom of the fluid, and then after another day's growth a new pellicle appears, and in this also copious spore formation is noticed; and this can be repeated for several days till the nutriment is exhausted. The same can be seen in hay infusion, in the case of *bacillus subtilis*. In neutral or faintly alkaline hay infusion kept at 37° C. spores of *bacillus subtilis* are present in the pellicle as early as the second day, and continue to be formed at the end of eight to ten days. The view has been expressed by some observers, amongst them Buchner, that the spore formation in bacilli occurs on exhaustion of the nutritive material, but it seems that the facts just mentioned as to the continuous and successive pellicle and spore formation occurring in broth are incompatible with that assertion; and, besides, the formation of spores in other bacilli can be shown to take place long before any exhaustion of the nutritive matter is noticeable, e.g. in anthrax bacilli, in the tetanus bacillus, and others. In the *bacillus filamentosus*, growing on agar or on potato, the spore formation is apparent even before the first day is over and long before the active growth and multiplication of bacilli all round is finished. In some species, however, that do not thrive under free access of air (e.g. *œdema bacillus*, *tetanus bacillus*) spore formation does not take place if free oxygen is present. A temperature of at least 16° C. is required for the formation of spores, though spore formation occurs in all temperatures between that and 45°; at least, spore formation has been seen to occur in *bacillus anthracis* even at 45° C. The mode of spore formation hitherto described is that of endo-spores, and it ought to be here stated that many species of bacilli exist in which no spore formation can be demonstrated—in this statement we rely on the morphological as well as the experimental test—e.g. typhoid fever bacillus, bacillus of glanders, of diphtheria, of fowl cholera, fowl enteritis, and many others. So far as actual demonstration is concerned, no other mode of spore formation can be accepted at present. A mode of formation of spores is described by Hueppe to occur in certain spirilla, according to whom the comma-shaped elements and the spirilla form special aggregations of protoplasm in the shape of terminal granules, to which the value of spores is ascribed, and which are called arthro-spores. But the evidence and proof for this is quite unsatisfactory, and, judging these appearances in the light of the

characters of well-ascertained spores of other bacilli, they are contrary to the assumption of spores. These arthro-spores of Hueppe do not look like spores, do not behave in staining like spores, and do not behave in drying and heating experiments like spores. In the first place they do not differ in aspect from ordinary protoplasmic granules observable in some of these bacilli under all conditions; they stain in the ordinary dyes and after the ordinary methods like the ordinary protoplasmic contents of bacteria; and they are killed by drying and exposure to 60° C. for five minutes.

It can be easily shown that artificial cultures of these comma bacilli growing under conditions very favourable for the formation of real spores in other bacilli, e.g. a good supply of oxygen, temperature, soil, and moisture, contain after some weeks and months those granules or supposed arthro-spores in enormous numbers; in fact, there is almost nothing else left, and yet no sub-cultures can be established from such a culture, it being barren of all life. Structures have been described also in the typhoid bacilli as occurring in potato cultures, which can be, however, shown by special modes of staining to be different from real spores, and the experimental test of drying and heating conclusively proves that they are not comparable to spores.

On the other hand, the tubercle bacilli have been shown experimentally (by Koch and others) to possess spores, although it seems difficult to identify them under the microscope. True, there are present in microscopic specimens made of fresh material, e.g. tubercular sputum, bright granules within many of the bacilli which might be taken for spores, and in specimens stained after the customary method of staining for tubercle bacilli, numerous stained granules occur in the bacilli—the bacilli appearing beaded—but, as has been stated above, most of them are merely elementary masses of protoplasm segregated in the bacilli. They occur in some tubercle bacilli more numerously than in others, e.g. in the tubercle bacilli of the human subject they are common; in the tubercular material of the fowl and in artificial cultures they are sometimes seen with great regularity, but there is no means available of identifying these granules with spores. But by thorough drying of tubercular material it can be shown that the tubercle microbes remain uninjured, and that heating them up to 100° C. for a minute leaves the tubercle bacilli unharmed.

Spores have been described also of some micrococci, but here again certain differentiation of structure cannot be taken as proving the existence of spores. The writer has examined very numerous preparations of the most varied cultures of different species of micrococci, and the test of drying and heating to 70° C. proves them barren of anything comparable to the well-known spores present in some species of bacilli. He also examined experimentally various species of spirilla, and agrees with Koch that no spore formation can be demonstrated in them. We arrive then at the conclusion that real spore formation, or the formation of permanent seeds capable of retaining life under very adverse conditions, and under favourable conditions capable of germinating and of giving origin to a new brood of the same species, can be shown to exist only in certain limited species of bacilli, but not in micrococci or spirilla. These real spores are endo-spores, they are formed within the protoplasm of the bacillary elements under favourable conditions, and they have certain definite morphological and experimental characters of their own, at the same time representing as it were the last phase in the life-history of these bacilli. Bacilli or bacteria not capable of this power of producing spores, though they go on multiplying as long as the conditions of nutriment, temperature, chemical by-products, competition, &c., permit of it, ultimately degenerate, some sooner, some later. To propagate their species they must as living bacilli find access to new soil before the stage of degeneration is reached, whereas in the spore-

bearing bacteria their spores can remain dormant, but possessing potential life for indefinite periods.

The statement has been occasionally made that spores are capable of dividing, and thus giving origin to two new spores. The writer has not been able to detect anything of this sort; he has never seen any appearances that would indicate such a division. True, in *bacillus anthracis*, in *bacillus filamentosus*, and in some species of 'potato bacillus,' spore formation may be going on so copiously that at some places every element constituting the threads contains a spore, some of the spores closely adjoining one another, sometimes so closely that it looked as if they were the two elements of a dumb-bell, and here a division of one spore into two could be thought of; but in these places the elements of the threads were extremely short and the spore occupied the main part of each element. It is also a fact that spherical globules occurring in some bacilli and being in aspect and staining power comparable to young phases of spores, are occasionally met with as dumb-bells within the same element; but as regards the oval, bright, unmistakable spores so prominent in the bacilli and their threads (*bacillus anthracis*, *bacillus subtilis*, *bacillus filamentosus*, *bacillus mesentericus*, &c.), it is very doubtful whether they are capable of dividing or of undergoing any other change than that of germination into bacilli when they are transferred to new soil.

Spores when placed under suitable conditions germinate again into bacilli. This is easily observed if, for instance, a trace of any culture-material containing spores is placed on a cover-glass, then covered with a tiny droplet of gelatine which is made to set rapidly, or in a droplet of broth ('suspended drop'), and is then observed under the microscope, particularly in the latter medium, which can be kept on the warm stage heated to 37° C.

Spores while fresh have a conspicuously sharp and dark outline, their general aspect is glistening, and it is supposed by Cohn that they are possessed of a double envelope, an inner one of a fatty and an outer one of a gelatinous nature: it is particularly the former which provides the spores with their great resistance to drying and to heat. The first indication that the spores are going to germinate is shown by their outline becoming less sharp at one point. This is generally at one of the poles, as in the case of the spores of *bacillus anthracis*, *bacillus filamentosus*, and *bacillus subtilis*; or it is at one point of the long side, e.g. in *bacillus amylobacter*, and also in the spores of some of the species collectively spoken of as 'potato bacillus;' the investment seems to become thinner at that point and a slight pale knob appears there. This knob gradually elongates in the form of a pale rod thinner than the spore itself; as it elongates, it protrudes more and more from the rest of the spore, its free end being rounded, while at the same time the rest of the spore outline becomes thinner and less dark; ultimately the whole spore has been consumed as it were in the formation of the rod, which now looks like a cylindrical bacillus of the same character and aspect as the bacilli from which originally the spore had been derived. The bacillus once formed divides and then continues to grow and multiply. The time required for the production of a bacillus from a spore varies with the different species. Koch observed the germination of the spore into a *bacillus anthracis* to be completed in about an hour; the writer has observed the time required for the complete formation of a bacillus from a spore of the *bacillus filamentosus* in broth, in the 'suspended drop,' at 37° C. to be certainly less than one hour; that of *bacillus anthracis* between one hour and a half and two hours; that of the *bacillus subtilis* of hay infusion to be more than one-half but less than one hour. Occasionally one meets in these observations with motile bacilli to which a spore which has not yet commenced to germinate is attached and is dragged about by the former: this evidently indicates that of two spores originally joined by interstitial

material (*see* spores in threads), only one has already changed into a motile bacillus, the other has not yet so changed.

As must be obvious from the examination of micrococci, bacilli, and spirilla in fresh specimens, the differentiation of the various species belonging to each of these three great groups is based on a number of points which, in some cases, are easily detected, but in others only with great difficulty. As regards micrococci, where the difference in the size of the microbes belonging to different species is conspicuous, the diagnosis is not difficult; likewise in the case of bacilli where some species are motile, others not; or where some bacilli have in a prominent degree the capability of growing into threads or of forming spores, others not; or where some micrococci easily and readily form chains, others not—in all these the diagnosis is not difficult. But from what has been said in the foregoing pages it must be evident that there are a good many species of micrococci, bacilli, and spirilla, which, though totally distinct amongst themselves, as will be presently shown when describing their cultural characters, are, nevertheless, in all the above points alike. In such species we may find, for instance, great resemblance in size and aspect; and we may find in the case of micrococci that they are alike capable of forming chains, or in the case of bacilli that they are similar as to size, motility, power of forming threads and spores; and in the case of spirillum that they are alike as to size, aspect, and motility. Or, to take another character: some species of micrococci and bacilli are capable of forming pigments; now, not a few of these form apparently the same kind of pigment and also under the microscope look very much alike, yet it can be shown by other methods that they are different species. This differentiation is possible by observing the mode of growth of these microbes on artificial culture media, and their different characters on and in these furnish in most instances a ready and conspicuous means of diagnosis and differentiation. These characters are generally spoken of as *biological* characters, to distinguish them on the one hand from purely *morphological*, such as are exhibited by them under microscopic examination, and, on the other, from *physiological*, such as can be ascertained by taking into consideration their chemical activity and their influence on living or non-living (organic) matter. Before entering on the study of these biological characters we shall give a short account (1) of the methods used in studying the morphological, and (2) of the methods used in studying the biological characters of bacteria.

CHAPTER III

STUDY OF THE MORPHOLOGY OF BACTERIA

EXAMINATION IN THE FRESH STATE

THE first and simplest, and at the same time a very interesting study is the study of bacteria in the perfectly fresh state under a sufficiently high magnifying power (300–500 diams.). Hereby the shape, size, motility, and general aspect are observed. The preparation is made by placing a drop of the material (artificial culture, tissue juice, or blood, as the case may be), or if solid by placing a trace in a drop of neutral (0.75 per cent.) saline solution or water previously sterilised by boiling, on a glass slide, and then covering it with a cover-glass. Shape, size, motility, and spores are easily observed by this method of study. An important method for observing the growth, multiplication, formation, and germination of bacteria and spores is the study in the ‘suspended drop.’ This means that a drop of the sterile nutritive material

containing the bacteria is placed on a sterile cover-glass. This is fastened by sterile oil, wax, paraffin, or shellac on an object-glass over a shallow pit cut out of it, i.e. Koch's hollow glass slide, or over a low glass ring cemented to the object-glass. The simplest method is the one used in the observations mentioned above on the period required for division of the different bacteria. It is this: With a sterile platinum needle a very tiny trace of the bacterial culture is deposited in the middle of a clean and sterile¹ cover-glass; then to it is added a thin film of gelatine or agar deposited with a sterile glass rod from a test tube containing sterile nutrient gelatine or agar on the point of liquefaction. The film sets almost instantaneously. The cover-glass is inverted, and fixed on in the above manner to a low glass ring cemented on to a glass slide. Since the bacteria are near the under-surface of the cover-glass, the observation can be made with high powers. If the observation requires a temperature higher than that of the room, up to 37° C., the glass slide is kept on a warm stage.

STAINING OF BACTERIA

In the next place the bacteria are studied in stained specimens: (a) in cover-glass specimens, and (b) in sections. The cover-glass specimens are prepared, after the Weigert-Koch method, by carefully spreading a thin film (the thinner the better) of the culture, tissue juice, blood, &c., pure or after dilution with sterile salt solution, over the middle of a clean cover-glass. Where the material is fluid or semi-fluid, this of course can be done without the addition of any fluid; but where this is not the case, a particle of the matter to be examined is distributed in a droplet of sterile salt solution and rubbed in a thin film over a cover-glass. Then this film is rapidly dried by holding the cover-glass over the flame of a spirit lamp or gas burner, but care is taken that no over-heating or charring takes place; the heating ought to be carried to such point that touching the palm of the hand with the cover-glass is just bearable. If the heating is not carried to a sufficient degree, the danger is that, besides the bacteria, also the ground substance (plasma of blood, the matrix of tissues in which bacteria are present, broth, gelatine, &c.) becomes stained; if the heating is carried too far, even if there is no charring, the danger is that nothing will stain distinctly. Passing the cover-glass rapidly three to ten times through the top of the gas flame is generally sufficient, or with great advantage the cover-glass after drying is steeped for a few seconds in acetic acid (1: 2 or 3 of water), then washed in water and dried again. Then the cover-glass with the side containing the film is placed over the solution of the dye contained in a watch-glass, and left there for the time required to well stain the bacteria. This time is very variable in the case of the different dyes, and in that of the different bacteria, and must be learnt by practice and by experiments almost for each case separately, as it is impossible to state any hard-and-fast rule that would apply to all conditions. But there are certain rules of thumb which might be useful in a general way: The dyes that are very useful for almost all cases, with few exceptions, are: gentian violet, methyl or methylene blue, fuchsin, Spiller's purple, and rubin. Gentian violet, fuchsin, or methyl blue are with great advantage used mixed with aniline water; fuchsin, gentian violet, or methyl blue are very useful with carbol water, rubin, and Spiller's purple as 2 per cent. watery solutions. Aniline water dyes are made thus: Water is saturated by repeatedly shaking it in a bottle with aniline oil and letting it

¹ A sterile cover-glass is always understood to be a cover-glass which has been sterilised by passing it through the gas flame several times or holding it above it for several seconds.

stand for several days ; of this 100 c.c. are taken : to them are added 11 c.c. of a saturated alcoholic solution of fuchsin, or gentian violet, or methyl blue, or methyl violet : Mix well, and filter into a stoppered bottle (Weigert's dyes).

Carbol fuchsin, carbol methyl blue, or carbol gentian violet are prepared like aniline water dyes, but instead of the saturated aniline water saturated carbol water (1 in 20) is used.

A 2 per cent. watery solution of a dye is made thus : 2 grammes of the solid dye are rubbed up in a mortar with 10 c.c. of absolute alcohol ; then, while mixing over a flame, 100 c.c. of distilled water are gradually added till all is dissolved.

In many cases methyl blue is used with great advantage after Löffler's method, viz. methyl blue (saturated watery solution) with equal volume of potassic hydrate (1 in 10,000).

Gentian violet, methyl blue, methyl violet, fuchsin, Bismarck brown, or vesuvin, rubin, iodine green, dahlia, Spiller's purple, Victoria violet, and a host of other basic aniline dyes are used ; some, like methyl blue, methyl violet, gentian violet, Humboldt's blue, and Humboldt's violet, and fuchsin, are useful sometimes as saturated watery, sometimes as saturated alcoholic, solutions.

The cover-glass specimens are left in contact with the dye for from a few minutes to several hours—sometimes exposure for as many as twenty-four hours is required. Keeping the cover-glass with the film downwards over the dye in a watch-glass covered up with a second watch-glass at a temperature from 35–40° C. increases in all cases the progress of staining ; holding it carefully over a flame till the dye begins to steam requires only a minute or so ; where in a given case twenty to thirty minutes are sufficient for achieving a result when the staining is carried on at the ordinary temperature, five minutes at 37° C. produces the required result ; when one to two hours, at ordinary temperature, are required, twenty to thirty minutes' heating to 37° C. produces the result. Many if not most bacteria in cover-glass specimens stain sufficiently well, in Weigert's gentian violet, in Weigert's methyl blue, or in carbol fuchsin in fifteen to thirty minutes at ordinary temperature, in five to ten minutes at 37° C., in a minute or two at 65° C. Then the cover-glasses are removed from the dye, well washed in common water—warm water being better than cold—then distilled water, next dried to remove all water, and mounted, film downwards, in a drop of Canada balsam or dammar varnish. The cover-glass is ultimately sealed down on its edges with a narrow thin layer of Hollis' glue.

In some cases after washing with water, a good but rapid washing with alcohol to remove the excess of the dye, and to bring out finer differences of structure, is required. In some few cases this is not practicable, since the alcohol too easily removes the dye from the bacteria themselves, thus, in the case of spirilla the washing in alcohol must be carried out very carefully, i.e. very rapidly, one dip in alcohol being sufficient. In other cases this washing is not required, e.g. when staining with methylene blue, whereas when staining with Weigert's gentian violet the washing in alcohol is often necessary in order to remove the dye from all except the bacteria, this dye having the undesirable faculty of seizing upon everything present in addition to the bacteria. But whenever the process of heating the cover-glass specimen in the first instance has been carried out successfully, and the staining has not been overdone, simple good washing in water is sufficient to insure a satisfactory result. A successfully stained cover-glass specimen should show the bacteria alone conspicuously stained ; also when nuclei of tissue cells are present these are well stained, but no tissue fibres, tissue granules, mucus, fibrin, or other ground substance should have the stain.

In order to stain cover-glass specimens with two dyes it is best to use one stain after the other, the two stains being a contrast in colouration : red and blue, blue and red, blue and brown, red and brown. Contrast stains are : violet (gentian violet or methyl violet)

and vesuvin, methyl blue and eosin, methyl blue and fuchsin, methyl blue and vesuvin, fuchsin and vesuvin, rubin and methyl blue, or *vice versâ*. If it is desired to demonstrate in different and contrast colours different parts of bacteria, e.g. the capsules and the bacteria themselves, the bacteria and their spores, granules in bacteria and their protoplasm, or the bacteria and the tissue-elements (blood corpuscles, pus-corpuscles) present in the specimen, double-staining is resorted to. The cover-glass specimen after heating is stained in one dye, then washed in water, alcohol, very dilute nitric or other acid (HCl or acetic) as the case may be, or treated in decolourising reagents, e.g. carbonate of potash, iodine, ferrocyanide of potassium, &c. (*see below*), then stained with the contrast stain, washed, dried, and mounted. For a successful double staining the proper degree of staining and washing with each dye must be carefully attended to, and for this purpose preliminary experimenting is essential, for what yields success in one case does not in another. To illustrate this point by different examples: (1) Anthrax bacilli of blood. A thin film of blood of spleen or heart of an animal dead of anthrax is spread out on a cover-glass, dried, and well heated, then this cover-glass is placed in Weigert's methyl blue or carbol methyl blue; after thirty minutes at ordinary temperature the cover-glass is well washed in water, and then placed in an alcoholic solution of eosin; herein the specimen is left for a few seconds to half a minute, then washed in water, dried, and mounted in balsam; the bacilli anthracis and the nuclei of the white blood-corpuscles are in many places deeply blue, the red blood-corpuscles bright red; in some places the blue has been altogether driven out by the red from all bacilli and blood-corpuscles. The point to be attained is that the blue is driven out from the red corpuscles only; if too little staining in eosin, then also the red blood-corpuscles have a bluish colour: the point to be avoided is driving out the blue from all bacilli and blood-corpuscles. Reversed use of methyl blue and eosin often yields good results. A simple and excellent method of double staining is the following: Cover-glass specimens are prepared by well drying a thin film of the material: blood, spleen juice, lung juice, pus, &c.; they are then placed in a 2 per cent. watery solution of rubin, kept there for half an hour or more at ordinary temperature—if warmed to 40 or 50° C. ten minutes suffices—then washed in water and placed in methyl blue aniline water (*see above*) for one-half to two minutes. In this, however, different bacteria differ, for while some take up the blue rapidly, others do it less so; if the cover-glass specimens have been stained for hours in rubin, the time for the second or blue staining can be extended to a few minutes. Then wash well for a few minutes in distilled water, dry, and mount in balsam. The red blood-corpuscles are stained pink, the bacteria deep blue; leucocytes and other cells are pink in their substance, blue in their nuclei. By staining the cover-glass specimens first in rubin the bacteria present take up afterwards the blue much more readily than if stained directly in blue. By this method the writer has obtained excellent and beautiful specimens of a large number of bacteria in fresh tissues. (2) A cover-glass specimen of anthrax blood is stained in gentian violet aniline water or in ordinary watery solution of gentian violet or methyl violet for thirty minutes, washed in alcohol, then placed in a dilute solution of iodine in iodide of potassium for a few seconds till the colour of the specimen changes into purple, then washed in alcohol and placed in a solution of eosin or vesuvin for a few seconds to half a minute, next washed in water, dried, and mounted in balsam: the bacilli are blue, the blood-corpuscles are red or brown: this method is the method of Gram. (3) A cover-glass specimen of the capsulated bacillus of Friedländer is stained in methyl blue aniline water, washed in alcohol, next kept in alcoholic eosin solution for several seconds (ten to thirty), then washed in water, dried, and mounted in balsam: the bacilli themselves are blue, the capsules are pink. (4) A particle of tubercular sputum, or tubercular deposit of any organ, is spread out in a thin film on a cover-glass, dried, and heated as above, and then placed over carbol fuchsin solution, as above described, kept therein for thirty minutes at 87° C., at ordinary temperature for one-half to one hour, then washed in warm water, then washed for five to thirty seconds in a solution of nitric acid of the strength of one part of commercial nitric acid in three to ten parts of water, next washed in water and placed over methyl blue aniline water for half a minute at ordinary temperature, then washed in water, dried, and mounted in balsam: the tubercle bacilli are red, all other corpuscles, cells, nuclei, and septic bacteria are blue. (5) To stain spores in bacilli. Spores do not stain after the ordinary methods so long as they are alive; they refuse to take any dye unless they are previously killed; and therefore in ordinarily stained specimens the bacilli are stained, but the spores remain without colour. The ordinary method of drying does not kill them, and it is accordingly necessary first to overheat the cover-glass specimen sufficiently. This is effected by drawing the cover-glass specimen several times (ten to twenty times) through the gas flame, or by holding it a good way above a small flame for several seconds up to half a minute, till the cover-glass is so hot that touching it with the palm of the hand would produce an

unpleasant sensation; but of course the heating must stop short of charring. The specimen is then put on a carbol fuchsin solution and kept there at 37° C. for one half to one hour, or the watch-glass containing the carbol fuchsin solution and the cover-glass specimen is covered with another watch-glass, and is then rapidly heated over a flame till boiling point of the fuchsin is reached; then the specimen is washed in water—sometimes it is necessary to dip it once or twice in alcohol—and placed in methyl blue aniline water for twenty to forty seconds, then washed in water, dried, and mounted in balsam: the bacilli and their threads are blue, the spores, both those in the bacilli as also those that are free, are bright red. The same effect is produced if, instead of carbol fuchsin, a 2 per cent. watery solution of rubin is used, the rubin being heated over the flame till the first bubbles appear, washed in water, then quickly stained in methyl blue. Another method is this: treat the cover-glass specimen as above, i.e. stain in fuchsin, then pass through nitric acid (1 : 10), wash in water, stain in methyl blue, wash in water; dry, and mount; the spores are red, the bacilli blue. The spores appear deeply stained also in specimens stained with one dye only, provided the specimen has been previously well overheated; and in such a case, while the colour can be easily removed from the bacilli either completely or brought down to a faint tint by washing the specimen in alcohol after the staining, the spores retain the colour with great persistency.

Another useful method of staining bacteria in a thin film, i.e. in a cover-glass specimen, is the Warsowa method. This method gives very good results, and shows the bacteria and other tissue elements in their natural shape and size, which occasionally is not the case in the dried cover-glass specimens, e.g. if the film dried is not thin. The method is this: A thin film of the material is spread out on a cover-glass and then immediately placed film downwards on a saturated solution of mercuric chloride, contained in a watch-glass, and kept there for five minutes, next over alcohol for ten minutes, then placed over the stain, film downwards; after it is well and sufficiently stained, it is washed in water, then placed in alcohol for a few minutes, next placed over clove oil for a few seconds, then over xylol, also only for a few seconds; the xylol is drained off with blotting-paper and the cover-glass specimen is mounted in balsam. If the right amount of washing in alcohol is reached, this method yields excellent results, and shows the bacteria and tissue elements more in their natural shape, size, and outline than the Weigert-Koch method of drying: the drawback is that the stain is not easily removed from the tissue groundwork, and that this ground substance, owing to its being coagulated by the mercuric chloride, takes easily the stain. If the film placed on the cover-glass is not thin, it is liable to come off in the mercuric solution and in the alcohol, but if it is applied thin it becomes fixed by the mercuric solution as a thin coagulated opaque layer, which the subsequent washings in the various fluids do not easily remove. (For Löffler's method of first treating cover-glass specimens with ink (tannin and ferrous sulphate) and then staining with aniline water methyl violet, methyl blue, or carbol fuchsin in order to stain the flagella, see 'Centralbl. f. Bact. und Parasit.' IV. Bd. No. 8, 9, p. 209.)

The methods of preparing thin sections of tissues and the methods of mounting them for permanent specimens are not subjects to be specially described here, since these are matters which concern the general methods used in normal and pathological histology. It is sufficient to say that any means by which thin sections of a fresh or hardened tissue are obtainable are good: whether this be by a freezing microtome of one kind or another, whether by ice freezing or other freezing makes no sort of difference. Cathcart's ether freezing microtome or Roy's ether freezing microtome, or the Cambridge microtome for ribbon cutting, answer every purpose. But we should recommend that the hardening of the tissues (always in small bits) be carried out in Müller's fluid or in alcohol, though the former is in many cases preferable. In the former the bits of tissue remain for two to three weeks, then are transferred to methylated spirit 66 per cent., and after a week to pure methylated spirit. What we are here concerned with are the methods of staining the sections. If these are made of fresh tissues, they must be put into salt solution, not water; but if previously hardened, directly into water. In both instances, before being placed into the staining fluid they are transferred to methylated alcohol. In the case of the sections from fresh tissues, this transferring them from the salt solution into alcohol is not without risk of their shrivelling up too much, and for this purpose they ought to be spread out on a section-lifter in the salt solution, and then, before floating them into the alcohol, the salt solution must be removed as much as possible, so that the edges here and there become fixed on the section-lifter; then a few drops of alcohol are dropped on to the section to coagulate and fix, as it were, the elements, so that when finally floated into the alcohol the tendency of the section to shrink is greatly reduced.

For staining of sections the same dyes are used as are mentioned above, and as regards simple staining very little is necessary to add to what has been said on a former page of

staining of cover-glass specimens. The sections are floated by means of the section-lifter from the methylated alcohol into the dye and left there, according to the character of the dye, the thickness of the section, and the nature of the bacteria, from ten or thirty minutes to an hour and more: in some cases it is necessary to leave them twenty-four hours. Also here staining at 35°-37° C. is more rapid than at ordinary temperature. Then the sections are washed in water and passed into methylated alcohol; there they remain for some minutes (two to ten) according to the amount of dye that it is required to remove from the section. In the case of carbol methyl blue, methyl blue aniline water, vesuvin, the treatment with alcohol must be carried out more carefully than after staining with gentian violet aniline water, carbol fuchsin, rubin, Humboldt's blue and violet, and methyl violet, since in the former the dye is more easily removed by the alcohol than in the latter. It is therefore advisable when using Spiller's purple, methyl blue, or vesuvin to rather over-stain the sections in the first instance; and when using gentian violet, methyl violet, carbol fuchsin, or Humboldt's blue, to leave the sections in methylated alcohol for a considerable time. From the methylated alcohol the sections are transferred to absolute alcohol for a minute or so in order to completely dehydrate them. This alcohol also removes some of the dye; but if this be not desirable, it is a good plan previously to colour the absolute alcohol with a droplet of the dye. From absolute alcohol the sections are passed through oil of cloves for a few seconds; this has great power to withdraw the stain from the sections, and it is therefore good to stain this previously with a droplet of the dye. Finally, the sections are placed in xylol for a few seconds to fix the dye, and then mounted in balsam.

For double staining of sections the same principles obtain as were mentioned above in connection with the cover-glass specimens. We will here give a few illustrations: (1) Double staining of sections with Weigert's gentian violet and eosin, rubin and methyl blue, Weigert's methyl blue and eosin, carbol fuchsin and methyl blue, such as can be practised with sections through tissues of anthrax, septicæmia, pyæmia, diphtheria, typhoid fever, erysipelas, pneumonia, swine fever and swine erysipelas, fowl cholera and fowl enteritis; the sections are well stained in the first dye, washed in water, then placed in the second dye. This second staining must be carried out very carefully in order not to lose the first dye altogether; then washed in water, methylated alcohol, absolute alcohol, oil of cloves, and xylol. (2) In the case of tubercle or leprosy the sections are stained well in carbol fuchsin, then washed a few seconds in nitric acid (1 : 3 to 1 : 10), then in water, then placed in Weigert's methyl blue or vesuvin, then washed in water, &c.

(3) Gram's Method.—The sections are taken from alcohol and placed in Weigert's gentian violet or methyl blue aniline water, then after being well stained, are passed through alcohol, transferred to a solution of iodide of potassium 2 grammes, water 300 c.c., iodine 1 gramme, wherein they remain one to three minutes, then they are washed in alcohol till apparently almost decolourised; then the sections are transferred to alcoholic solution of eosin for from a few seconds to half a minute, washed in water, passed through alcohol, oil of cloves, and xylol, and mounted in balsam; the bacteria are of a deep blue colour, the tissue is pink. Many bacteria retain the first dye when passed through the iodine solution; others, however, become thereby decolourised; of course in these latter cases the method is useless—as with the typhoid bacilli, cholera spirilla, and others.

(4) Lustgarten's Method for Staining the Syphilis Bacillus in Sections.—Weigert's gentian violet, in which the sections remain twelve to twenty-four hours at ordinary temperature, and two to three hours at 37° C.; wash in alcohol, then pass them for about ten seconds through 7.5 per cent. solution of permanganate of potassium; wash for a moment in dilute solution of sulphurous acid in water, then wash well in water; if not decolourised, repeat the washing in the permanganate and sulphurous acid solution, dehydrate, clarify, and mount finally in balsam. Tubercle bacilli, leprosy bacilli, syphilis bacilli, and the smegma bacilli retain the dye even after the permanganate; other bacteria become thereby decolourised. De Giacomini, Gottstein, Doutrelepont, and Schütz have modified this method by using, instead of the permanganate and sulphurous acid solution, either dilute solution of perchloride of iron or dilute nitric acid (1 : 15).

(5) Löffler's Method for Glanders Bacillus.—The sections or cover-glass specimens are well stained (from thirty minutes to twenty-four hours) in watery solution of methyl blue (2 per cent.) to which in equal parts liquor potassæ (1 in 10,000) is added, or: 1 c.c. concentrated alcoholic methyl blue solution, 200 c.c. dist. water, two to four drops of a 10 per cent. solution of potassic hydrate; then wash in water acidulated slightly with acetic acid, then pass through alcohol (sections), oil of cloves, and xylol, and mount in Canada balsam. In the case of cover-glass specimens, after washing in acidulated water,

wash in distilled water, then dry and mount in balsam. This method of staining is not only useful for glanders bacilli, but for many other bacteria.

There is sometimes great difficulty in staining the bacteria in sections of hardened tissue, far greater than in the cover-glass specimens, and it is therefore important in some cases to stain in a prolonged way, and to be careful in the subsequent washings with alcohol. In many instances cover-glass specimens, made of the fresh tissue, present no difficulty in easily and rapidly staining the bacteria, while after hardening, the demonstration of the same bacteria seems to be very difficult and only possible after prolonged staining, and the conclusion seems justified that the hardening has some deleterious effect; this, for instance, is the case in swine erysipelas, in typhoid fever, in pneumonia, in fowl enteritis, and in glanders. As a rule sections made of tissues first hardened in Müller's fluid are easier to stain for bacteria than if hardened directly in alcohol. In some cases the difference is very striking, the bacteria in tissues hardened in Müller's fluid being easy to stain (tubercle, swine fever, fowl enteritis, grouse disease), while from alcohol the same cannot by any means be said.

CHAPTER IV

STUDY OF THE BIOLOGICAL CHARACTERS

IN the whole range of bacteriological studies there has been no greater advance made within a short space of time than in the introduction by Koch of the methods of cultivating bacteria on solid media. These methods, now universally adopted, mark a definite epoch. Before Koch, bacteria had been studied largely and successfully by a large number of observers, and their nature, morphology, and biology in many cases very accurately determined: we need only mention Pasteur, Davaine, De Bary, von Naegeli, F. Cohn, E. Klebs, Miquel, R. Maddox, Lister, Billroth, Tyndall, W. Roberts, Heiberg, Zopf, Prazmowski, van Tieghem, Fitz, Weigert, and many others. But the results obtained by these observers, although remarkable, and of fundamental importance, cannot—we say it with the greatest deference to the acumen and thoroughness of these observers—for a moment compare with the results achieved by Koch by his exact methods of cultivation of bacteria on solid media and by his and Weigert's method of staining bacteria. More than that, by the introduction of these exact methods of Koch there has suddenly set in a flow of researches and discoveries which, like a snowball in rolling from the top of a snow hill, is constantly increasing in size, and its advent is felt in wider and wider circles. While before Koch bacteria had been chiefly studied when grown in fluid media, and thereby the right path from error only with difficulty avoided, since Koch these studies are comparatively easy and tolerably exact, though even in this it requires care, attention, and a sufficient amount of preliminary knowledge in bacteriology and pathology. There seems to be an idea abroad, and of which one hears only too often and sees its mischievous results continually, that for bacteriological and pathological studies and research all that is required is a platinum needle for inoculation, sterile nutrient gelatine in test-tubes (which can be easily bought by the dozen), a microscope with oil immersion, a microtome for sections (sections can be of course cut by any dexterous assistant), one or two aniline dyes (also easily bought ready made), and a book on bacteriology. Well, this is not bacteriological or pathological study and research any more than the possession of flasks, burettes, test-tubes, and reagents in bottles well labelled, and a balance and a book on chemistry, represent chemical study and research.

In the study of the biological characters of bacteria, the study of the fresh and living bacteria occupies, or ought to occupy, the foremost place; the

aspect and characters in the living state, as seen under the microscope in a drop of salt solution or watched in the 'suspended drop,' are the first step. We have already described and spoken of these on a former occasion, as also of their characters in stained specimens. The cultivation and study of bacteria on artificial media is the next step.

Culture Media: A. Broth.—All bacteria with few exceptions grow well in beef broth, neutral or, better, faintly alkaline, also in chicken broth and in pork broth. Great differences are shown by the different bacteria when growing in this medium. As mentioned above, some few do not grow in this medium when kept at temperatures above 25° C., although most species grow more abundantly at temperatures between 35° and 38° C. Most of them produce a uniform turbidity, some quickly-growing ones in twenty-four hours, others later, the amount of turbidity differing, however, considerably; in some it makes the broth thick and uniform, in others the turbidity is slight, and in others the broth contains smaller or larger flaky or floccular granules. In some, after one, two, to three days' growth a pellicle appears on the surface; this pellicle in some cases is a continuous membrane, in others more scaly; in some it is thick and smooth, white or slightly yellow, in others it is folded, white or slightly brown; in some the pellicle is easily broken up on shaking, in others it is tenacious and remains complete; in some it easily falls to the bottom of the fluid, in others it remains floating. Micrococci, bacilli, and spirilla are capable of forming such pellicles. In some the height of growth—i.e. the maximum amount of turbidity of the broth—is reached after a few days, in others not until after ten days to a fortnight. During the growth there appears in most a whitish or greyish or more or less coloured precipitate, which increases in amount; this precipitate is either small in amount or bulky, it is either powdery or in some cases flaky, or again in others tenacious and viscid. In some instances the broth, the height of growth having been reached, clears again; in this also great differences obtain—for in some the clearing up proceeds uniformly and gradually from the surface, in others it proceeds in distinct layers; in some species of bacilli the broth does not clear at all; in some species the broth after a day or two is tolerably limpid, although copious pellicle-formation goes on on the surface (potato bacillus, scurf bacillus, and others). If this pellicle by shaking be brought to the bottom a new pellicle is formed. In some species the broth never becomes turbid, but the growth begins at the bottom of the fluid in the shape of grey nebulous flocculi which gradually increase in bulk—e.g. bacillus anthracis, bacillus filamentosus.

From this it will be seen that the character of the growth in broth is in the case of the different species most varied, and the aspect and progress of the growth in many cases very characteristic.

Broth is best used as beef broth in sterile flasks or test-tubes, plugged with sterile cotton wool; the broth is made neutral or, better still, faintly alkaline. Acidity or strong alkalinity of the broth is injurious to the growth.

Instead of broth there can be used: (a) beef infusion; (b) one small tin of Brand's essence, or meat extract, 500 c.c. dist. water, peptone (1 per cent.); (c) broth with peptone (1 per cent.); (d) hydrocele fluid; (e) blood serum; (f) alkali albumen; (g) broth and glycerine (6 per cent.); (h) beef broth in test-tubes to which has been added a small piece of boiled white of egg.

For some non-pathogenic bacteria other fluids can be used instead of broth which contain nitrogen only as simple nitrogenous compounds—e.g. tartrate of ammonium, or even carbonate of ammonium, urea, or uric acid—although many non-pathogenic, and all pathogenic bacteria require, as stated above, some kind of albumen. Those albumen-free fluids are principally composed of salts (potassium and calcium salts, phosphates) and tartrate of ammonium—Pasteur's fluid and Cohn's fluid.

Other fluids similar in composition (Meyer's and others) are used according to the special requirements of specific fermentative actions shown by the particular species of bacteria—e.g. nitrifying bacteria, acetic acid-producing bacteria.

B. Nutrient Gelatine.—Gelatine as a medium for the study of the growth of fungi and microbes was first used by Brefeld and by Klebs, but not until Koch showed its immense value was gelatine of peculiar composition universally used and with such signal success. The nutrient gelatine is used in test-tubes (a) as beef infusion gelatine peptone, and (b) as beef broth gelatine peptone. The gelatine is added to beef infusion or beef broth, as the case may be, besides 1 per cent. of peptone and 1 per cent. of sodium chloride.

Instead of either beef infusion or broth as the solvent medium, there can be used with the same success: (a) Brand's essence, one small tin, in 500 c.c. water; or (b) meat extract dissolved in water on the same scale.

To the nutrient gelatine special substances can be added as the special cases may require—e.g. grape sugar, lactic sugar, cane sugar, whey.

The writer uses generally beef broth gelatine peptone, or Brand's extract solution, or gelatine peptone, faintly alkaline. The gelatine is used from 2 per cent. to 10 per cent. Two per cent. gelatine mixture sets well and remains solid at 20° C.; gelatine of 10 per cent. remains solid at 22° C. to 23° C., and even a little higher. In the test-tubes the gelatine, if used for stab cultures, is set straight and upright, for surface or streak cultures it is set slanting; for the latter purpose the writer uses special trays made of wood or tin and offering a slanting surface, so that about twenty-four tubes containing the liquefied gelatine are at the same time placed side by side on the tray, and the gelatine, when set, offers the largest surface without being too near the cotton-wool plug.

For studying the growth of bacteria on the surface or in the depth, the liquefied gelatine is set in a sterile watch-glass, or in a flat glass capsule, or on a glass plate, or in a test-tube, or in other vessels; the gelatine is then inoculated with a capillary glass pipette or a platinum wire, previously dipped in the material containing the bacteria, either by a stab, or at a point, or at several points, or in a line or lines, i.e. streak; and if the gelatine is kept covered up, so as to prevent accidental germs entering from the air, the growth of the inoculated bacteria can be easily examined from time to time under a magnifying glass or under a simple microscope. Because development on the solid nutrient gelatine takes place only at, and is limited to, the spot of inoculation, or the spot where a bacterium had been present or deposited, several different species can grow side by side, and their differences be watched; and it is this simple fact which enabled Koch to isolate and keep isolated different species of bacteria growing simultaneously in a limited area. By this means it is not difficult at once to recognise that a particular material with which the gelatine had been inoculated contained several different species of bacteria, while each can then be isolated by sub-cultures separately. This is an advantage which cultures in fluid media do not offer; hence, if originally into a fluid medium several species are introduced, these all multiplying in the fluid, a mixture of the different crops is the result, all intermingled with each other. By using solid media, not only can the growth of individual species be watched and controlled better and with more exactness, but a separation and isolation from an original mixture of the different species composing it can be achieved with facility. The characters presented by the different species when growing on and in nutrient gelatine are most varied, and for many of them very characteristic. We will here give some of these characters. First and foremost is the power that some bacteria (micrococci, bacilli and spirilla) possess of liquefying the nutrient gelatine in the course of their growth and multiplication, while others do not liquefy it. In stab cultures the liquefaction proceeds from the surface or upper part of the stab—i.e. the channel of inoculation—towards the depth and towards the sides, so that the liquefied gelatine occupies and fills as a rule a more or less funnel-shaped space in the gelatine, the broader basis of the funnel being on the surface; as growth and liquefaction proceed the funnel-shaped space becomes cylindrical. In streak cultures the liquefied gelatine flows down to the bottom of the tube, and the gelatine on which the streak growth takes place becomes transformed into a broad and deep groove, the diameter of which depends of course on the rapidity of the growth and the rapidity of the liquefaction. There exist marked differences (a) as regards the rapidity of liquefaction independent of the amount of the growth, for some liquefy with great rapidity and early, others only very slowly and not before some days have passed; (b) as regards the character of the liquefied gelatine; in some this is perfectly limpid, the growth settling in the depth of the liquefied gelatine (bacillus anthracis, spirillum cholerae), or it remains on the surface as a scum (bacillus filamentosus), or both (bacillus subtilis); in other cases the liquefied gelatine is not clear, but either uniformly turbid (spirillum Finkler, bacillus Jequirity, and some species of staphylococcus liquescens) or it contains granules and flakes (several species of staphylococcus liquescens, bacillus of potato); and (c) as regards the density of the liquefied gelatine; in some the liquefied gelatine is thin and fluid (many species of bacillus subtilis, potato bacillus, spirillum of Finkler and cholera, staphylococcus aureus liquescens); in others the liquefied gelatine is like thick syrup and scanty (some species of staphylococci, bacillus fluorescens of veal pie—bacillus of swine erysipelas).

Those species which do not liquefy gelatine, and their number is legion, show likewise marked differences one from another in stab and streak cultures. As to stab culture, the channel of inoculation is after a few days a grey or white or brownish line made up of isolated or more or less confluent droplets, in some very minute, only visible under a lens, in others large enough to be seen with the unaided eye; in some again the channel of inoculation is a uniform broad growth, in others it remains always very thin; in some species

the droplets forming the channel of inoculation are white or grey in reflected, and light yellow-brown in transmitted light, while in others they are conspicuously white in reflected, dark brown in transmitted light. Again, the surface of the stab contains no growth, or only very little in the shape of a few superficial minute granules or droplets (e.g. some species of streptococci), or the surface of the stab is covered with a convex, more or less well-defined circular disc or button (bacillus of Friedländer, some species of staphylococcus), and this again is either white or grey or of a pinkish or slightly yellowish colour; or the surface of the stab becomes covered by a flat thin filmy disc, small in some species, rapidly spreading in others, circular or only slightly angular in some, very markedly crenate and serrated in others; in some limited only to the surface of the stab, not larger than one to two millimetres in diameter, in others soon covering the whole surface of the gelatine.

Equally great are the differences in streak culture—i.e. when the growth takes place in a line drawn longitudinally on the surface of the gelatine (set obliquely in a test-tube) by a capillary pipette or platinum wire. First: the rapidity with which the growth appears in this streak differs considerably in the different species; in some after only twenty-four hours the line of inoculation is already marked as a greyish or whitish streak; in others the growth is not noticeable before two to three days. Then the manner and rapidity with which the streak grows in breadth; in some the growth does not spread much in breadth, in others it rapidly broadens into a band; in some the streak is uniform and homogeneous, in others it is uniform but slightly granular, in others, when seen under a glass, it is made up of minute droplets, more or less separate, but in close contact and even confluent; the margin of the streak is either smooth, and more or less straight, in others it is knobbed, serrated, or bent, with numerous shorter or longer filamentous projections. Then as to the colour and aspect of the streak; in some the streak is greyish, in others white, and in others more or less coloured (pinkish, yellowish, golden brown, greenish, vermillion, &c.) in reflected light; in transmitted light the streak is greyish or brownish, uniform in thickness, or thicker in the centre than at the margin, or the reverse; in some the streak is terraced, in others of uniform thickness; in some species the margin shoots out in thin irregular plaques, in others in fan-shaped patches. In some the growth is moist, in others it looks dry; in some species the growth is extremely limited while on the surface but copious in the depth of the gelatine, or *vice versa*, the former growing much better under access of air, the latter under deficient supply of air—aërobic and anaërobic.

As a useful means for study, and interesting for differentiation, is the method of adding to the nutritive gelatine traces of an aniline dye, sufficient to stain the gelatine—too much aniline dye acts more or less poisonously on the bacteria and prevents their growth. But when certain dyes are added in very minute quantity, a curious result is obtained by the different bacteria when growing in or on such gelatine: while some refuse to grow even when a mere trace of the dye has been added, others grow slightly, and others very well. Amongst the latter the gelatine will be seen in some cases to become gradually decolourised, beginning from the growth and spreading further and further; the growth itself, at first stained, gradually losing also the colour, beginning from the part nearest to the surface. Great differences in the time and manner are shown in these respects by different species of bacteria. The gradual discolouration of the gelatine, beginning at the growth and gradually spreading through the gelatine, indicates that certain diffusible chemical substances pass from the growth into the gelatine.

Then as to the nature and aspect of the gelatine on which the streak grows; in some this gelatine remains perfectly limpid and unaltered, in others it becomes opaque, while in some it becomes gradually coloured greenish or bluish though it remains limpid.

Another useful method is that of Buchner, viz. adding to the gelatine sufficient litmus to stain it; the change of this substance by the growth is an interesting index of the character of the bacteria.

All these are points which in themselves are sometimes small and insignificant, but, occurring with constancy, and taken with the appearances presented by the growth in other media, are reliable distinguishing characters. (See the illustrations of stab and streak cultures.)

PLATE CULTIVATIONS

The most significant characters presented by bacteria are those shown by them when growing in plate cultivations. Owing to the fact that when a particular bacterium is planted into (or finds itself at) a particular spot of solidified nutrient gelatine or agar mixture, it forms a circumscribed growth

at that spot—viz. a colony—it is possible to separate different species of bacteria from any mixture, care being taken to first distribute by shaking a limited number of the bacteria in a comparatively large amount of liquefied nutrient gelatine or agar, and then to pour this out into, and allow it to set in, a protected glass vessel, plate, or the like. As the material sets, it fixes, as it were, the bacteria (that have been introduced) in isolated fashion, and each of them (being few and far between) by multiplication gives rise to a pure colony, sufficiently separated from others by material not containing any bacteria, and therefore capable of being easily studied during their various phases of growth by the unaided eye or by a lens. Their mode and rapidity of increase, their aspect, colour, outline, &c., can be watched, and from them by inoculation of new media new pure sub-cultures can be established. All one has to do is, then, to introduce a limited number of bacteria (with a capillary pipette or a platinum wire) from a given tissue, blood, or bacterial culture or mixture into a test-tube containing nutrient gelatine (5–8 c.c.), then liquefy this in warm water, then shake the gelatine carefully so as uniformly to distribute the introduced bacteria, and then pour this out on a flat sterile glass dish covered up with a sterile glass plate or glass dish,¹ or to pour it out on sterile glass plates, watch-glasses, or other vessels, provided that the gelatine forms a thin layer; then the gelatine is allowed to rapidly set, by either leaving it to itself in a cool place or by placing the vessel on cold water or ice (according to the temperature of the air). As the colonies make their appearance in the plates in the course of a few days, the plates being kept in the incubator at 19–21° C., if necessary in a moist chamber—e.g. under a glass globe containing moist filter paper—the examination of them can be easily effected under a magnifying lens or with a low power under the microscope. If, however, the material from which the plate cultivation is to be made originally contains large numbers of the same or of different bacteria—e.g. intestinal evacuations, sputum, anthrax blood, bacterial culture fluids, &c.—then the direct inoculation of the gelatine with however small a trace of the material brings forth in the plate too large a number of colonies. In these cases it is necessary first to dilute the material, placing a trace of it into a few cubic centimetres of sterile salt solution or broth; shake this up, and then inoculate from this with a small quantity—with the platinum wire or platinum hook—the tube of nutrient gelatine, which, liquefied and shaken up, is used for the plate cultivations. In order to have a successful plate cultivation the number of colonies in a plate must be limited, the colonies must be sufficiently isolated and, if possible, in a single layer. Of course during all these manipulations accidental contamination with other bacteria (air, unclean instruments, or hands) must be avoided as much as possible, rapid and precise working with sterile apparatus being essential for success. (Agar plates, *see* p. 41.)

TUBE-PLATE CULTIVATION

Another kind of plate cultivations, yielding very good results, and giving excellent permanent specimens of plate cultivations, is obtained in the following manner. Everyone who has practised the above method (Koch's) of plate cultivations in a laboratory in a large city knows that accidental introductions of air-germs, particularly moulds, &c., cannot be wholly avoided, and therefore many such plates become by-and-by spoiled, apart from those in which liquefying organisms develop—for in this case all plates become

¹ The writer had described and figured this glass dish in 1886, long before Petri, to whom generally this form of plate cultivation is ascribed. (*See Micro-organisms and Disease*, 3rd edition.)

useless as plates—as soon as the liquefaction has reached a certain degree. But in the case of non-liquefying bacteria excellent permanent plate cultivations are obtained by spreading out by means of the sterile platinum wire or platinum loop or hook (*Oese*, Germ.) a trace of the material (pure or after dilution as the case may require) over the whole surface of the gelatine or agar, set slantingly with large surface in a test-tube—that is to say, rubbing it over the surface; during this operation the tube is held inverted, is then plugged again with its sterile cotton-wool plug, and placed in the incubator. After a few to several days a limited number of colonies are noticed on the surface of the gelatine or agar, which can be easily studied with a magnifying glass or used for sub-cultures. Such a tube-plate cultivation can be kept uncontaminated and in a perfect state for an indefinite time, provided the gelatine or agar is kept from drying up by covering it with a well-fitting india-rubber cap, or, better, with gutta-percha paper melted to the glass wall of the neck of the test-tube.

Another mode of plate cultivation is that described by Esmarch, and known as 'roll-plate.' The procedure is here the same as in Koch's method, but after inoculation, liquefaction, and shaking up of the gelatine contained in a test-tube plugged with sterile plug, this gelatine is not poured out on plates, but having been covered with a tight-fitting india-rubber cap is rolled, while holding it horizontally, in cold water, till all the gelatine has well set in a thin layer on the inside of the glass tube. The tube is then wiped on the outside and placed in the incubator. Such a roll-plate retains its character as a plate cultivation intact for an indefinite period provided no liquefying colonies be present. By carefully twisting the plug this can be easily removed and sub-cultures be made from the colonies. The examination with magnifying lens is of course also easily carried out, even under the microscope, the tube being placed horizontally in a sort of cradle across the stage of the single microscope.

From what has been said above it must be evident that plate cultivations are indispensable means of study, since by them not only can the different species be readily and exactly studied, but they can be isolated with precision and pure cultivations from them be obtained; besides, by these plate cultivations the purity or impurity of different materials and cultures, and the nature and number of the different species, can be accurately determined (*see* below).

It must also be obvious that however pure—i.e. containing a single species—a given material or culture in broth or other fluids or in stab or streak culture may appear under the microscope or to the naked eye inspection or to a magnifying lens, there is no guarantee that it is really so unless it can be shown that the colonies derived from it in plate cultivations are all the same as regards rapidity of growth, size, aspect, outline, colour, and general nature. And it follows from this that, with justice, the study by plate cultivation (not only in gelatine but also in other solid media, agar, serum, &c.) is considered one of the most essential steps in the study of bacteria, and unless the bacteria have been isolated by plate cultivation of one kind or another no guarantee can be said to exist as to their purity. (*See* illustrations of plate cultivations.)

Now, the different species of bacteria differ in many ways one from another in plate cultivation: e.g. as regards (1) the rapidity with which the colonies grow in a plate after inoculation; (2) their appearance when first noticed; (3) the rapidity and amount of their increase in a given time; (4) the period at which the colonies reach their maximum development, provided they do not liquefy the gelatine; (5) whether or not the colonies liquefy the gelatine; (6) the manner in which the liquefaction takes place, whether clear or turbid, regularly outlined or irregularly; (7) the outline of the colonies; (8) the breadth and thickness of the colonies; (9) their general aspect, whether grey or white or coloured, moist-looking or dry, homogeneous or granular, uniform in thickness or not; (10) whether they show a difference in their size

are very similar in and on gelatine and agar, but yet there exist definite though small differences. There exist a number of various species of motile bacilli which have about the same size and general morphological characters, including formation of leptothrix and spores, and which also in plate cultivation, in stab and streak cultures on gelatine, and in broth cultures are very similar; nevertheless there are in these media slight differences that are easily overlooked, but on potato the differences are striking. So also the typhoid bacillus; the writer is acquainted with, and has in his possession, a species of bacillus which was isolated from the mesenteric glands of a child dead of summer diarrhoea, and which it is impossible to distinguish from the typhoid bacillus in gelatine cultures, on agar, and in broth, but on potato the difference is striking. These examples could be easily multiplied.

In studying those species of bacteria which are connected with specific disease, besides the above points to be attended to there is one which ought not to be lost sight of—that is, while demonstrating the existence of the particular species of bacteria by cultivation from the blood or tissues, these bacteria must be demonstrated also by stained cover-glass specimens of the fresh morbid tissues or fresh blood, as the case may be. There may be difficulties in this—as, for instance, if the number of the specific bacteria present in those tissues and blood be too limited for showing them in a small particle of the tissue or a droplet of the blood used for such cover-glass specimens—but such cases are only few; in most instances they can be shown to occur in such specimens, even if sometimes several such specimens may be needed to demonstrate the bacteria. Plate cultivations and stab and streak cultivations yield, as matter of course, much more positive results, even where the number of bacteria appears small in the tissues when examined under the microscope.

Classification.—Owing to the rapid increase and perfection of the methods above described, the number of different species of bacteria already well differentiated has become very great, and it is steadily on the increase. Numbers of species of micrococci, which in former years were all considered as one, have become a whole family of species. We need only refer to staphylococcus and to bacillus subtilis. Staphylococcus is now merely a generic name, and there are a very large number of species of staphylococci known, well differentiated one from another; nay, more, staphylococcus pyogenes was once the name of a species, but it is now well known that this is a group of species. The name of staphylococcus pyogenes aureus is still by many given to one species; but the writer is sure, from definite and systematic observations on this subject, that this name comprises also several species. Streptococcus is another such name, as was mentioned on a former page; it indicates the power of micrococci to form chains; there are a considerable number of different species of micrococci which belong to this group; streptococcus pyogenes of Ogston and Rosenbach, until a few years back, was regarded as a single species of streptococcus, but this also comprises a number of different species. Bacillus subtilis, formerly the name of a species, is really the name of a family of similar species. In choosing the name for any one species there does not, at present, exist an understanding on the part of bacteriologists about any standard character, but a name is chosen by one bacteriologist according to one prominent character, by another according to another; hence there exists a tendency to name the same species by different appellations. This is particularly and strikingly illustrated when one looks through the literature that has accumulated in recent years on water bacteria and other saprophytic bacteria; it is less so in the case of bacteria connected with disease, for in these cases the name is the name of a class, and to it is added the name of the disease with which it is associated—e.g. bacillus cholerae gallinarum, bacillus anthracis, bacillus tuberculosis, bacillus mallei; but occasionally also in these groups one meets with appellations which are most misleading—e.g. bacillus cuniculicida, meaning by this a particular bacillus (formerly called a bacterium) which is fatal to rabbits, and discovered by Koch. As, however, there exist a host of different species of bacilli which are cuniculicida, it would be better to call the former *B. cuniculicida* Koch, and to distinguish it from other *B. cuniculicida*. Or take the case of

spirillum sputigenum, the name given to *spirilla* occurring in the fluid of the mouth, it is clear that this is not one but several species, and that when their characters are better studied other names will have to be added.

The best account of this constantly and rapidly growing subject has been published by James Eisenberg, 'Bacteriologische Diagnostik,' 3rd edition, 1891.

HEAT

In a former chapter we have stated the important differences that exist as regards the influence of different temperatures on the different species of bacteria; amongst other points it was mentioned that while in most cases certain higher temperatures (80–88° C.) favour, lower temperatures (16–17° C.) and also too high temperatures (42–45° C.) diminish, the power of multiplication. In some few species—e.g. *bacillus thermophilus*—extremely high temperatures (65–70° C.) are still appropriate. We further mentioned that in some cases the bacteria do not grow at temperatures above 25° C., while others do not grow at temperatures below 22° C. Now in all cases of the multiplication of bacteria heat is evolved; in those that grow rapidly at ordinary temperatures (16–22° C.), amongst them the typical putrefactive organisms or saprophytes, the amount of heat evolved is considerable and easily noticeable; and there is a fruitful field of research opened in this respect—viz. to ascertain accurately the manner and amount of this heat production in the different species. As we shall point out later, Birch-Hirschfeld has ascertained this evolution of heat also for tubercle bacilli. It has been stated that the febrile rise of temperature in the body in the case of rapidly growing pathogenic bacteria is probably due to such a cause; but this is not based on any direct evidence; on the contrary, it is more probable that where in a disease rise or fall of temperature takes place, it is due to a direct action of the chemical products of the bacteria on the heat-regulating mechanism in the body. Take, for instance, the case of anthrax and some cases of septicæmia; here the rise of temperature is greater in the initial stages of the multiplication than in the later stages when the bacteria have become more numerous—in fact, towards the stages when they have become very numerous the temperature of the body rapidly falls. An apparently opposite result is noticed in the case of the *spirilla* of relapsing fever and the plasmodia of malarial fever, for here the onset of the fever-rise is coincident with, or even follows the immigration into the blood of the parasites. We say apparently, because there is ample evidence that rise of temperature has not its cause in the blood but in the tissues. But the general observations of artificial cultures of various species of saprophytes, as well as of pathogenic bacteria, prove that under these conditions, and growing at ordinary temperatures, there is often an appreciable rise of temperature noticeable. This rise is obviously the result of oxidation and disintegration processes going on in the nutrient medium in connection with the multiplication of bacteria. The writer has noticed in a general way, as was *a priori* to be expected, that in the case of motile bacilli and motile *spirilla* this rise is more appreciable, *cæteris paribus*, than in other not motile bacilli; and the same is the case in those species which grow best on the surface of the nutritive gelatine as compared with those that grow better deep in the fluid. On the detrimental influence of higher temperatures see the chapter on Disinfection.

LIGHT

Diffused light has a small, direct sunlight has a powerful, inimical influence—both inhibitory and germicidal—on the growth and life of many bacteria.

The observations of Duclaux on this point comprise many species of bacteria, and those of Dr. Downes have added much valuable knowledge to this subject. Dr. Downes finds that the influence of direct sunlight has a very marked germicidal effect on various microbes—e.g. anthrax bacilli, sporeless and spore-bearing, and tubercle bacilli.

But while in many cases the direct sunlight has an inimical influence, there exist certain species of bacteria, which in their growth and development evolve phosphorescent light.

Engelmann studied a species of bacillus (*bacillus photometricus*) which, owing to its great sensitiveness to light, he used to accurately measure light.

As regards the phosphorescent bacteria, the following observations have been made. It has been known for a long series of years that decomposing wood and decomposing sea fish occasionally become phosphorescent, but Pflüger was the first to show that the phosphorescence in the case of fish was due to the growth thereon of bacteria. Recent researches by Ludwig, Fischer, and particularly Lehmann ('*Centralbl. f. Bacterien.-u. Parasitenk.*' V. 24) showed that the phosphorescence of *bacillus phosphorescens* Fischer is a result of its growth and multiplication under certain conditions, but not under others. This bacterium growing in gelatine, broth, boiled or raw meat or fish, potato, &c., under access of oxygen, produces phosphorescence; when growing in the depth phosphorescence is absent; in CO₂ (Fränkel) the microbe grows but does not show phosphorescence. Whether the phosphorescent condition of the nutritive medium is due to special chemical products, to a mucous capsule, or to the protoplasm of the bacteria is not decided. (See also on phosphorescent bacteria, Oscar Katz, '*Centralbl. f. Bact. u. Parasit.*' IX. No. 5.)

For highly interesting observations on infection of various sea animals (invertebrates) in the living state with phosphorescent bacteria, see M. Girard, '*Comptes Rendus*,' Sept. 23, 1889.

PIGMENT

This is a remarkable and interesting phenomenon exhibited by a great many species of bacteria when growing in one or another medium. While some possess in this respect a highly specialised character, capable of forming a well-defined pigment in most conditions of growth, others assume a pigmented aspect only while growing on one particular medium. Of the former class are those well known since Schröter and Cohn as the 'pigment bacteria'—e.g. *bacillus prodigiosus*, *micrococcus aurantiacus*, *micrococcus chlorinus*, *bacillus ruber*, *bacillus pyocyaneus* of green pus, *bacillus cyanogenus* of blue milk, *micrococcus magenta*, *spirillum rubrum*, and many others; to the latter class belong bacteria which only on a particular medium, e.g. on potato, form a pigmented growth, such as *bacillus* of glanders, *spirillum* of cholera; or on agar, *staphylococcus aureus*. With few exceptions (*spirillum rubrum*) the production of the colouring matter is dependent on free access of air; in the absence of this the colour is not produced, though the microbe shows active growth.

REACTION OF MEDIUM

Most species of bacteria grow best in faintly alkaline media, but there are some species which show good growth in even slightly acid media, and a few show very good growth in acid media of a certain definite chemical character—e.g. certain bacilli found in normal acid urine, and growing in this urine as long as it remains acid. In fact, the increase of acidity in the normal urine during the first twenty-four hours is referred to the fermentative action of these bacilli. Then the various species of bacilli which are concerned in converting

sugar into lactic acid (souring of milk) grow well when the medium has become distinctly acid by this reagent ; likewise the bacillus aceti, which is concerned in the oxidation of alcohol into acetic acid (e.g. souring of beer, wine) grows well in acid media. On the other hand, many bacteria refuse to grow in even faintly acid media—e.g. the anthrax bacillus, the streptococci, many saprophytic bacilli and micrococci, most of the pathogenic bacteria, micrococci, bacilli, and spirilla—although, as has been mentioned, many of them grow well on potato, which has a slightly acid reaction (malic acid).

The addition of acid to an otherwise favourable medium—e.g. acetic acid, citric acid, various mineral acids, as free acid or as acid salts—converts this medium into an unfavourable soil, and if, before the addition of the acid, growth of one or another species of bacteria had been taking place in that medium, the addition of the acid stops the further growth, and, if added in sufficient amount, kills the bacilli and micrococci—but not the spores—if acting on them for a sufficiently long time. And in this respect it must be mentioned that, though many bacteria themselves as they multiply produce no change in the reaction of the medium, others produce distinct acidity, while others produce only slight acidity and some even alkalinity. A useful method for studying this change going on in a given medium by the growth of bacteria is to add, after Buchner, a small amount of litmus solution ; this in itself has no bad effect on the growth, and by the change of its colour, the change of the reaction—if any—brought about by the growth of the bacteria can be easily followed.

Petruschky describes minutely the results of a very large number of observations made on several dozens of different species of bacteria in this respect, and he finds ('*Centralblatt f. Bact. u. Parasit.*' VII. Bd. Nos. 1 and 2) that some species do not alter the original alkalinity of the medium (whey) ; of this kind is the bacillus of fowl cholera, bacillus of Koch's septicæmia of mice, and bacillus of rabbits' septicæmia. Others produce a greater or lesser amount of acidity ; others produce alkali. In No. 2 of vol. vii. (p. 53) of that journal Petruschky gives a very interesting tabular statement of those species, and of the amount of acidity or alkalinity they are capable of producing when growing in that particular medium.

It is probably to the production of acidity as also of other substances—e.g. phenol—that the growth in certain cultivations ceases before the nutritive value of the medium has become exhausted, and, further, that the microbes (except the spores) remaining in that medium gradually die. Take, for instance, the two species of spirilla known as Koch's cholera spirilla and Finkler's comma bacilli ; both species when growing in broth or in nutrient gelatine, which latter becomes liquefied by either of them, produce acidity, slight but distinct, after some days' growth more pronounced in the former, i.e. Koch's spirilla, than in the latter, i.e. Finkler's comma bacilli ; and in accordance with this it is seen that the cultures of Koch's spirilla undergo degenerative changes and altogether die much sooner than those of Finkler's comma bacilli. This same phenomenon can be demonstrated also on some species of staphylococci and streptococci and on non-spore-forming bacilli. But not in all species of bacteria in which the gradual dying off, or at any rate the stoppage of further growth, in the culture takes place is this fact due to acidity, for in some no acid is formed and still the microbes gradually die off, or their growth comes to an end even where no exhaustion of the nutritive material had taken place. Thus, for instance, the typhoid bacillus, many species of streptococci, the diphtheria bacillus when growing in streak cultures on nutritive gelatine or other solid media, after some weeks' growth, come to an end, and yet there is no appreciable acidity or exhaustion of the medium produced. The writer has had many plate cultures (on slanting gelatine in

papillæ of the tongue; streptococcus, a capsulated bacillus (Sternberg) similar to the bacillus of Friedländer; a leptothrix forming motile bacillus; a leptothrix forming non-motile bacillus, this sometimes in enormous masses on the surface; occasionally a bacillus similar to the Klebs-Löffler diphtheria bacillus (Hoffman): the pseudo-diphtheria bacillus of Löffler; a small bacillus motile and not forming leptothrix; a diplococcus similar to the diplococcus pneumoniæ of Fränkel and Weichselbaum; a fine spirillum, spirillum denticola, particularly in caries of teeth; a comma-bacillus similar to Finkler's comma-bacillus (Miller); a comma-bacillus somewhat smaller than this (Miller, 'Die Microorganismen der Mundhöhle'; Podbielsky, 'The Microorganisms of the Oral Cavity'; Centralbl. f. Bact. u. Parasit., IX. Nos. 18, 19; also Dr. Th. David, 'Les Microbes de la Bouche,' Paris, 1890). Also in the mucus of the nasal cavity various species of micrococci, bacilli, and spirilla are present; in the contents of the lower parts of the small intestine their number is considerable, and in those of the large intestine their number is legion. In the normal mucus of the respiratory system¹ they are also present, and in the mucus and inflammatory products in the pharynx, larynx, less in the bronchial system, their number is sometimes great. There is a very great difference noticeable in this respect between the mucus derived from the bronchial system in the normal and the inflammatory secretions, as in catarrhal and other inflammatory states—since while in the former the number and species of bacteria are comparatively limited, in the bronchial inflammatory states their number is sometimes found extremely great. So also in inflammatory conditions of the fauces—e.g. in tonsillitis and in ulcerations—the number and species are extremely great.

The superficial layers of the epidermis—i.e. the stratum corneum—contain under normal conditions bacteria of different species (Bizzozero, Bordoni Uffreduzzi), which were studied by the latter observer in their morphological and biological characters. In sections through the normal mucous membrane of the alimentary and respiratory organs it is difficult to find any bacteria even in the most superficial layers of the epithelium. In sections through the normal mucous membrane of the mouth, the tongue, fauces, pharynx, larynx, and trachea, particularly the tongue and tonsils, the bacteria attached to or lodged between the most superficial scales of the epithelium are not difficult to demonstrate, and occur chiefly in the form of micrococci and bacilli. They cannot be traced into the middle and deep layers of the epithelium unless there is degeneration or breaking down of the surface, due to inflammation, ulceration, or the like, going on. In these conditions the bacteria can be seen in large numbers attached to these points of broken-down tissue, and from here they extend also into the depth; and it will be noticed that as we pass from the most superficial points of broken-down tissue into the depth towards the inflamed, but not broken down, parts, the number of bacteria gradually diminishes, and as we come to more or less normal parts, they altogether disappear. This would show that these bacteria thrive only where dead or diseased tissue exists, and that the more healthy the tissue the less they are capable of existing. This refers to bacteria of which the representatives are normally present on these surfaces—i.e. bacteria which are true saprophytes—and amongst these micrococci are better able to penetrate than bacilli. As regards the epidermis under normal conditions, no bacteria are found deeper than the surface of the stratum lucidum; in inflammations, ulcerations, and breaking down they can be traced also into

¹ Weibel, Babes, and particularly Besser, on the bacteria present in mucus of the nasal passages.

the depth according to the amount of breaking down of the tissue. In extensive ulcerative processes in the skin and mucous membranes the number of the bacteria on the surface, and their power of penetration from the surface into the depth, particularly that of micrococci and some small bacilli, goes on on a large scale, often forming continuous streaks and patches, sometimes filling a vessel containing blood in stasis or a lymph space or cleft, or other discontinuities in the tissue. In severe degenerative changes of the mucous membrane of the alimentary canal one sees bacteria covering in continuous masses the surface and extending thence into the depth, particularly into crevices and clefts like those formed by the detachment of the epithelium lining the glandular tubes in the mucous membranes; thus in dysentery, diphtheria, and Asiatic cholera, crowds of bacteria are seen extending in masses into the cavity of the Lieberkühn's follicles, and into clefts left by the detachment of the epithelium lining these glands. And the same may be said for other tissues where inflammatory and ulcerative processes occur in connection with a surface. It has been shown experimentally by Wyssokovitch ('Zeitschrift f. Hygiene,' Bd. I.) that when bacteria are introduced into the normal blood system they disappear without settling anywhere in particular; but if previously some ulcerative or degenerative process had been established, the introduction of saprophytic bacteria into the vascular system is followed by these bacteria becoming carried to and settling in these foci of degeneration, and here they are seen to multiply with great rapidity. Similarly it has been shown that various species of saprophytic bacteria introduced into the vascular system of a normal animal soon disappear altogether, the spleen being the organ to retain them longest; some disappear sooner than others; spores of *bacillus subtilis* remain for some time in, and are recoverable by cultivation from, the spleen.

From all these considerations it follows that the presence of various species of bacteria in various organs in which degenerative, ulcerative, suppurative, and necrotic changes are going on may be of the character of secondary invasion. It is a well-known fact that has been observed over and over again during many years past that such changes in the various organs in connection with, or subsequent to, some infectious and other diseases are in some cases associated with the presence of various bacteria, not having any causal relation to the primary disease, but due to their secondary access to these parts. But also for the normal alimentary canal (the lower ileum) it has been proved by Bizzozero, Ribbert, and Ruffer, that bacteria are capable of penetrating from the surface into the tissue of the mucous membrane; in the perfectly normal Peyer's glands of the cæcum of the rabbit it is not difficult to trace small bacilli from the intact free surface into the lymphatic tissue of the lymph follicles, either as isolated bacilli or enclosed as masses in large cells. The same applies to the tonsils, as shown by Ruffer. How much more will such an invasion be possible if the alimentary canal be in a diseased state, or after death? There is no difficulty in finding various species of bacteria in the portal system of the liver and in the spleen if the *post-mortem* be delayed for some time, say, forty-eight hours or more, notably in warm weather. On the peritoneal or serous surface of the alimentary canal bacteria, chiefly motile bacilli, are sometimes found on *post-mortem* even after a few hours in warm weather; these by their motility and rapid multiplication must have penetrated into the dead mucous membrane, and through this out into the peritoneum. Koch found in a strangulated person bacilli in the vertebral arteries six hours after death. In the large vessels of the inflamed lung and in the

tissue around, the writer has seen septic bacilli twelve hours after death. Similarly he has found motile septic bacilli on the capsule of the spleen of mice and guinea-pigs twelve hours after death. In the larger branches of the portal vessels he has met in the summer with micrococci and motile bacilli six hours after death. In necrotic patches of the liver, in the tissue of the tubercular lung, septic micrococci and septic motile bacilli are found in masses, where they must have been present during life.

From all this it follows that while saprophytic bacteria of various kinds are normally present on surfaces and in cavities connected with the free surfaces—i.e. the outer air—they have not the power, unless specific, to thrive in the normal living tissue; but the tissue becoming diseased, disorganised, or dead, can easily become the seat of growth for bacteria that have invaded the tissue or been carried there. Now the important question arises, What is the cause of this inimical power exerted by the normal living tissues on non-specific bacteria? A large number of observations carried on within the last few years by Fodor, Buchner, Ogata, Sirotnin, Nutall, and particularly Niessen, have shown that the plasmatic fluids of the body—lymph and blood—have in their fresh and living state the power to destroy and kill bacteria brought into contact with them. The experiments of Buchner, Nutall, and Niessen have shown that the fresh blood plasma used in the test-tube has a remarkable power of doing this, although this power differs considerably as regards different species. Thus, *micrococcus aquatilis*, *cholera spirillum*, *anthrax bacillus*, *typhoid bacillus*, and the *bacillus* of Friedländer are easily killed after a few minutes (five to twenty minutes), while others, e.g. *staphylococcus pyogenes aureus* and *albus*, *streptococcus erysipelatos*, *bacillus* of fowl cholera and swine fever, and *proteus hominis* are only very slightly affected by it; on *proteus vulgaris*, *bacillus fluorescens liquescens*, *bacillus aquatilis*, and *bacillus prodigiosus* it has no appreciable effect. But also in the cases where the fresh blood exerts its inimical action, this only takes place if the relative number of bacteria added is limited, for the killing power of a given quantity of fresh blood is limited, so that if the number of bacteria introduced be too large, the killing power of the blood does not extend to all bacteria; and having been consumed and exhausted in killing a certain number of them, others escape, and these, then, are capable of rapidly multiplying, as in any other medium. The power of the blood to kill certain bacteria rests with the plasma, and it is the same power that also kills the leucocytes. There is a remarkable parallelism between blood plasma and leucocytes on the one hand and blood plasma and bacteria on the other, for when the blood plasma kills the leucocytes it also kills bacteria, i.e. when the blood and blood plasma are fresh; fresh peptonised blood and peptonised plasma, on the other hand, kill neither the bacteria nor the leucocytes. When blood is heated to 52° or 58° C. for twenty to thirty minutes (Nutall) it loses the power of killing bacteria, which it otherwise killed; blood mixed with magnesium sulphate loses the killing power; when blood is kept for several hours it also loses this power. Blood to which bacteria had been added and thereby killed, coagulates quicker (Grohmann), just as blood which kills the leucocytes coagulates quicker.

Buchner ('*Centralbl. f. Bact. und Parasit.*' VI.) has made very extensive observations on the germicidal power of blood plasma and blood serum; he points out an important antagonistic action vested in these fluids, on the one hand, as to their power of being *nutritive*, and on the other, as to being *germicidal*; the first depends on materials no longer living, e.g. dissolved or broken-down blood corpuscles, the latter on the 'living' or 'active' condition of albumen. Buchner shows that the circulating blood possesses the germicidal property in a higher degree than blood after removal from the body: evidently

the former contains in a much smaller degree the particular nutritive elements than the latter, which of course contains the products of the dead or broken-down blood-corpuscles. Buchner further shows that the germicidal power of the blood is not dependent on the leucocytes at all—an important fact, since Metschnikoff and others attribute this function solely to the white corpuscles—and further that it depends entirely on the albumen present in the plasma or serum, as long as this is in combination with salt, or, as he terms it, is in an 'active' state. Plasma or serum free of cells acts germicidally; if from it, by dialysis, the salt is removed, it loses its germicidal power; the salts of the plasma or serum themselves possess, however, no germicidal power. Lubarsch ('Fortschritte d. Medizin,' Bd. VIII. No. 17) thinks it probable that the germicidal action and inhibitive power of the living tissues may in a large measure depend on the chemical activity of the tissue cells, that is, on chemical substances excreted or produced by the cells; hence the battle against bacteria is essentially of a bio-chemical nature, as has been ably demonstrated by Petruschki in a series of papers. It is, however, not decided of what exact nature this chemical substance is; it remains to be shown whether, as Hankin maintains ('Br. Med. Jour.,' 1890), it is a cell globulin, or whether it is a ferment, as is maintained by Ogata ('Centralbl. f. Bact. und Parasit.,' Bd. IX. Nos. 18 and 19).

Sanarelli ('Centralbl. f. Bact. und Parasit.,' Bd. IX. No. 14) gives an excellent *résumé* of the literature of this important subject; he at the same time describes carefully conducted experiments in which he proves the germicidal action of the living pure cell-free lymph of the frog on bacillus anthracis.

It follows, then, from these observations that the killing power of fresh blood on bacteria (strong for some bacteria, slight for others, and wanting altogether for still others) is a power which resides in the plasma, that it is a power which is exhaustible according to the number of bacteria introduced, and that also in some of the positive cases it is lost after the death of the blood, after heating, or after the addition of certain chemical substances.

PHAGOCYTES AND IMMUNITY

It has been maintained by Metschnikoff that the power of the living tissues of an insusceptible animal to kill certain bacteria, e.g. bacillus anthracis in the frog, rests with the white blood corpuscles, that these accumulate at the place into which the bacteria have been introduced, and here take up—'eat up'—the bacteria and destroy them, hence he called the leucocytes 'phagocytes.' [For the literature and history of phagocytosis, see Lubarsch, 'Centr. f. Bact. und Parasit.,' Bd. VI. No. 20.] Normal frogs are insusceptible to anthrax infection: Metschnikoff introduced bacilli anthracis into their dorsal lymph-sac, and herein noticed that after some time the bacilli are taken up by leucocytes and destroyed; hence in this case the immunity of frogs to anthrax was explained by saying that leucocytes—phagocytes—acting as scavengers, eat up and destroy the bacilli. This theory was then extended by Metschnikoff and others to all cases of immunity and insusceptibility, inasmuch as it was maintained that in all cases of insusceptibility the leucocytes or phagocytes, as soon as the bacteria are introduced, give battle to the bacteria, and by eating them up and destroying them, do away with their potential virulent activity. Susceptibility was explained to mean that the leucocytes do not act as phagocytes, they cannot overcome the bacteria, and therefore the latter, being victorious, multiply and produce infection. Immunity against a second attack was explained by the same theory; that by the first attack the leucocytes have acquired a certain germicidal power,

not possessed before, viz. to take up and kill the bacteria, while non-immunity against a second attack was due to the leucocytes not having acquired this power. This 'mechanical' theory of phagocytes was, however, soon subjected to severe criticism.

There is no doubt about the fundamental fact that leucocytes can act as phagocytes, that is to say, no one doubts that the amoeboid corpuscles of the blood, and of the lymphatic and connective tissues, do take up into their interior various kinds of formed matter: granules, organic and inorganic, blood-corpuscles or fragments of blood-corpuscles, and therefore there is no *a priori* reason why they should not take up bacteria or other microbes with which they are brought in contact. But this is not what the theory of phagocytosis teaches; for this theory maintains that the leucocytes are the aggressors; that they take up and destroy the living specific bacteria, thereby protecting the animal against infection.

Although Metschnikoff himself has recently somewhat altered the theory by admitting that not only the leucocytes but also the lymph and blood-plasma *per se* are capable of destroying the microbes, yet there is still left the fundamental assertion that the leucocytes of the tissues of an animal insusceptible to a particular disease do destroy the particular microbes, and are the principal instruments of procuring this immunity. Against this various objections have been raised.

In the first place it has been shown that after injection of anthrax bacilli into the lymph-sac of the normal frog, degeneration and death of many bacilli take place, which are free in the lymph and not included in the leucocytes (Fischel, 'Fortschritte der Med.,' IX. 2); that the cell-free, living lymph of the lymph-sac of the frog possesses germicidal action has been mentioned already above (Sanarelli). In the second place it has been shown by Petruschky, and also by Fischel, that long before any appreciable phagocytosis occurs in the frog's lymph-sac, there are already present in the lymph degenerating and dead bacilli, and that not before three hours after the injection of anthrax into the lymph-sac are there the first indications of commencing phagocytosis, viz. bacilli are seen adhering to the surface of leucocytes, but not yet enclosed in their substance.

Thirdly, it can be shown that long before any appreciable leucocytosis occurs in the lymph-sac, and before any bacilli are found enclosed within the leucocytes, numerous microbes are absorbed into the blood circulation, and can be demonstrated in a living condition in the blood and in the spleen. On injecting 5 minims of a salt mixture of anthrax bacilli or of bacillus prodigiosus into the dorsal lymph-sac of a normal frog, and testing the blood of the heart and the spleen by cultivation, ten minutes, half an hour, one hour, and two hours afterwards, innumerable colonies of these microbes are obtained, thus proving conclusively that they are absorbed into the general circulation, and the process of the destruction of the anthrax bacilli, and the immunity of the frog against anthrax, cannot depend on leucocytosis and phagocytosis going on in the dorsal lymph-sac, i.e. at the seat of inoculation.

A further set of objections was raised by Baumgarten, Flügge, and others to the effect that phagocytes, i.e. cells which include the bacteria, are found in many acute and chronic infectious diseases, where they—the phagocytes—are not required, where they can be of no use as regards destruction of the bacteria and the production of protection, for the simple reason that in these cases no protection is ever produced. Thus in mouse-septicæmia of Koch the white blood corpuscles become crowded with the specific bacilli and gradually become disintegrated, and hereby the bacilli, active and

virulent, become liberated. In Koch's Egyptian ophthalmia, and in gonorrhœa, the leucocytes are the special nidus for the specific microbes; in pigeons dead of swine erysipelas the leucocytes in the vessels of the liver are crowded with the specific bacilli, and are destroyed by them. In leprosy the bacilli select with pre-eminence leucocytes and round cells: in fact, a leprosy nodule is an aggregation of round cells, small and large, crowded with the living leprosy bacilli, and by disintegration of the cells the bacilli become free; in tuberculosis the tubercle bacilli are found in cells, small and large, and that is how the bacilli multiply and the cells become disintegrated. It is true that in the typical giant cells, e.g. in bovine tubercle, the bacilli present in them are most numerous at first, and gradually disappear from the giant cells (Koch), but this is probably due to the necrotic change of the cells themselves (Weigert, Koch), but in many instances we see the tubercle bacilli in leucocytes and round cells of all sizes destroying the cells themselves.

On the other hand, it has been urged that where one might expect that phagocytes should be present, as at the seat of inoculation in an animal possessed of immunity, the phagocytes are either absent altogether or are not present in commensurate numbers. Thus instances have been mentioned where after injecting into an insusceptible animal a dose of the microbes—such as would cause a general fatal infection in a susceptible animal—no phagocytosis, or none to speak of, could be demonstrated. Dogs, cats, and rats are insusceptible to anthrax, yet on injecting a good dose of anthrax material into the subcutaneous tissue of these animals no phagocytosis of an appreciable degree occurs. Fowl enteritis is not virulent for pigeons: on inoculating these animals with a large dose of the living bacilli of fowl enteritis, no phagocytosis occurs at the seat of inoculation. In some other cases, however, of inoculating insusceptible animals with the specific microbes, leucocytosis and phagocytosis do take place at the seat of inoculation.

Thus in symptomatic charbon, to which the rabbit possesses a certain amount of insusceptibility, on inoculating the virus subcutaneously, distinct leucocytosis and phagocytosis are observed (Ruffer); the same is the case with the bacillus pyocyaneus in the guinea-pig, whereas inoculation into the rabbit leads to acute general infection without leucocytosis at the seat of inoculation. The inoculation in the guinea-pig produces the opposite result (Charrin).

The virulent bacillus of diphtheria inoculated into the groin of the guinea-pig produces œdematous swelling, hæmorrhage, and necrosis at the seat of inoculation, and the animal dies; but if the culture is attenuated the injection produces local leucocytosis. The injection of the bacillus of grouse disease in sufficient doses produces in the guinea-pig acute and fatal infection, but it produces no local leucocytosis, whereas if a small dose be used, or if the microbe be attenuated, a distinct local leucocytosis, but no general acute infection, takes place.

On the other hand, in tuberculosis after subcutaneous inoculation with the tubercular virus into the guinea-pig the local leucocytosis is the first change in the course of the general infection: the local suppuration of the lymph-glands at the seat of inoculation being the first step in the whole chain of the inflammation (leucocytosis) and suppuration of the lymphatic glands, the lungs, and abdominal viscera.

We see from all this that while in some cases the introduction of certain specific bacteria into an insusceptible animal produces a local leucocytosis and phagocytosis, in other cases of insusceptible animals it does

not produce this, and further that in some cases of acute and chronic infections distinct phagocytosis is observed in animals susceptible to the particular microbe.

Petruschky showed ('Dissertation,' Königsberg, 1888) that even in the frog infection with anthrax can be produced after introducing the bacilli anthracis into the dorsal lymph-sac, and keeping the frog at a higher temperature (80° to 85° C.); under these conditions the bacilli are not killed by the lymph in the lymph-sac or by the leucocytes, and can multiply and produce infection, which they could not do at the ordinary temperature at which the lymph of the frog has the power to destroy the bacilli. Von Emmerich showed that if broth culture of the bacillus of swine erysipelas be injected in considerable quantities subcutaneously into a rabbit which had been some time previously rendered refractory against infection (by injection of culture direct into the blood-vessel), such subcutaneous injection produces no further infection; innumerable masses of bacilli had been thus injected under the skin, and after twenty minutes or so, though they are still to be found at the place of inoculation, they are all dead, no sub-cultures can be established with them; and there is a total absence here of all phagocytosis. The bacilli are here killed, not by the cells, but by something else present in the tissue. Interpreted in the light of the above observations we are quite justified in saying that it is the tissue juice (lymph or plasma) which had produced this effect, i.e. had killed the bacilli.

Dr. Coxwell and the writer have found that while the normal *Rana temporaria* possesses insusceptibility against anthrax, it succumbs to anthrax (bacilli are found in the blood and spleen) if the frog is, either at the time of the anthrax infection, or shortly before or shortly after, subjected to chloroform-ether narcosis. The lymph-cells after the animal recovers from the narcosis, i.e. after a few minutes, as also the lymph-cells taken from the animal while under narcosis, show normal amoeboid movement; the abolition of the insusceptibility of the normal frog against anthrax by chloroform-ether narcosis cannot therefore depend on the lymph-cells themselves, but must be due to some chemical change induced by the narcosis. The same results were obtained in rats. White rats are insusceptible to anthrax, but become susceptible when chloroformed; the narcosis with chloroform-ether lasting about one and a half to two minutes is sufficient to render them susceptible to fatal anthrax. The narcosis is made either previously to or immediately after infection with anthrax; the injection of the anthrax bacilli or spores a few hours after narcosis does not lead to fatal infection. In the positive cases death from anthrax takes place in three to five days, and the blood and spleen contain very numerous anthrax bacilli.

It is probable, then, that whenever bacteria, introduced into the blood or tissues, are unable to multiply there and to produce infection (as in the case of saprophytic bacteria, or in the case of insusceptibility of a particular animal against a particular specific microbe), this inhibition is brought about in the first place by the living and normal blood plasma or the lymph present in all tissues; that this inhibitory power is exhaustible according to the number of the bacteria; thus while in some particular instances (they are tolerably numerous, as will be shown later) infection cannot be produced by small doses, it can be produced by large doses; further, that this inhibitory power varies with the various species of bacteria and with the different animals.

The next question is as to the nature of these germicidal substances. As mentioned on a previous page, Buchner has given good reasons for regarding them as belonging to the albuminous bodies of the lymph and plasma, and he calls them 'alexines' (ἀλέξειν, protecting). It must, however, be clear that

whatever the nature of these alexines, they cannot be the same, either in all animals or for the different pathogenic bacteria. The alexines against anthrax in an insusceptible animal, e.g. rat, frog, cannot be the same as the alexines in glanders in an almost insusceptible animal, as the tame mouse. Nor can the alexines which are present in the tissues, and which act germicidally on saprophytic bacteria, be the same as the alexines protective in insusceptible animals against specific bacteria. Again, the alexines protecting a naturally insusceptible animal against a specific microbe cannot be the same as the substances protecting against a second infection a susceptible animal which has passed through one mild attack; that is to say, the natural immunity of an individual cannot be due to the same kind of protective substance as the acquired immunity.

In order to understand the argument it is necessary to mention certain important observations first made by Pfeffer on the remarkable power possessed by different chemical substances towards bacteria and other micro-organisms, substances which either attract or repel bacteria, these phenomena being spoken of as chemiotaxis; the former as positive, the latter as negative chemiotaxis. Pfeffer ('Unters. a. d. bot. Inst. Tübingen,' 1887, p. 582) found that motile organisms (bacteria, flagellata, and volvocineae) are stimulated by many organic and inorganic substances in solution—positive chemiotaxis. To mention only a few of the substances: the salts of potassium have a great 'stimulating' power, likewise peptone, glycerine, and morphine. Alcohol, free acids, and free alkalies have a negative chemiotactic action i.e. repel the microbes. Ali Cohen ('Centr. f. Bact. und Parasit.,' VIII. 6) made systematic observation on this same subject with various kinds of bacteria.

Gabritschewsky, Massart, and Bordet ('Annales de l'Institut Pasteur,' 1891, IV. 6), and others tested the action of bacteria on leucocytes, introducing chemical substances in capillary glass tubes into the living body of animals, and then examining these capillary tubes and seeing whether they attracted leucocytes or not; in this way they found that chemical substances either attract or do not attract leucocytes. Thus, for instance, Massart and Bordet found the lactic acid acting powerfully negative chemiotactic; Buchner found collagen, alkali albumen, gluten, and casein acting powerfully positive chemiotactic. Now, Buchner argues, and it seems with justice ('Centralbl. f. Bact. und Parasit.,' X. 22 and 23), that when in an insusceptible animal leucocytosis does occur at the seat of inoculation, this leucocytosis is not an expression of the commencing battle between the microbes and the leucocytes, as is maintained by Metschnikoff and his followers, but is due to a positive chemiotactic action on the part of the bacteria (dead or alive), by which the leucocytes are attracted. Extensive leucocytosis (suppuration) has been shown by Koch to occur after injection of tuberculin containing the products of the tubercle bacilli previously killed; suppuration (miliary abscesses) has been produced by Prudden and Hodenpil in the rabbit after injection into the vascular system of the substance of the tubercle bacilli, previously sterilised; also the insertion of sterilised tubercle culture by means of capillary glass tubes into the subcutaneous tissue of the rabbit proves positive chemiotactic action of the dead bacilli towards leucocytes. This chemiotaxis is brought about by the substance—protein—derived from the bacteria themselves, and is dependent on the inimical action of the tissue juices on the bacteria. Where the tissue juices possess this action the bacteria are thereby weakened or destroyed, and only under this condition does their substance—protein—become available to attract the leucocytes; in such cases

the weakened and also the killed bacteria are easily taken up by the leucocytes, and these then help to remove them. Under this theory the local phagocytosis observed in insusceptible animals is therefore dependent on the preceding inimical action of the tissue juices on the bacteria. But in the case of a susceptible animal, that is, when the introduction of the bacteria produces general infection and no local leucocytosis at the seat of inoculation, the bacteria, because they remain vigorous, and because they withstand the action of the tissue juices, do not yield the chemiotactic substance—protein—and therefore no leucocytes are attracted to the seat of the inoculation.

In connection with the phenomena of chemiotaxis it ought to be borne in mind that just as certain bacteria exert an attraction on the leucocytes, so also is it imaginable that the cells and tissues exert chemical attraction on certain bacteria, just as in the case of Pfeffer's experiments. This at any rate offers a ready explanation of the conspicuous attraction that one or the other tissue seems to exert towards certain specific microbes. It is well known that in the acute exanthemata, the skin is the tissue which pre-eminently exerts such a positive chemiotaxis on the specific microbes. In anthrax, in typhoid fever, in malaria, and in relapsing fever, the spleen has a conspicuous attractiveness for the microbes; in tuberculosis it is the lymphatic tissues and the spleen. In this disease the lymph-cells seem to be the particular nidus for the growth and multiplication of the bacilli. It is quite possible that the presence of saprophytes in the lymph-cells of the superficial parts of the tonsils, pharynx, and Peyer's glands (Bizzozero, Ribbert, Ruffer), mentioned on a former page, is to be explained in this way, viz. that these cells possess a chemiotactic action, being a more favourable nidus than the tissue juice, which has a germicidal action.

We have from the foregoing arrived at the conclusion, which seems on the whole the most feasible one, that the principal and essential agent in preventing the growth and multiplication of particular specific microbes within the insusceptible animal is the inimical action of the lymph and blood plasma, the alexines; this action may or may not produce phagocytosis. In the former case the phagocytosis may help to remove the weakened or dead bacteria, but this is not an essential or necessary factor in the process of the weakening and destruction of the microbes. Where the blood or the tissue juices do not possess this power, the animal is susceptible.

The question now arising is this, Is the acquired immunity due to the same physiological conditions as the natural immunity? Take, for instance, the case above quoted of Von Emmerich's experiments: a rabbit inoculated subcutaneously with a small quantity of culture of the bacillus of swine erysipelas becomes affected with the disease and generally succumbs to it; here, then, the juices of the connective tissue have no power to kill or to inhibit the growth of the bacilli. The disease—swine erysipelas—with which such a rabbit becomes affected after subcutaneous inoculation is a blood disease, the vessels in all the organs contain the bacilli, and every drop of blood is capable of producing infection. But if a normal rabbit be inoculated with the bacilli directly into the circulation, although disease follows, it is only of a mild type and the animal recovers. Such an animal is then found to possess immunity against a second infection with these bacilli; for, as Emmerich shows, injection of large quantities of the bacilli into the subcutaneous tissue produces no result, and the bacilli are found killed at the place of inoculation after twenty minutes. Therefore, by the first attack, something must have happened to the blood plasma and of necessity also to the tissue juices, since these are derived from the blood plasma; the most feasible view seems to be that some-

thing not previously present in the plasma has been added to it by the activity of the bacilli during the first attack. This theory would then be in harmony with the 'antidote theory' of Klebs, by which is meant that the bacilli during a first attack add a product of their own to, or produce a specific chemical change in, the tissue juices and blood which is antagonistic to themselves. And as long as this obtains in the blood and tissue juices the animal retains immunity against the same kind of bacilli. This view would be also in harmony with the fact that, although an animal has acquired by a first attack of one disease immunity against a second attack of this disease, it has no immunity against any other infectious disease. It would be in harmony with this, because since one species of bacilli produces a totally different disease from another, so also the products of one species, or the chemical changes induced, must be of a different character from those of another species; and if therefore a certain substance is added to the blood and tissues by one species, which substance is an antidote to the development of a second crop of the same bacilli—i.e. a second infection—this substance need not be an antidote to another species.

The fact that in a particular infectious disease not leading to death the multiplication of the specific microbes ceases, that is to say, the disease has reached its acme, and the microbes not eliminated from the body are destroyed, is in harmony with the above theory. In the first place, when the multiplication of the microbes has reached its acme, this is not due to an exhaustion (as was once thought in analogy to what takes place in alcoholic fermentation by yeast, i.e. the yeast multiplies in a particular sugar-solution, till this sugar is exhausted and is all converted into alcohol), for the simple reason that the tissues of an animal in the above case are far from exhausted as regards the particular microbe. There is nothing easier than to show that the blood and tissues of such an animal which, while alive, have served for the multiplication of the microbe, when used as artificial media—i.e. after death—are excellent soils for the multiplication of the particular microbe. On the other hand, we have given on a former page reasons which lead us to assume that the arrest in the process of multiplication and ultimate death of the microbe in artificial media is brought about by chemical substances produced by the microbes themselves, that is to say, formed by the microbes in the culture medium, and when this substance (or substances) has reached a certain amount the further progress of the microbe is inhibited and in many cases the microbes themselves are gradually killed off. And it is this same cause which we assume to obtain in the living animal body, in which the multiplication of the specific microbes ultimately reaches its end, i.e. the disease reaches its crisis, and the microbes are gradually killed off and the animal completely recovers. In accordance with this, as long as this antidote remains in the body, the animal is insusceptible to a new infection; but when this antidote in the course of time is eliminated, used up, and altered, the immunity wears off, and the animal is again susceptible to a new attack.

A further support to this theory is given by the recent researches on the behaviour of the animal body to the chemical products of specific microbes. Various observations have been recorded, by which in several infectious diseases the injection into an animal of certain chemical substances, the *toxins*, produced by definite specific microbes in artificial cultures, gives to such an animal immunity against a virulent attack. Wooldridge, Sirotinin, Beumer and Peiper, Salmon, Roux and Chamberland, Löwenthal, Roux and Yersin, and others have proved this. Wooldridge has shown that in a particular form of guinea-pig's septicæmia the injection of the culture fluid, from which the bacilli have first been killed off by heat, gives complete protection against

an infection with the virulent bacilli. Wooldridge has further shown that if a particular albuminous fluid in which the bacillus anthracis has been growing, and after the removal of the bacilli, be injected into the rabbit in sufficient quantity, a complete protection is produced against virulent anthrax. Sirotnin, Beumer and Peiper have shown that when Gaffky's (or the typhoid) bacillus is grown for some days in broth, and this broth, after the removal of the bacilli, is injected into mice, no further infection of such animals with the living bacilli, even in large doses, can be produced. (It ought to be mentioned that these bacilli injected in a sufficient dose into mice produce a fatal septicæmia.) Salmon has shown the same to hold good for swine fever; Roux and Chamberland, Löwenthal, and others have shown it for some forms of septicæmia; Roux and Yersin for the disease produced in guinea-pigs by the bacillus of diphtheria. In all these cases it is necessary to inject a sufficient quantity of the culture fluid free of the bacteria, or smaller quantities repeatedly, in order to produce protection against a second infection. The protective inoculation of Pasteur against rabies is based on the same principle. Again, if the quantity of the chemical products, the toxins, be too large, death may be produced in the same way as the bacilli growing in the body produce death by too much of their products; if the quantity be too small, not sufficient of the toxins has been added to the tissues to be protective, but if the quantity is of sufficient amount protection is produced. It seems, then, that the theory of acquired immunity as above stated is in complete harmony with these various facts, viz. that the microbes produce by their growth and multiplication in the tissues certain toxins, which, while present in the blood and tissues, furnish these with the power to inhibit and kill the same species of microbes on being reintroduced a second time—a power not previously possessed by the blood and tissues; further, that as long as these antitoxic substances remain in the tissues, the body remains insusceptible.

The observations of Ogata and Jasuhara ('Mitth. aus d. hyg. Inst. Tokio') and of Hankin ('Centr. f. Bact. und Parasit.' IX.) show that on injecting into an animal (mouse) susceptible to anthrax a sufficient quantity of the blood (Ogata) or of spleen substance (Hankin) of an insusceptible animal, the previously susceptible animal becomes protected, or if already infected the disease becomes arrested. In these experiments the protective alexines present in the naturally insusceptible animal are added to the blood and tissues of the otherwise susceptible animal, and so the blood of this latter is rendered unsuitable for the growth of the anthrax bacilli. This kind of acquired immunity can hardly be the same as the immunity produced by a first attack, or by the injection of the products of the specific bacteria formed in the blood or in the artificial cultures. Besides the experiments above quoted of Wooldridge, Roux and Yersin, and others, the experiments also of Behring and Kitisato on tetanus ('Deutsche med. Woch.' 1890, No. 49), prove this. In these experiments the blood of a rabbit previously made refractory against fatal tetanus is injected into a mouse, which is notoriously susceptible to tetanus; by this injection the latter animal becomes refractory to tetanus. Tizzoni and Cattani prepare certain albumens from the blood of a dog, previously made refractory against fatal tetanus; these substances are injected into an animal susceptible to or already infected with tetanus; by this injection the latter animal is rendered refractory to tetanus, or the development of fatal tetanus is prevented. Moreover, by such injection they have actually cured tetanus in the human subject ('Centralbl. f. Bact. und Parasit.' X. 24). In these experiments, then, the antagonistic and inhibitory action against the specific bacteria is produced by adding to the blood and tissues of the susceptible animal certain substances which are not

the same as the alexines present in the naturally insusceptible tissues, but include the products of the specific bacteria themselves.

In acquired immunity a distinction must therefore be drawn between a refractory state produced by adding to the blood and tissues of a susceptible animal the natural alexines of an insusceptible animal, or certain germicidal or germ-inhibiting drugs (e.g. trichloride of iodine in diphtheria used by Behring), on the one hand, and a refractory state caused by the addition of the chemical products of the bacteria themselves to the blood of the susceptible animal on the other hand.

CHAPTER VII

ANTAGONISM AMONGST BACTERIA

THAT the chemical products of some species of microbes, while acting inimically on the further multiplication of this species, are not inimical to that of another species has been proved by various observations, but it has also been proved that an inimical action is undoubtedly exerted by the growth of particular species on that of others. It is well known that a number of species of bacteria can exist and thrive under conditions under which other bacteria cannot so exist; take, for instance, the water bacteria, i.e. the bacteria inhabiting common drinking water; these are capable of living and of multiplying on the very small amount of nutritive material present in ordinary drinking water, nay, *micrococcus aquatilis* and *bacillus erythrosporus* (Flügge) and others, as mentioned above, multiply even in distilled water (Meade Bolton, Niessen, Percy Frankland); whereas numerous species of bacteria not habitually in water cannot do so under the same conditions; therefore the water bacteria will persist and even multiply, whereas others added to the water, or accidentally finding entrance into the water, will perish, some sooner, some later. Numerous observations have been put on record by Meade Bolton, Wolffhügel and Riedel to show in what way and to what extent various bacteria—the *bacillus anthracis*, cholera spirilla, the typhoid bacillus, *micrococcus tetragonus*, and *staphylococcus aureus* gradually die off when kept in ordinary drinking water, i.e. water very poor in nutritive materials. (The results of Meade Bolton are published in the 'Zeitschrift für Hygiene,' I. 1, p. 76; those of Wolffhügel and Riedel in the 'Mittheil. aus dem k. Gesundheitsamte,' Berlin, I. p. 455.)

It need hardly be said that if nutritive material be added to water, these bacteria will have a better chance of survival and of multiplication, and this chance will be proportionate to the amount of nutritive material added. Similarly De Giæza ('Zeitschrift f. Hygiene,' VI. 2, p. 162) made observations with reference to the conditions of existence of various bacteria in sea water, and his results are parallel to those made on ordinary drinking water. It need not be specially insisted on that neither ordinary nor sea water in themselves have any killing power on bacteria, but that where such an inhibitory power is observed it is due to the want of sufficient nutritive material, and that the greater the dependence of bacteria on organic material, and the poorer the water in such material, the more unfavourable is such water for the existence and multiplication of those bacterial species.

Next we have to consider the relations between two or more species simultaneously present in the same medium with sufficient nutritive material. Here more rapid multiplication will naturally depend, *ceteris paribus*, on the greater assimilative power; the greater this is, the more predominating will the species become. Thus, for instance, if in any organic material, say

dead animal tissues, saprophytic bacteria are present together with bacillus anthracis, this latter has not much chance of growing and multiplying; and hence in any part of an animal dead of anthrax, at first full of the bacillus anthracis, as soon as putrefaction has actively set in, the anthrax bacilli will be gradually killed off by the saprophytes, so that such material becomes deprived of the power of producing anthrax infection. The same obtains with other highly specialised, bacteria, e.g. the streptococci, the typhoid fever bacillus, and others. While this process of killing off of the more specialised and less assimilative bacteria by the more rapidly growing and more assimilative bacteria is essentially a survival of the fittest in the struggle for existence, there is another factor to be considered that not immaterially helps to bring about that result: it is the inimical influence the chemical products of the saprophytic bacteria have on the more sensitive and more highly specialised pathogenic bacteria. If, for instance, a filtered solution is made of a putrid albuminous substance, the putrefactive bacteria being all removed, and of this solution a considerable amount is added to an otherwise favourable nutritive material, e.g. alkaline broth, it will be found that this mixture is unfavourable for the growth of some species, in some cases more than in others. To the same class of inimical influences belongs the influence of faecal matter on various species of bacteria, e.g. anthrax bacilli, cholera spirilla, and the typhoid fever bacillus investigated by Kitisato. His results on the death of cholera spirilla in faecal matter are instructive. They are published in the 'Zeitschrift f. Hygiene,' V. p. 487.

Of a similar character are the observations recorded by Garré ('Correspond. f. schweizer. Aerzte,' XVII. 1887), who showed that nutritive gelatine which has already served for the growth of bacillus fluorescens putidus—a common saprophyte in water and putrid fluids—is no longer capable of serving the growth of some bacteria: e.g. the bacillus of Friedländer, typhoid bacillus, pink torula, staphylococcus pyogenes aureus; while others are capable of growing in such gelatine, though slightly retarded: e.g. cholera spirillum; and still others grew normally: e.g. Finkler's spirilla, bacillus anthracis. Towards the former, therefore, the bacillus putidus has a decided *antagonistic* action, while with the latter it is symbiotic. But this antagonism, when existing, is not necessarily mutual, for while the typhoid bacillus renders the gelatine also unfit for the growth of the bacillus fluorescens putidus—these two species being mutually antagonistic—it is not so with the bacillus of Friedländer or the staphylococcus aureus. Diphtheria bacilli can grow well in broth previously exhausted by proteus vulgaris.

To the same category belong the observations of Soyka and Bandler, who studied the manner in which certain bacteria are capable of growing in media previously exhausted by other bacteria ('Fortschritte der Med.' 1888, p. 76).

Cash has made similar observations with bacillus anthracis and certain micrococci when growing simultaneously. He found that the growth of the bacillus anthracis does go on to a certain extent, but that the virulence of it is impaired by the growth of the micrococci. This subject deserves a more exhaustive study than it has hitherto received. It is mainly of importance to ascertain whether and to what extent an inimical influence is exerted by one species on the other capable of growing simultaneously in the same medium. There is good reason for supposing that hereby, in some cases at any rate, one species is capable of attenuating the virulence of another. Thus in the cultures which Pasteur used as attenuated cultures for producing protective inoculation in fowl cholera, it was not, as Pasteur believed, the prolonged exposure to air that produced the attenuation of his cultures, but the impurity of his cultures (Kitt), and likewise the attenuated condition of the culture fluids that Pasteur used for protective inoculations against swine erysipelas,

were probably caused by the impurity of the culture fluid (Schütz); there being present in Pasteur's fluid, besides the true bacillus of swine erysipelas, a contaminating micrococcus.

Watson Cheyne, Von Emmerich ('Archiv f. Hygiene,' VI. 1887), and others showed that the streptococcus erysipelatos possesses a similar attenuating influence on the bacillus anthracis, for by inoculating simultaneously pure cultures of the two microbes into rabbits they were able to show that the bacillus anthracis was unable to produce fatal anthrax, though when separately inoculated it exerted its full virulence. It depends, however, to a considerable degree how much of the one and how much of the other microbe is injected in order to produce this inhibitory effect, for if too little of the streptococcus be injected the bacillus anthracis will exert its full virulence, or *vice versa*. This whole subject is obviously a very important one from a practical point of view—from the point of view of finding antidotes against the action of pathogenic bacteria—and it deserves greater attention than it has hitherto received.

The writer has himself made some experiments with regard to injecting simultaneously two species. In one series the bacillus of fowl enteritis was grown in broth with the swine fever bacillus; in the other, the bacillus of swine erysipelas with that of swine fever, but neither in the amount of multiplication nor in the virulence of the swine fever bacillus could any change be noticed. He has, however, succeeded in neutralising the fatal effect on mice of the grouse bacillus, if at the same time the aerobic malignant oedema bacillus (*see later*) be injected.

Bouchard, Charrin, Woodhead and C. Wood have shown that there exists a strong antagonism between the bacillus pyocyaneus or its products and the bacillus anthracis; so much so that upon the injection of the former, simultaneously with or immediately after the latter, into an animal susceptible to anthrax, this latter disease does not take place at all, whereas a control animal not treated with the pyocyaneus succumbs to anthrax.

It is well known that certain infectious diseases, of which infection occurred simultaneously in the same body, do not take place simultaneously, but that the one probably has to wait, as it were, till the other has gone through its course. In other cases one disease has clearly an inimical influence on another. Take, for instance, the observations repeatedly made by surgeons that erysipelas has a curative influence on certain tumours; Fehleisen had by direct experiment with pure cultures of streptococcus erysipelatos proved that certain sarcomata can be made to disappear and a cure effected by producing erysipelas in the skin of the part.

But there is a converse side to this, namely the question whether, and if so, to what extent, one condition, one species of bacteria or its products, enhances the power of multiplication and the action of another. Monti ('Ac. d. Linc.,' October 6, 1889) pointed out that the culture of the diplococcus pneumoniae—which, as is well known, gradually (by age and by continued subcultures) loses its virulent action on animals—regains the virulence if injected simultaneously with broth culture of the common saprophyte proteus vulgaris, from which the bacilli themselves are previously removed or killed by heat. This increased virulence of the pneumococcus may be achieved either by injecting this and the proteus culture at the same place, or at distant places simultaneously or soon after one another. The writer has similarly found that cultures of streptococcus of erysipelas, which had lost their action on rabbits, regained virulence if injected mixed, i.e. simultaneously, with broth culture (four days old) of the proteus vulgaris; and it made no difference whether the latter culture was or was not previously sterilised.

Charrin and Roger ('*La Semaine Méd.*,' 1890, No. 4) show that while normal rats are, as is known, very little susceptible to anthrax, they become highly susceptible if by working at a treadmill they are made fatigued, and H. Leo ('*Zeitschrift f. Hygiene*,' VII. 8) finds that by the presence of much sugar in the blood and tissues the susceptibility to anthrax and tubercle is not increased, while for glanders it becomes greatly enhanced. Phloridzin is administered in small doses with the food, sugar thereby becoming present in the tissues. Rats thus prepared resist anthrax as much as unprepared rats, guinea-pigs first prepared with phloridzin and then inoculated with tubercle do not show more intensive or more rapid tuberculosis. While normal white mice are almost insusceptible to glanders, they become highly susceptible to such infection, if prepared with phloridzin. Mya and Sanarelli give an account ('*Fortschr. d. Med.*,' IX. No. 22) of a large number of experiments, in which by introducing acetylphenylhydrazin into an animal insusceptible to a particular disease this animal becomes thereby susceptible. This substance is known to produce destruction of the red blood corpuscles (Gottstein) and hæmoglobinæmia; pigeons and rats thus prepared proved susceptible to anthrax.

CHAPTER VIII

BACTERIOSCOPIO METHOD OF EXAMINING WATER, AIR, DUST, FOOD

AN important branch of bacteriological research of water, air, &c., has been opened by Koch's gelatine method of plate cultivation as illustrated by the researches of Koch, Angus Smith, Hesse, Wolffhügel, Heyroth, Hochstetter, P. Frankland, Carnelly, Fraenkel, Kraus, Karlinsky, and others in accurately determining the number and, what is of far greater importance, the nature of the microbes present in water, soil, air, sewage, and various other materials, such as milk and a variety of other foodstuffs.

In all these researches a definite quantity (measured or weighed as the case may require) is added under proper precautions to nutrient gelatine (about 5 or 6 c.c. in a test tube); this is then liquefied in warm water, well shaken up and poured on to a plate. The character of the colonies and their number is then studied and easily ascertained, subcultures of the isolated colonies are made, and these further investigated. If the number of microbes suspected to be present in the materials be great, of course only small quantities must be used for the plate cultivations, or better still a small quantity should be previously diluted with a large quantity of sterile water or salt solution, and then of this a measured small quantity is used for each gelatine plate. The counting of the colonies in the plate cultivation is carried out by means of ruled plates. The writer uses for plate cultivations circular shallow glass capsules of the same diameter; this diameter being known permits of ascertaining the area of the plate either in square inches or centimetres. For counting the colonies, if the number of them be too large, and in order not to altogether lose the experiment, the plate is placed on a slate ruled in square half-inches or centimetres, and then the colonies, ten or twelve, are counted in squares, the average is taken and multiplied by the area of the plate. An example will illustrate this: the number of colonies in the plate being too great to be directly counted, the plate dish is placed on a slate ruled in square half inches; the average number of colonies in one square half inch (as a result of counting them in ten squares) is twenty-two, the plate dishes have each an area of thirty-two square half inches, therefore the number of

colonies present in the above plate is approximately 704. Now, for one of those plates in the experiment in question 0.5 c.c. of tap-water was used, consequently this particular tap-water contains 1,408 microbes per c.c.

Another mode of determining with exactness the number of colonies present, if moderately great, in a plate dish, is to invert the plate dish and then with a pen to draw lines on the glass dish so as to subdivide the area into four, eight, sixteen, or thirty-two as the case may require, and then to count seriatim the colonies in these areas. If the number of colonies present in a plate be limited, say 20 to 50, of course there is no difficulty in directly counting them.

It is advisable to at once prepare more than one plate, in order to obtain a more accurate result as to the number of colonies.

1. *Water*.—By means of a sterilised graduated pipette a small quantity—0.1 to 0.5 c.c.—of the water is added to a gelatine or agar tube for each plate dish. If the water is notoriously very rich in microbes, the quantity added must be of course much smaller, only $\frac{1}{100}$ to $\frac{1}{50}$ c.c. The writer uses for this and other similar determinations a set of graduated capillary pipettes of exactly the same kind as are used in the Hæmocytometer,¹ 10 c.mm. 20, 50, 100, 250, 500 c.mm., and 1 c.c. A short india-rubber tube, with a mouthpiece into which a small plug of sterile cotton-wool is pushed, can be easily fixed at the upper end of the pipette and this is then ready for use. The pipette before being actually used for measuring is filled by drawing up into it solution of bichloride of mercury (1 in 1,000) once or twice, then washed out well in the same way with sterile (boiled) distilled water, and next with the fluid to be tested. A measured quantity of the fluid is then drawn up (10 c.mm., 20, 50, 100 c.mm. or 1 c.c. as the case may require) and blown out into a gelatine or agar tube; this is then used in the ordinary way for a plate dish.

In the case of the ordinary London drinking water—tap-water—0.5 or even 0.25 c.c. yields sometimes too many colonies to be easily counted. When making a first examination of water of unknown character it is advisable to make several plate cultivations, one with 20 c.mm., one with 100 c.mm., and one with 250 c.mm.; thereby the amount is determined necessary to use for a plate for convenient counting.

In the case of ordinary London sewage even 10 c.mm. yield far too many colonies to be counted easily; add therefore 0.5 c.c. of the sewage to 99 c.c. of sterile water, and of this take 5 c.mm. for one plate.

The number of the bacteria present in London drinking water of various sources and under various conditions has been investigated by Dr. Percy Frankland; especially their morphological, biological, and chemical characters have been the subject of special study by this observer, and we must refer the reader to his papers in the 'Proceedings of the Royal Society,' and in the 'Phil. Transactions,' where they are fully described. As to the difference of water when fresh and stored, see Bischoff, Percy Frankland, and others.

2. *Soil*.—The method which the writer uses to isolate, to count, and to determine the character of the bacteria present in soil, dust, &c., is this: One gramme of the material is added to 1,000 c.c. of (boiled) sterile distilled water, while still warm; it is then shaken up repeatedly for from fifteen to twenty minutes; the mineral matter is allowed to subside and plate cultivations are made of the fluid as above. Ten c.mm., 20 c.mm., 100 c.mm. are added each to one gelatine or agar tube, to be used for one plate each.

¹ Mr. T. Hawksley, of Oxford Street, made for us a very useful case containing these graduated capillary pipettes, with india-rubber tubes and mouthpiece.

The number and character of the organisms in soil and dust (taken from the plugging or deafening material of the floors of dwelling-rooms, &c.) has been investigated by Emmerich, Percy Frankland, Carnelly and Miss Johnston and others. The character of the microbes has been studied by Dr. and Mrs. P. Frankland (On Nitrifying Organisms, *see* a later page). Hæreus describes four different species in the soil, of which he maintains that they are nitrifying microbes.

8. *Air*.—Miquel, Hesse, P. Frankland, Carnelly, Robertson and others have investigated the number of microbes present in various samples of air, under various conditions (town air, country air, mountain air, air of schools, dwelling-rooms, hospitals, &c.). The method is always in principle this: by means of an ordinary gas clock or gasometer a measured quantity of air is drawn at moderate rate by means of an aspirator—fall of water or mercury—through a cylindrical tube (Hesse), or through a flask (Frankland, Carnelly) containing a thin layer of solidified nutrient gelatine. Hesse's tubes are cylindrical, in which nutrient gelatine while still liquid has, by slightly rolling the tubes, set at one side in a thin film; they are plugged at each end with a sterile india-rubber stopper containing a sterile glass tube; to each is fixed a sterile tube, one is connected during the experiment with the gas clock, the other with the aspirator; the time during, or the rapidity with which the air is drawn through and the amount of air so drawn through are accurately noticed; after the experiment the glass tubes are plugged with cotton wool so as to serve as a filter against the entrance of further microbes. In Dr. P. Frankland's experiments, the air is passed through a sterile plug (asbestos or glass wool) contained in a tube or flask, and this having retained all microbes is then thrown into the gelatine; this is liquefied and well shaken so as to wash out of the plug all microbes and to uniformly distribute them in the gelatine. The gelatine is then used for making plate cultivations in the ordinary plates, or is set as a thin film on the inside of the flask. The writer uses a glass tube four to six inches long, half an inch wide, containing in the middle a cotton wool or glass wool plug about one and a half to two inches long; at each end the tube is plugged with a small cotton-wool plug. One end is drawn out in the shape of a large canula; the whole is sterilised. When used the plugs of the ends are removed, the canula end is joined to an aspirator and air is drawn through; at the end of the experiment the ends are again plugged. In order to use it afterwards for plate cultivations, the plugs of the ends are removed, the central plug is then pushed out by means of a thin glass rod, placed in liquefied nutrient gelatine or agar, well shaken, and then plate cultivations are made.

In Carnelly's experiments the nutrient gelatine is allowed to set at the bottom of a large sterile flask, the mouth of which is closed by a sterile india-rubber stopper, through which two glass tubes are passed—one long one through which the air passes from the gas meter, the other a short one connected with the aspirator: as the air passes into and out of the flask the microbes are deposited on the surface of the set gelatine.

4. Milk is treated in the same way as water; so also various table waters, seltzer water, soda water, &c.

5. Fæces and various substances more or less solid are treated in the same way as was mentioned of dust and soil.

6. Ice of different sources has been examined; a lump of ice is washed on the surface with sterile water, then from the middle a piece is chipped out and placed in a sterile test tube; immediately upon being melted a definite quantity of the ice water is taken for plate cultivations in the same way as for water analysis.

As regards the character of the microbes present in ordinary cow's milk

in ice, in fæces, see the papers by Escherisch, Hueppe ('Mitth. aus d. k. Gesundh.' II. 1884), Heim ('Mitth. aus d. k. Gesundh.,' V.), Brieger, Bienstock, and Escherisch, Heyroth,¹ Hochstetter,² C. Fraenkel, and others.

It ought to be mentioned, however, that to obtain an accurate knowledge of the exact number of microbes present in any material, it is not sufficient to examine the material by gelatine plate cultivations alone: errors of a considerable degree have been made in this respect, inasmuch as the number of microbes ascertained by the gelatine plate method has been shown to be lower—in some cases considerably so—than the actual number present in the materials, as ascertained by other methods. For this reason it is necessary to supplement the gelatine plate cultivations by agar plate cultivations. Fol has shown to what enormous errors one may sometimes be led by estimating the number of bacteria in water by the gelatine method alone; it has been repeatedly mentioned in previous chapters that there exist numerous species, some very important, which do not grow at 20° to 22° C., i.e. at temperatures at which gelatine plate cultivations are kept.

CHAPTER IX

ACTION OF BACTERIA

CHEMICAL FUNCTION

It would carry us beyond the scope of the present article were we to attempt to describe in detail the results of the numerous researches carried out by a host of investigators in connection with the specific chemical activities of bacteria and fungi. This subject has made important and remarkable progress within the last ten years, but it may be said to have in reality only recently seriously commenced. The researches of Pasteur, Cohn, Nencki, Fitz, Prazmowski, Brieger, Hueppe, Winogradski, and in this country P. Frankland, Warington and others, have brought to light a vast number of important facts; to the pathologist and sanitarian those of Selmi, Brieger, Bouchard, Hankin, Sidney Martin, Allan Macfadyen, Fraenkel, Behring and others, dealing with the chemical products of bacteria, so far as they are capable of affecting the animal system, must as a matter of course have precedence over those which, though interesting, are so more or less only from a general biological point of view.

It has now been shown that by bacterial life, outside or inside the body, alkaloidal substances are produced which belong to two kinds: some belong, judging from particular chemical reaction, to the group of alkaloids first demonstrated by Selmi, and called by and after him *ptomaines*; these substances are, if one might say so, the result of general activity of a large group of bacteria capable of producing putrefactive decomposition of proteids. Another kind are due to some more specialised microbes, and are of a specifically poisonous nature; they are said to be different in composition from the former and are called *toxines*; such are the poisonous substances demonstrated by Brieger, Wooldridge, Roux, Hankin, Martin and others from pathogenic microbes growing in artificial media; and it is believed that the same poisonous principles are also produced by them within the animal body (anthrax, diphtheria bacillus, and tetanus bacillus); and it is by such substances that within the affected body the particular infectious malady is produced. To this group

¹ *Arbeit. aus d. k. Gesundh.* IV. p. 1.

² *Arbeit aus d. k. Gesundh.* II. p. 1.

belong the toxins formed by the bacillus anthracis isolated by Hankin, Sidney Martin, Fraenkel, and others.

The exact chemical nature of these toxins is not determined, for some maintain that they are albumoses, others consider them as alkaloids, and others again compare them to ferments.

Reviewing the chemical function of bacteria, it will be found to be a manifold one:—

(1) In the first place some have a specific fermentative action. The proteolytic or peptonising action is a widespread one, both amongst putrefactive and non-putrefactive bacteria; albuminous substances so essential to the life of many species of bacteria by it are rendered soluble and available for these. Claudio Fermi published in 'Archiv f. Hygiene,' X. No. 1 an extensive research on enzymes produced by various septic and pathogenic bacteria, their nature and dependence on the nutritive media, and their non-identity with the various toxins. The production of acetic acid and lactic acid is also common to many organisms; then the action in converting urea into carbonate of ammonium, the mannite fermentation, the dextrose and indigo formation belong to this category, viz. specific fermentative action; so also the butylic alcohol fermentation of fatty and starchy matters, and the ginger-beer fermentation (Marshall Ward, 'Proc. of the Royal Society,' December 12, 1891). (On lactic acid fermentation see Pasteur, Lister, Hueppe, Grotenfelt, and Marpmann.)

(2) The nitrifying power is, according to Haereus ('Zeitschrift f. Hygiene,' 1886), common to many soil bacteria. The first of these actions is of necessity one that has a general, and one may say a very high importance, inasmuch as it is the great process by which certain nitrogenous bodies, like the salts of ammonia, are converted into nitrites and nitrates so essential to the vegetable kingdom; but neither the assertion of Haereus nor of Schloesing and Müntz, who claim to have isolated 'nitrifying microbes,' can be accepted, for Winogradski,¹ G. Frankland, and Warington² have shown that the microbe which converts ammonia salts into nitrites is different from that which converts nitrites into nitrates.

(3) The pigment action. The actual position and value of this is not yet well understood. The power to form pigment, generally dependent, *ceteris paribus*, on free oxygen (except in a few cases: spirillum rubrum), is very general, and, one would suppose, must be playing some great rôle in nature. Erdmann first pointed out the similarity existing between certain aniline dyes and the pigment produced by bacillus prodigiosus; Schrötter has minutely investigated this, and found that its pigment bears in every respect a striking resemblance to fuchsin.

(4) The production of substances acting as poisons on the animal body. This must be taken to be as much a specific action as that of pigment. As has been mentioned above, while some of these poisonous substances correspond to the group of alkaloids known as ptomaines, others are of the nature of albumoses (Hankin, Martin, Fraenkel, and Brieger). It is known through Selmi, Brieger and others that in some of the common putrefactive processes of albumen, poisonous alkaloids, the ptomaines, are produced; but it must not be supposed that only in putrefactive processes are these poisonous alkaloids generated, nor that all alkaloids produced in putrefaction are poisonous (Brieger).

(5) The pathogenic action, in other words, the power to produce in the animal body definite pathological changes, that is, a definite infectious disease.

¹ *Annales de l'Institut Pasteur*, iv. 4 and 5, v. 9.

² *Chemical News and Proceed. of the Royal Society*, 1891.

But on more carefully looking at such action in the light of the researches published during the last few years it appears that this pathogenic action depends entirely on the power of certain bacteria to produce toxins, and poisonous albumoses; and further it has been shown that such poisonous substances are actually isolable from the tissues of the diseased animal body. These poisonous substances are also elaborated by the bacteria outside the body (i.e. in artificial cultures), and when they are introduced separately, but in sufficient quantities, into an animal, the same pathological changes are thereby generated as when the bacteria work within the body; it has also been shown that by graduating this quantity of the poisonous substances, a mild and transitory illness may be induced, which in some instances is for some time protective against a second attack.

(6) As an action not generally considered a specific fermentative one is the putrid decomposition of albumen by certain bacteria, e.g. *proteus vulgaris*, *bacillus foetidus*, and others, which, growing and multiplying in proteid materials, break these down into lower nitrogenous principles (leucin, tyrosin, indol, skatol, and phenol), while at the same time foul gases like NH_3 , H_2S , H , CO_2 , and CH_4 are evolved; but it is a question whether this putrefaction is not as much a specific fermentative action, as for instance the peptonising action of bacteria or the acetic, lactic, and butyric fermentation; and further, it is not decided whether in all decomposition of albumen during putrefaction this is always brought about by the action of a single species, or whether in some instances of putrefaction several species are at work in the different phases of the process.

PATHOGENIC ACTION

In a former chapter we have shown that many bacteria cannot multiply within the normal tissues of the animal body; such bacteria, termed saprophytes or septic bacteria, do not produce pathogenic results when introduced into the normal tissues. But here again we have to differentiate those that, though saprophytes, can nevertheless assume the character of parasites when the normal character of the tissues is lost, as will be shown later on in various examples. From these septic bacteria must be distinguished those that can live and multiply within the normal living tissues. But in this last respect there exist the greatest differences: (a) while some species, as we have mentioned on a former page, are capable of doing so in one species of animals, they cannot do so in another; (b) some species, although their natural breeding ground is the normal body of an animal (secretions, mucus, &c.), in which, however, they produce no pathogenic effect, when introduced into the tissues of another animal show definite pathogenic action; (c) again some species producing in one set of animals (and man) a constant well-defined pathogenic action of one kind, when transferred to a different kind of animal produce a different though constant pathogenic action; further: (d) some species producing a definite pathogenic action in the animal body are capable of retaining this power unimpaired when growing outside the animal body, while other species manifest definite pathogenic action only when they have grown in the animal body and have been transferred from this directly to another animal body; when growing outside the animal body, in artificial media, they gradually lose this pathogenic power, some rapidly, others slowly; some of them, when growing under certain conditions (altered nutritive material, a different animal body), can again assume their pathogenic action, while others have apparently lost it permanently.

We proceed to illustrate these different groups:

(a) Bacteria capable of multiplication and of producing disease in one but

not in another animal. Most bacteria known to possess decided pathogenic power come under this category. The bacillus anthracis, while pathogenic for man, rodents, and herbivorous animals, has not this power, or only slightly so, on carnivorous animals, or on rats; bacillus mallei, bacillus tuberculosis, bacillus lepræ, bacillus cholerae gallinarum, spirillum of relapsing fever, (micrococcus) staphylococcus pyogenes, streptococcus erysipelas, streptococcus scarlatinæ, bacillus of fowl enteritis, bacillus of swine erysipelas and swine fever, bacillus of typhoid fever, diplococcus pneumoniae, and a host of others, belong to the group of bacteria which, though specific to one animal, are without pathogenic action on another. When they cannot produce a pathogenic effect, the animal is spoken of as insusceptible, and we have explained this condition by ascribing to the blood and lymph in this animal the power to kill the particular bacteria, or at any rate to inhibit their multiplication.

(b) There are several species of bacteria known, which, although derived from non-pathological materials, nevertheless possess the power of effecting definite pathogenic results in some animals. The capsulated bacillus known to occur in the normal fluid of the mouth (human), produces in the rabbit, when subcutaneously inoculated, a well-defined septicæmia (Pasteur, Sternberg). In human sputum (normal or pathological) there occur various species of bacteria, some of which (streptococcus, bacillus of Friedländer, diplococcus pneumoniae) have the power to produce septicæmia in rodents. In normal human faecal matter Brieger isolated a short bacillus which produces septicæmia in the guineapig; likewise Bienstock isolated a thin bacillus which is capable of causing septicæmia in mice. Von Emmerich's bacillus isolated from choleraic evacuations—the Naples bacillus—probably the same as Brieger's bacillus—causes likewise septicæmic infection in guineapigs. Koch's and Finkler's spirilla both act under certain conditions of experimentation like pathogenic microbes on guineapigs. Koch's septicæmia in rabbits, Davaine's septicæmia in rabbits, Koch's septicæmia in mice are caused severally by microbes occurring in putrid animal materials. The typhoid bacillus of Eberth, Koch, and Gaffky is capable of causing septicæmic infection in mice. The micrococcus tetragonus occurring in the purulent matter of tuberculous deposits (lung, and other localities) has a powerful septicæmic action on guineapigs (Gaffky).

(c) In this group may be mentioned the typhoid bacillus just referred to; which, however, it must be added, has not been as yet satisfactorily proved to be really the cause of typhoid fever: the evidence on this point is, though strong, not complete. This microbe, when introduced in more than minimal doses, produces septicæmic infection in mice; but this result is acute, the animals die in from one to three days with the symptoms of septicæmia, and the bacilli are present in the blood of the general circulation. The diplococcus pneumoniae of Fränkel and Weichselbaum, when from recent sub-cultures, causes general septicæmic infection in guineapigs and mice.

The spirillum of Koch and that of Finkler, when inoculated in considerable doses subcutaneously in mice and guineapigs, both sometimes cause septicæmic infection with the comma-bacilli in the blood of the general circulation. The staphylococcus aureus liquescens causes abscess, in some instances, when inoculated into rabbits, but in many other instances when injected into the vessels of rabbits and guineapigs produces a general septicæmic infection. So also the streptococcus pyogenes derived from human abscess occasionally causes general septicæmic infection in rabbits and guineapigs.

(d) Many bacteria of well-ascertained pathogenic power lose this gradually, owing to successive cultivation on artificial media, or to varied conditions

of cultivation. Examples illustrating these points are very numerous. The bacillus anthracis, so long as it is cultivated at ordinary temperature, or at a temperature up to 88° C. in neutral or very faintly alkaline broth, in neutral or faintly alkaline nutrient gelatine, on nutrient agar, or boiled potato, retains full virulence; Buchner is incorrect in asserting that by successive cultivations in broth the bacillus anthracis gradually loses all virulence. But there are numerous pathogenic bacteria which by gradual cultural removal from their original breeding-ground, i.e. the animal body, lose all pathogenic power; e.g. the bacillus of diphtheria, the diplococcus pneumoniae, the bacillus of Middlesbrough pneumonia, the streptococcus erysipelas, the streptococcus scarlatinae. And various species of bacilli causing septicæmia in rodents are of this category, e.g. the bacillus of Davaine's septicæmia, of Koch's septicæmia in rabbits, of malignant œdema, and others. While at first, i.e. directly derived from the human or animal body, they act virulently, and also possess this character in the first few sub-cultures, they lose this power—some sooner, some later—on continued cultivation. The spirillum of Koch is of the same nature; the sub-cultures of this microbe carried on for a large number of generations become certainly attenuated in so far as their action on the guineapig is concerned. Others, like the bacillus anthracis, bacillus cholerae gallinarum, bacillus of fowl enteritis, bacillus tuberculosis, bacillus mallei, bacillus of swine fever and of swine erysipelas, do not show any attenuation when the cultivations are carried on under unaltered conditions of temperature and soil. But if either the nature of the medium, or the temperature in which the sub-cultures are kept, becomes altered, these also undergo gradual attenuation, and may even lose the power to produce any pathogenic effect. The bacillus anthracis grown in broth at 42–48° C. undergoes distinct loss of virulence after eight to twelve days, and after fourteen to sixteen days altogether loses the power of producing fatal infection; when grown under unsuitable conditions of soil it also undergoes attenuation which may lead to the complete loss of pathogenic effect (Buchner). When the bacillus anthracis is grown in the mouse its virulence becomes attenuated, inasmuch as it does not produce fatal anthrax in sheep; when derived from the sheep or cattle its virulence is greater than when grown in the mouse or guineapig; when the bacillus anthracis is grown for a week or two on alkaline nutrient agar its virulence is less than when grown in slightly alkaline broth. The bacillus of swine erysipelas when passed through a succession of rabbits is attenuated as compared with the bacillus derived from the pig. The bacillus anthracis, the bacillus of fowl cholera, the bacillus of swine erysipelas when grown as impure cultures lose their virulence. The bacillus tuberculosis when grown at temperatures between 85–87° is less virulent than when grown at 88–89° C.; when grown on Koch's solid serum at 87–89° C. it retains its full virulence through numerous sub-cultures, while when grown on nutrient glycerine agar (Roux and Nocard) through a few sub-cultures it loses a great deal of its virulence.

The bacillus tuberculosis derived from bovine tubercles is more virulent for the guineapig and rabbit than that derived from the human tubercle; and the former produces general tuberculosis in guineapigs and rabbits in a considerably shorter time and more extensively than the latter. But it must be added to all these considerations that by growing these microbes again on the most favourable soil and under the most favourable condition of temperature the full pathogenic action is, as a rule, again attained, if not in the first at any rate in the subsequent sub-cultures. When the bacillus anthracis is grown for a fortnight in faintly alkaline chicken broth at 42.5° C., it will be found to have lost its virulence altogether, but on inoculating fresh

chicken broth from such a culture and keeping it at 35–37° C. for a few days, a culture is produced which possesses renewed virulence, and after another sub-culture its full virulence is recovered. On growing bacillus anthracis in nutrient gelatine to which perchloride of mercury is added in the proportion of 1 perchloride of mercury to 40,000 nutrient gelatine, the resulting growth fails in many instances to produce fatal anthrax; so does the sub-culture in a new set of medicated gelatine; but if from such an attenuated culture a fresh sub-culture is made in normal nutrient gelatine, virulent bacilli are again obtained. The same holds good for other microbes which have become attenuated by one cause or another. Take, for instance, the case of attenuation produced by growing in subsequent cultures the bacillus pneumoniæ (Middlesbrough); while at first the cultures of this bacillus are very virulent for mice, this effect is gradually lost as the sub-cultures are carried on through several generations, but when of such attenuated bacilli cultures are made in alkali-albumen the virulence of the bacilli is again recovered. Hueppe shows that by growing the spirillum cholerae and spirillum of Finkler in egg, closed after inoculation so as to ensure an anaërobic condition, a very poisonous culture is produced. Van Ermengen has shown that by growing the spirillum cholerae in aqueous humour a much more poisonous product is obtained than by growing it in broth. The streptococcus scarlatinae by successive cultivation in broth gradually loses its virulence on mice, but by again growing it in alkali-albumen a virulent growth is recovered.

In all these cases of attenuation, through one cause or another, the morphological and biological characters of the microbes remain practically unaltered; in all cases their microscopic character, the manner of their growth in plate, streak, and stab cultures remain very nearly the same, and from the study of these alone no alteration of their function could be recognised. It is important to bear this in mind, viz. that, so far as actual and careful observation hitherto shows, there is no definite morphological alteration going on hand in hand with the alteration in virulence; Buchner maintained that as bacillus anthracis in his sub-cultures gradually loses its virulence, it morphologically gradually becomes more like the motile hay bacillus, and he at first went so far as to say that not only does the anthrax bacillus in this course of attenuation and complete loss of its pathogenic power become the motile hay bacillus or something similar to it, but the typical motile hay bacillus can by cultivation become the non-motile virulent anthrax bacillus. All these and similar assertions must be held to be disproved by numerous observations carried on by exact methods of cultivation. The motile hay bacillus can no more assume the pathogenic character of the anthrax bacillus than a common non-poisonous snake can become an adder, though an adder by drawing its poison-fangs and poison-bags can become a non-poisonous snake; the offspring of such an artificially made non-poisonous adder are none the less poisonous adders.

It will be readily understood from the above considerations that cases may occur when one organism bears a striking morphological and biological resemblance to a pathogenic one, or to one of a similar though weakened pathogenic action; in such cases it is quite possible that they may have a genetic relation to one another, that is to say, the pathogenic species may have through one cause or another lost its pathogenic power, or this particular action may have become modified, but still the microbe may have retained all its morphological and biological characters. We will illustrate this by examples. The streptococci are a very numerous family, many species of streptococci resemble one another in their morphological and biological characters; the streptococcus erysipelatos, the streptococcus pyogenes, the

streptococcus scarlatinae are by some observers considered to be genetically related to one another, inasmuch as they maintain that the streptococcus erysipelatos may become modified into the streptococcus pyogenes and that this is only the same as the streptococcus scarlatinae. The action of the streptococcus pyogenes is uncertain, as in some cases it causes abscess, in others it does not; the streptococcus scarlatinae causes in some cases no other action than the streptococcus pyogenes, and finally the streptococcus erysipelatos in some cases does not produce any other action than that of the streptococcus pyogenes. Now, from an extensive and long-continued study the writer has come to the conclusion that these assertions are not in harmony with the facts, because the real streptococcus erysipelatos is in functional respects totally distinct from the others; what is described as streptococcus pyogenes comprises a number of different species, and the streptococcus scarlatinae can be morphologically, biologically, and functionally distinguished from the streptococcus pyogenes. We may add to these species also two other streptococci: the streptococcus of the Edinburgh cow disease and the streptococcus of human puerperal septicæmia. We cannot therefore agree with those observers who do not distinguish between these different species, although we agree with the general proposition that each one of them by successive cultivation on gelatine after some time does gradually lose its original pathogenic function.

Hueppe described a non-motile bacillus which he cultivated from soil, and which he finds in morphological and cultural characters very difficult to distinguish from bacillus anthracis, yet according to him it does bear a certain relation to this latter, inasmuch as it—the former—inoculated into mice, gives protection against the latter, although itself it does not cause anthrax. Hueppe is inclined to think that this species of bacillus may have once been bacillus anthracis, but have lost its pathogenic power. Now, assuming that Hueppe's observations as to the protective power of that soil-bacillus against the bacillus anthracis be a reality, we still find that this latter, on its change into the former, has retained its morphological and biological characters.

The important question that presents itself is—What causes in some bacteria this attenuation in function?

On a former page we have mentioned that in several instances it has been shown that the chemical products of certain pathogenic bacteria, when injected in certain quantities, have the power of causing a mild form of the particular disorder, and thus to protect against the virulent disease; and we have also mentioned that these protective chemical products are elaborated by the bacteria, not in all, but only in certain media. Thus it is known that the anthrax bacilli grown in broth do not form those toxic substances in any appreciable quantity: Wooldridge's success was only achieved by growing the bacilli anthracis in certain albuminous fluids. Then we mentioned that Van Ermengen has shown that the chemical products of the choleraic spirilla in aqueous humour, and in the intestinal fluid of the intestine of guineapigs, and Hueppe that those grown in eggs are infinitely more poisonous than when grown in broth. We have also seen that certain other bacilli and micrococci when grown in alkali-albumen regain poisonous action, when by previous successive cultivation in gelatine they had lost it. From these facts, it seems that the conclusion is justified that the virulent activity of pathogenic bacteria (that is the production of the toxins) depends, *ceteris paribus*, in a great measure on the medium or the soil in which they, the bacteria, grow. We might suppose that owing to certain substances being absent from, or other substances being added to, the original soil in which the bacteria multiply, or that owing to certain other conditions, such as tem-

perature, obtaining in their growth, the chemical activity of the bacteria becomes changed, and that the result of their own chemical constitution and of the molecular changes brought about by those so altered bacteria also becomes changed. And we would further conclude from these facts that a bacterium itself chemically changed, and altered in its chemical action, would manifest a different chemical action at least for one generation when again transferred to the suitable soil. There can be little doubt, and it is now generally agreed, that in the pathogenic action of bacteria it is not merely their presence in numbers nor their mechanical effects which cause the disease, but that it is by their chemical products, by their toxins, that they manifest their poisonous action, that it is the character and amount of these chemical substances which determine the peculiar pathological states characterising the different diseases caused by the bacteria. In anthrax, though the number of bacilli in the blood and spleen is, as a rule, great just before death, it nevertheless can only be owing to their particular specific chemical action that the disease is brought about; in many specific diseases the number of the bacteria is not large enough to cause mechanically those grave local and general effects that we meet with; besides, the character of the changes is so utterly different in the different diseases. This undoubtedly must be due to specific chemical action, i.e. to action of the toxins of those bacteria, differing in nature and in amount in the different cases. This is confirmed by the observations above stated, according to which it has been possible to obtain these poisonous substances in artificial cultures and to separate them from the bacteria themselves. Add to this also that a powerful necrotic influence is exerted by some bacteria over considerable areas of the tissue in which they grow, e.g. in swine fever, certain septicæmia bacilli, the tubercle bacilli, and the diphtheria bacilli.

We can readily understand that if the bacteria, owing to one cause or another, have undergone a modification in their chemical constitution and chemical activity, their poisonous products which cause the virulent disease may have become modified either in character or in amount or both, and that such modified bacteria will not be able to produce the same results, either as regards intensity or the character of the disease, as the unmodified bacteria. Under the same theory we can also understand how certain bacteria growing in the blood produce a different effect from that which results from the same bacteria growing in the subcutaneous tissue, e.g. the bacillus of swine erysipelas, or the bacillus of symptomatic anthrax. In the former case the bacilli after injection into the blood-stream produce a mild disorder, but growing in the subcutaneous tissue produce a severe fatal disease. In the first case the chemical conditions evidently cannot be the same as in the latter; while for other microbes just the reverse is the case, viz. when growing in the subcutaneous tissue the disease produced is milder than when they are growing in the blood, e.g. in small-pox; inoculated small-pox is milder than small-pox in which the contagium has passed (by the respiratory organs) into the circulation.

In connection with the chemical products of specific bacteria there is one question which it is important to clearly understand and to define, viz. the question of the exact nature and meaning of infection. In the first place it is necessary to remember that it has been proved for several pathogenic (in the wider sense) species of bacteria—*bacillus anthracis*, *spirillum cholerae* Koch, *spirillum Finkler*, several species of septicæmic bacilli, *bacillus diphtheriæ*—that also outside the animal body they can produce the same toxins which they produce when growing within the body, and by which they set up the particular diseased state. There is, however, this great difference in this

respect, that when multiplying within the body the amount of the specific chemical poison can reach, *cæteris paribus*, fatal proportions, whereas when multiplying in an artificial medium the nature and amount of this poison is subject to determinable conditions. But not only do pathogenic bacteria produce chemical substances acting like poisons, since other bacteria not capable of multiplying within the body can, as has been mentioned already, outside the animal system produce chemical substances, ptomaines, which act like poisons, and which when introduced into an animal body cause definite pathological states.

Since Gaspard, Virchow, Panum, and Bergmann have shown that various animal materials, blood and tissues, having undergone putrefactive changes, contain substances which by purely chemical means can be separated from the bacteria that produced them, and which, when injected into the body of animals, produce, according to the amount injected, more or less severe disturbances, leading even to a fatal result. When injected in small quantities, very soon after (from a quarter to one hour) a rapid rise of the body temperature with shivering takes place, followed by gastro-intestinal disturbance, retching and vomiting, tenesmus and purging; pain in the abdomen, muscular contractions and spasms; in large doses congestion and hæmorrhage occur in the stomach and intestines, particularly the duodenum and rectum, the peritoneum and the pleura; sanguineous and fluid evacuations pass from the bowels; a rapid fall of the body temperature, collapse, and death takes place. On *post-mortem* examination congestion is found in all the viscera and the serous membranes, hæmorrhages in the lungs and liver, in the serous and mucous coat of the stomach, the duodenum and rectum. The disturbance, or set of disturbances, called *sepsis*, *septic intoxication*, or *sapremia*, takes place a short time after the injection; they are therefore caused by chemical poisoning, as is also proved by the fact that the substances injected are separated from the putrid materials by means which destroy and kill all living micro-organisms (precipitation by alcohol and boiling). Selmi has shown that the substances produced by putrefaction and causing the above *septic intoxication*, or *sapremia*, possess certain well-defined chemical characters which show them to be alkaloids, and he called them *ptomaines*. Brieger isolated and more accurately defined these substances (see also Dr. Hunter's researches), and showed their production to be dependent on certain materials; he obtained them in crystallised form and showed that some, e.g. neurine, act very poisonously, while others (choline obtained from yolk of egg) do not do so. Hueppe obtained some ptomaine (cadaverine) of a very poisonous nature by cultivating Finkler's and Koch's spirilla in egg anaërobically. Various forms of toxic disturbances similar to the above have been observed to follow the ingestion of certain substances, as cream, cheese, pork and ham, sausages, mackerel, and other foodstuffs, and in all these the presence of ptomaines, as causing the severe gastro-enteritic disturbances followed sometimes by collapse and death, is also assumed. In most of these cases it has been shown that the intensity of the disturbance is proportional to the amount of the material consumed, and further that the symptoms set in very soon after the introduction of the material, so that there can be no doubt that we have to deal here with poisoning by chemical substances as opposed to infection with living micro-organisms, in which latter case there is always a considerable lapse of time or incubation period before the symptoms supervene, owing of course to the fact that in this case the micro-organisms must first multiply to a certain degree in order to produce the toxins, and by these the pathological results. But because the symptoms of the intoxication are very similar in all these cases—gastro-enteritic disturbances, retching and vomiting,

tenesmus and diarrhoea, congestion and hæmorrhage of the internal viscera, even leading to collapse and death—it would be unwarranted to assume that they are all caused by the same chemical poison. Septic intoxication or sapræmia is no doubt produced by ptomaines—Selmi's and Brieger's ptomaines (*see a former page*)—the result of putrefaction, if these ptomaines are introduced by injection into the subcutaneous tissue or directly into the vascular system, but we are not aware that anyone has succeeded in producing it by simple ingestion; whereas the poisoning produced by certain articles of food has always followed ingestion. It is probably correct to assume that, besides the putrefactive changes, other similar, but by no means identical, changes brought about by micro-organisms occur, by which certain chemical poisons—possibly alkaloids—are produced, which, though similar to the ptomaines of putrefaction, are not identical with them. By putrefaction is generally understood a set of changes due to the multiplication of certain micro-organisms in albuminous materials, in the course of which albumen is peptonised, then further split up, yielding leucin and tyrosin, and also indol, with the simultaneous evolution of ammonia and sulphuretted hydrogen, &c.; in these processes, conspicuous by the evolution of ammonia, sulphuretted hydrogen, and indol, the ptomaines of Selmi and Brieger are produced. But there are a number of micro-organisms known, which do not cause in albuminous substances the typical putrefactive changes in the above sense. There is never any ill smell evolved, and no ammonia or sulphuretted hydrogen, yet very poisonous substances are produced. Take, for instance, the bacillus enteritidis of Gärtner, and the bacillus isolated by Gaffky from sausage; in one instance horseflesh, in the other sausages, produced severe gastro-enteritic disturbance. The writer has isolated several species of bacilli, one from veal pie that had caused in some members of a family gastro-enteritic poisoning, another from pork pie of a similar poisonous nature, and still another from beef pie. These bacilli when grown in broth or in nutrient gelatine produced poisonous chemical substances, as was proved by ingestion of the cultures into mice, in which severe gastro-enteritis and death were caused; no bad smell of any kind was noticeable in the culture-fluids; on the contrary, there was present in them a rather pleasant aromatic smell. Neither of the above bacilli is capable of growing and multiplying in the animal body, but when grown in broth or in gelatine they produce those poisonous substances. In some cases of poisoning by cream and cheese, Dr. Vaughan has isolated from such cream and cheese, crystals of a certain substance (tyrotoxin) which acted very poisonously. It follows, then, that the term ptomaine poisoning or sapræmia should be reserved for that set of cases in which the ptomaines of Selmi and Brieger, produced by putrefaction of albumen, are at play, while those special changes caused by special, non-putrefactive microbes in certain albuminous substances—e.g. cheese, mackerel, veal pie, sausage, pork, tinned meat of one kind or another—should be considered as special fermentative changes in no way similar to putrefaction, and the products of such fermentative changes should be, as is becoming more and more the custom, placed under the same group of toxins as are produced by the true pathogenic microbes.

Different from these intoxications, produced by chemical substances elaborated by bacteria outside the animal body, are the *true infective processes*. In these latter the microbes enter the system, multiply therein, and when sufficiently numerous, i.e. when their special toxins have been produced in sufficient quantity, set up a particular diseased state. The time that elapses between their entrance and the setting in of the symptoms is, as is well known, spoken of as the *incubation period*. In the true infective processes, then, there

is an incubation period. The length of this is, as is also well known, different in the different diseases, and it marks one of the characteristics of the particular disease. But in the same disease it is subject to more or less marked variations, depending on the number of microbes introduced, on the susceptibility of the individual, and on other conditions not yet understood. It has been shown by the observations of Wyssokowitsch, W. Cheyne, and others that, within certain limits, the number of microbes introduced influences the length of the incubation period and even the severity of the disease, but beyond a certain number of microbes introduced no influence is noticeable. In some cases of very great susceptibility, e.g. rodents towards anthrax, pigeons, fowls, and rabbits towards fowl cholera, the influence exerted on the duration and severity of the disease by the number of bacilli originally introduced is very slight or almost nil, while in other diseases it is very marked, as in certain septicæmic disorders, e.g. malignant cedema and swine erysipelas. Here, when small numbers are introduced, the incubation period becomes very prolonged; in malignant cedema this prolongation is sometimes very considerable, and the disease is mild. The writer has seen in several cases of septicæmic infections (guineapig's septicæmia caused by a bacillus, the microbe of grouse disease, the bacillus of pneumonia, Middlesbrough) the incubation period vary, according to the amount injected, between twelve hours and two to four days, whereas in the case of the bacillus of fowl cholera even small quantities act like large doses, no change in the incubation period being noticed. It is feasible to assume that in those cases of slight susceptibility, where considerable quantities are required in order to produce the disease, this is due to the fact that the blood or the tissues have a certain killing power on a certain number of microbes (*see* the experiments of Niessen, Buchner, Lubarsch, and others previously mentioned), which killing power becomes, however, exhausted beyond that number of microbes. If, then, more than this number be introduced the surplus will survive, multiply, and cause the disease; if below this number, no living microbes remain and no disease is produced.

The coincidence of minor severity with prolonged incubation noticed in some, though by no means in all, cases of infections is a very curious phenomenon, and may possibly be due to the fact that in these cases a small number of microbes being introduced, they by gradual multiplication produce only sparingly the chemical poison, so that the animal body has time to accommodate itself, as it were, to the action of these toxins; and, further, that, owing to the small number of bacteria introduced, the incubation period becomes prolonged, the total amount of the toxins small, and the disease itself of a mild type. It is different if at the outset a large number of microbes be introduced; by the multiplication of these at once a large host and a large amount of toxins is produced, and the body not having time to accommodate itself, being as it were suddenly overtaken by these poisons, rapidly manifests the reaction and in a more striking and severe form.

CHAPTER X

MANNER OF INFECTION OR CONTAGION

THE way in which the microbes or contagia enter an individual is subject to considerable differences, differences depending on the conditions of the surroundings as well as on certain conditions of the subject. While in some diseases the contagium enters the body by inoculation, i.e. through an abrasion, wound, or the like in the skin or mucous membranes; such as surgi-

cal septicæmia, traumatic erysipelas, tetanus, hydrophobia, malignant pustule, glanders; in others the alimentary canal or respiratory organs or both are the portals, though artificially also by inoculation the disease can be readily produced. Take for instance anthrax in sheep and cattle; this under natural conditions is always contracted by these animals through the spores of anthrax gaining access to the alimentary canal or respiratory tract; in woolsorters' disease also by the alimentary or respiratory tract; in small-pox, typhus, scarlet fever, measles, foot-and-mouth disease, diphtheria, tuberculosis, actinomycosis by the respiratory or alimentary canal; ¹ in typhoid fever and in cholera by the alimentary canal; in relapsing fever and malarial fever by the respiratory organs. That in cases like scarlet fever, diphtheria, tuberculosis, typhoid fever, foot-and-mouth disease, cholera, dysentery, and others, infection takes place by ingestion of the contagium by articles of food—water, milk, fruit, and other materials—is well established. The division of bacteria into *ectogenic* and *endogenic*, first introduced by v. Naegeli, is accepted by many bacteriologists. Ectogenic are those pathogenic bacteria which are capable of living and thriving outside, and independent of the animal or human body, while endogenic are those pathogenic microbes which can only thrive in the living animal body: the former, though capable of carrying on life as saprophytes, can also exist in the living animal body as parasites producing a specific disease, while the latter exist only as parasites. Thus, to quote a few examples, the malarial microbe (plasmodium), the anthrax bacillus, various septicæmic microbes, e.g. of malignant oedema, the bacillus of typhoid fever, of cholera, of fowl cholera, fowl enteritis, swine fever, swine erysipelas, tetanus, human erysipelas, and a host of others, are all ectogenic; while the undiscovered microbe of small-pox and vaccinia, the undiscovered microbe of rabies, the microbe of leprosy, and that of syphilis, are endogenic. It is well known that rabies, syphilis, gonorrhœa, and vaccinia have never been known to occur other than by direct contact, the virus being transferred directly from one individual to another. While for general practical purposes this distinction into ectogenic and endogenic contagia may be admitted, nevertheless, when looked at from a theoretical and scientific point of view, its correctness becomes doubtful. Take, for instance, gonorrhœa: there is every indication that, as in syphilis, so also in gonorrhœa the microbe is ordinarily only of parasitic nature; but since it, i.e. the gonococcus, can be, and has been cultivated in artificial cultures, i.e. outside the human body, there is no reason whatever to assume that it cannot also carry on existence independent of the human body. Or take tuberculosis. Here also the microbe, i.e. the bacillus tuberculosis, is, as a rule, of the nature of a parasite, which, though it can grow well in certain artificial media independent of the animal body, does not, under natural conditions, carry on an independent existence: this is due to the fact that the bacillus tuberculosis being so highly specialised requires special media, and special temperatures, to carry on existence, conditions not met with ordinarily in nature. But Schottelius has shown ('*Centralbl. f. Bact. und Parasit.*, VII., No. 9) that tubercle bacilli taken from the lung of phthisical bodies buried for years still possess the characteristic reaction, and are capable of producing tuberculosis on inoculation. This would mean that the tubercle bacillus, even after years, was still living and capable of carrying on an existence outside the *living* human or animal body, and, therefore, is not strictly endogenic.

¹ Karkanoff made experiments on rodents with the bacilli of fowl cholera, and found that with these, infection is producible by ingestion, with anthrax very rarely, and with Emmerich's Naples bacillus not at all. He thinks that, as regards anthrax, infection probably takes place principally by way of the respiratory organs.

Of the same doubtful nature is the classification into fixed and miasmatic contagia. In former years this distinction was much used in practice; the first name 'fixed contagia' was meant to denote those contagia which in the narrower sense of the word act by contact, i.e. where the virus passes from the sick directly to the healthy, while the latter name 'miasmatic' indicated that the virus spreads not by direct contact but is volatile, i.e. communicates itself to the air, and thence finds entrance into a new individual. Those contagia were considered as a third group, which in some cases proved themselves to be either fixed or miasmatic, i.e. in some cases act by direct contact, in others by the air. But since the contagia have been proved to be of the nature of minute microscopic particulate matter, some of which can easily become distributed through the air in a living and active state, the above distinction has lost somewhat of its scientific basis. Take, for instance, anthrax: this would certainly be considered as a fixed contagium, i.e. the anthrax bacilli contained in any material derived from an animal dead of anthrax; and yet we now know that the spores of *Bacillus anthracis* when adhering to dry material can easily become distributed through the air and produce infection, e.g. woolsorters' disease. Or take diphtheria: this was always considered to be a disease in which the contagium is typically a 'fixed' one; yet why the diphtheria bacillus, or particles of matter enclosing the diphtheria bacillus, when protected from drying and dying, should not be able to become distributed through the air, there is no theoretical reason whatever. As a matter of fact, some health officers ascribe to sewer air, in localities in which diphtheria cases had been prevalent, the power to convey to the healthy the contagium of diphtheria. That diphtheria can be, and often is, communicated through infected milk, there can be no manner of doubt, and there can be no theoretical reason adduced why the diphtheria bacillus enclosed in and guarded by albuminous or other matter from drying up (the diphtheria bacillus does not form spores, and is killed by thorough drying) should not be able to produce infection through the air into which it had become distributed. From a practical point of view, it is perfectly true that vaccinia, rabies, tetanus, glanders, and syphilis are not communicated by the medium of air, but by direct contact, i.e. their contagia are fixed, and it is equally true that malaria and relapsing fever are naturally communicated as miasmatic contagia; but when we come to anthrax, variola, tuberculosis, erysipelas, pneumonia, fowl cholera, swine fever, and many others, the distinction breaks down, and we have then to speak of contagio-miasmatic diseases.

An important point in the method of infection is the question, whether microbes can pass the uninjured mucous membranes. The opinions of pathologists are divided: some maintain that such is the case, while others doubt it. Those who are for such a passage of the bacteria into the uninjured membrane rely on the fact ascertained in numerous cases, that certain diseases (small-pox, syphilis, tubercle) have been transmitted by the mother (in human beings and in animals) to the fetus in utero, while those who doubt the passage of bacteria through the uninjured membranes rely on the fact that in equally numerous cases, equally well established, such a transmission did not take place. Now it must not be omitted, in judging of this question, to bear in mind that the individual cases in which the disease was or was not transmitted by the mother to the fetus must be judged by themselves. For instance, there have been cases described, in which in cattle anthrax has been certainly transmitted by the mother to the fetus; but here it must be remembered that ruptures of small vessels in different organs (lung, intestine, kidney) is of a common occurrence in an animal affected with malignant anthrax, and there is no reason why such ruptures of vessels

and hæmorrhage should not occasionally occur also in the placenta. The blood of such an animal, containing the bacillus anthracis, could in these cases of ruptures in the placenta easily get into the vascular system of the fœtus; all one has to suppose is that some of the capillaries of the fœtal placenta, at one or the other point of hæmorrhage in the maternal placenta, might also have been involved in the process of destruction by pressure or otherwise. In such a case the bacilli might easily pass into the vessels of the fœtal placenta, and further into the fœtal circulation, and here set up anthrax. On the other hand, the placenta of the mother might remain uninjured, and in such a case no bacilli would penetrate from the mother into the fœtus, which would escape anthrax though the mother died of it. The writer has in several instances had the opportunity of dissecting guineapigs and mice that had died (after inoculation) of virulent anthrax; there were present in these animals several fœtuses, whose blood and spleen were carefully examined for the bacilli anthracis, both with the microscope and by culture, yet no bacilli could be discovered in the fœtus. Sections through the whole placenta (maternal and fœtal) showed very clearly that the bacilli anthracis, though abundant in the vessels of the maternal portion, stopped short of the vessels of the fœtal placenta; in fact, there could be seen a sharp line of demarcation between the maternal vessels containing the bacilli and those of the fœtal vessels free from them.

Lingard has shown for anthrax ('Proceedings of the Royal Society,' 1889) that it is possible to inoculate with anthrax the fœtus in utero of the rabbit, without the disease being communicated by the fœtus to the mother; in these instances the blood of the fœtus swarmed with anthrax bacilli, and the fœtus of course died of anthrax, yet the mother remained free of anthrax, thus proving that the bacilli did not pass from the vessels of the fœtal placenta into those of the mother. Although hereditary tuberculosis of the offspring is notoriously a common occurrence, tuberculosis of the mother need not necessarily always be communicated to the fœtus, though in cases of experimental tuberculosis in the guineapig, tuberculosis in the fœtus may be and has been occasionally found: this notably occurs when tubercles are present in the placenta.

But although the passage of microbes from the maternal circulation into that of the fœtus does or does not occur according as the placental vessels are or are not involved in the disease, there can be no doubt that microbes can pass through the wall of the intestine and become absorbed into the system: this is the case in mycosis intestinalis; i.e. anthrax starting from the intestinal canal; in primary intestinal tuberculosis, in tabes mesenterica, and other diseases. Besides, since many infectious diseases have their portals in the lungs (small-pox, measles, tuberculosis, typhus, woolsorters' disease, pneumonia, ragsorters' disease), it is clear that a passage of the specific microbes through the walls of the alveoli is in these instances a notorious occurrence. Similarly, microbes using the alimentary canal as a portal must be capable of becoming absorbed from the intestinal canal; for instance, in milk scarlatina, and in typhoid fever, such must be of normal occurrence. A further important question is how far the microbes can pass unscathed the influence of the gastric juice. The acidity of the gastric juice (0·2 per cent.) is sufficient to kill in a few minutes some species of bacteria which are non-spore-bearing, but it is doubtful whether in all cases and under all circumstances such an effect is exerted or is produced. To quote a few instances only. The bacillus prodigiosus (micrococcus prodigiosus) is a non-spore-bearing microbe, and yet readily passes the stomach in a living state. When added to the food of animals there is no difficulty in demonstrating it, a few

hours after, in the contents of the small intestine: the bacillus of pneumonia (Middlesbrough) is a non-spore-bearing bacillus, but there is no difficulty in infecting mice with it, if mixed with the food; the micrococcus scarlatinae is non-spore-bearing, but the writer has proved that, by adding it to the food of mice, these animals become infected and die; the bacillus of fowl cholera and of fowl enteritis is non-spore-bearing, yet under natural conditions the alimentary canal is the portal of entrance, the animals picking up the contagium with their food from the tainted soil; and there are other similar cases. In all these instances, then, though the microbes are non-spore-bearing, they escape the action of the gastric juice, pass into the intestine in a living state, become absorbed, and produce infection. On the other hand, it has been proved that anthrax bacilli in the non-spore-bearing state do not produce infection when administered with the food, while spores of the anthrax bacilli have been shown to pass unscathed through the stomach, and in some cases have been capable of setting up malignant anthrax (Falk, Koch, Gaffky, and others). Indeed, under natural conditions, we must suppose that when sheep or cattle, grazing and sojourning on infected soil, contract infection with anthrax, they contract it by spores which, having entered into the stomach with the food, have thence passed unscathed into the small intestine, and by becoming absorbed have set up the disease. But this does not apply to all animals and to all cases (Koch, Buchart, and others), since in many experiments, e.g. on mice, ingestion of spores of anthrax bacilli has failed to produce fatal anthrax, while in other cases (Falk, Koch, and Gaffky) it certainly has been followed by positive results. The spirillum of Asiatic cholera is a non-spore-bearing microbe; it is killed on exposure for a few minutes to the action of hydrochloric acid of 0.2 per cent. (Koch, Watson Cheyne); yet Macfadyean has shown that when the microbes are administered with the food (milk) to dogs, they pass unscathed through the stomach, while with water they do not do so. Koch and others have shown that when this microbe is administered with the food to the guineapig, it does not pass unscathed through the stomach, unless the contents of this organ be previously made neutral or alkaline (*see* a further chapter). There can then be no doubt about the fact that the gastric juice has an inimical effect on many non-spore-bearing microbes, but it does not follow—indeed, experience disproves it—that under all conditions non-spore-bearing microbes are killed in their passage through the stomach. Whether the cause for this is to be found in particles of the contents of the stomach not becoming sufficiently exposed to the action of the gastric juice, or in the condition of the gastric juice itself—not always being of the same degree of acidity—or to both, or to some other condition, is undecided; the fact remains that non-spore-bearing microbes administered with the food do in some instances pass unscathed through the stomach, spore-bearing ones easier and oftener. The tubercle bacilli, which as experiment proves are spore-bearing, pass always unscathed through the stomach, and produce tuberculosis of the intestine. Anthrax spores often pass and set up malignant fatal anthrax.

CHAPTER XI

DISINFECTION

IN the foregoing chapters we have mentioned various agencies which, though not destructive of the life, are nevertheless inimical to the growth and multiplication of bacteria; and further, other conditions which are capable of altogether destroying their life. We will now proceed to systematically consider these

agencies. We may subdivide them into two great categories, just indicated, viz. influences which inhibit the growth and multiplication of bacteria, these are called restraining influences; and those that altogether annihilate the life, these are called killing influences.

A. RESTRAINING INFLUENCES

As such are to be regarded (a) temperatures below 12 to 14° C. and above 40 to 42° C. It will be, however, remembered that we mentioned several species which can well exist and multiply below 12 or 14° C., e.g. some water bacteria, and bacteria in the soil; while others, e.g. *Bacillus thermophilus*, grow well at temperatures far above 42° C. There exist some species on which temperatures above 25° C. have a restraining power, e.g. *Bacillus prodigiosus*, the bacillus of veal pie, and a spore-forming bacillus of dust; then there are other species on which a temperature below 25° C. has a restraining power, e.g. a streptococcus of the fluid of the mouth, the streptococcus of Schütz, causing infectious pharyngeal abscess in the horse, the diplococcus pneumoniae, and the tubercle and leprosy bacilli.

These restraining influences of certain temperatures do not possess any direct killing effect on these several bacteria.

(b) *Moisture*.—Absence of sufficient moisture is in itself a powerful restraining agency, for no growth can take place if the amount of moisture is insufficient. In a dry atmosphere or under conditions causing dryness or absorption of much moisture, e.g. caustic potash or soda, quicklime, or sulphuric acid, kept side by side with the culture medium in a closed space, no growth takes place.

(c) *Light*.—Diffuse daylight and sunlight have a restraining power on some species more than on others, for all bacteria grow best in the dark. Direct sunlight has, as has been mentioned, a powerful killing power.

(d) *Unfavourable Soil*.—Acidity of the soil is a powerful restraining influence; in many cases, if strongly acid, exerting a killing influence on non-spore-bearing bacteria. Strong alkalinity of the medium is likewise a restraining condition. The presence or absence of certain substances, as we have seen, is a powerful influence: e.g. the presence of sugar is unfavourable to some bacteria, while it favours others; the presence of minute traces of phenol, of perchloride of mercury, of iodine, the absence of sodium and potassium salts, and particularly the absence of sufficient proteid material, have each a powerful restraining influence on many species; the presence and accumulation of the chemical products, a result of the multiplication of the bacteria themselves, have a restraining influence; the presence of various substances such as occur in faecal matter acts injuriously on the multiplication of some bacteria; the presence of other more vigorously growing bacteria interferes with the growth and multiplication of some less vigorous bacteria. Examples of this kind have been mentioned in a former chapter, as also the influence of putrefaction on some pathogenic bacteria.

The methods for testing the restraining power of carbolic acid, perchloride of mercury, and others are briefly these: The disinfectant is mixed with nutrient broth or nutrient gelatine in definite proportions; this mixture is then inoculated with a trace of the bacterial culture in the ordinary manner, and on incubation the result is watched, viz. whether no growth appears, whether the growth is normal both as to time, aspect, and amount, or whether it is retarded, different in character, and less in amount.

Carbolic acid added to the nutritive medium in the proportion of 1 in 400 to 500 has a decided restraining power; perchloride of mercury added

1 : 20,000–50,000 has a restraining power on some (*bacillus anthracis*, *bacillus mallei*, *streptococcus scarlatinæ*), while it has no restraining power in these proportions on others (*bacillus subtilis*, *spirillum* of Finkler and Koch, *staphylococcus pyogenes aureus*, *bacillus prodigiosus*, *streptococcus pyogenes*, and some others). In these latter cases the amount of perchloride required is 1 : 10,000, or even greater.

B. KILLING INFLUENCES

(a) *Temperature*.—It has been mentioned that with few exceptions in all non-spore-bearing forms of bacteria exposure to a high temperature, such as 75° C., 70° C., 60° C., or even 58° C., for several minutes [70° C. for one minute, 58° C. for 5 to 10 minutes] kills the bacteria; further that the spore-containing bacilli and the free spores withstand considerably higher degrees of temperature. Between the very resistant spores found in some tinned food-stuffs by Cohn not killed by exposure to the temperature of boiling water for half an hour, and the spores of *bacillus anthracis* killed by boiling in from one half to one minute, there exist all intermediate degrees of resistance. Thus, for instance, the spores of hay bacillus must be kept in boiling water for six minutes before they are killed with certainty; the spores of the Jequirity bacillus do not survive boiling for seven minutes; the spores of the *bacillus filamentosus* of sewage are killed by five minutes' boiling; the spores of the scurf bacillus and of some potato bacilli just escape being killed by five minutes' boiling.

As regards the killing power of the temperature of 100° C., i.e. of boiling water, it has been shown by Koch and others that flowing steam, i.e. steam in movement, is the most efficient, dry heat the least efficient. Dr. Parsons's report to the Medical Officer of the Local Government Board in 1886 on Disinfection by Heat gives a full account of the means employed and the results obtained. The spores dried on silk threads previously sterilised, or on bits of calico or flannel previously sterilised, are exposed directly to the high temperature, and with them inoculation is made of broth tubes or of gelatine plates and the result watched. Or, in order to test their resistance to boiling water, sterile broth is inoculated with the spores from a pure culture, and this broth tube is then exposed to the temperature of boiling water (over the flame) for the desired time. If the influence of water of any given degree up to 100° C. is to be tested on bacteria or their spores, broth tubes are inoculated with the culture and exposed to the desired temperature in an apparatus which is described in the report of the Medical Officer of the Local Government Board for 1889. The writer has found it easy to keep the temperature of the water in the apparatus constant by a small flame of the Bunsen burner; alterations of quarter to half a degree Centigrade are the utmost differences noticeable. The temperature of the broth in the test tubes is, as a rule, equalised within half a minute to about half a degree Centigrade, so that if it is desired to expose the broth in the test tubes to, say, 60° C., the temperature of the water in the apparatus is kept at 60 to 60·5° C. When the tubes have been kept in the water for the desired period they are taken out and at once placed in cold water, so as to immediately stop any further influence. Just like broth tubes, gelatine tubes can also be heated, and then with these plate cultivations can be made. Owing to the great resistance of spores to high temperature, differentiation of spores from a mixture of bacteria can be easily made by heating the mixture up to 80–90° C., for all bacteria except the spores are hereby killed without fail, the spores remaining alive. For example, if a culture containing spores of hay bacillus, that happens to be contaminated by micrococci and

non-spore-bearing bacilli, be heated to a temperature of 80 to 90° C. and then from it fresh broth or gelatine be inoculated—as tubes or as plate cultivations—the result will be pure cultures of the hay bacillus only.

(b) *Drying*.—By thorough and complete drying, all bacteria are killed, except spore-bearing ones and the spores themselves. The spores of all the known species resist drying for indefinite periods. The writer has kept the spores of various potato bacilli, of bacillus anthracis, of hay bacillus, of scurf bacillus and of Jequirity bacillus, contained in agar tubes, in a perfectly dry state, i.e. the tubes were kept in a closed desiccator over sulphuric acid till the agar covered by the culture had dried up into a thin shrivelled film:—and yet even after two years and a half on inoculating fresh materials from the above tubes typical and good growths were obtained. Non-spore-containing bacteria, e.g. various species of staphylococci, various species of streptococci, bacilli of typhoid fever, of swine fever and swine erysipelas, Koch's and Finkler's spirilla, bacilli of pneumonia, bacilli of fowl enteritis and fowl cholera, of grouse disease, as well as a number of other species of bacteria, were kept—as agar cultures—in the incubator till these latter had well dried up; no sub-cultures could then be raised from any of them. On drying non-spore-bearing bacteria over sulphuric acid, in a brisk current of air, or simply placing a very thin film on cover-glasses in the manner employed by Koch and leaving them till dry, such as the bacillus anthracis of the blood or spleen, Koch's or Finkler's spirilla, the bacillus of typhoid fever, staphylococci or streptococci, bacillus of swine erysipelas or swine fever, bacillus of pneumoniæ (Middlesbrough), bacillus of fowl enteritis and fowl cholera, and many others, and then placing these cover-glasses in broth or on gelatine and incubating them, no growth of the above bacteria takes place. If the drying is not complete, that is, if the film be too thick, so that the superficial layer forms a protecting crust for the rest, then the result of inoculation with such material will be positive; the bacteria in the depth, having been protected against drying, survive, and can produce a new crop. Similarly when particles of solids containing bacteria are dried, it will be found that here also the centre escapes thorough drying, being protected by a superficial crust. Ordinary dust particles, however small, are never so dry that the bacteria contained in them are killed. The proof of this is, that numerous species of non-spore-bearing bacilli and of micrococci can be cultivated from dust, be it dust floating in the air as motes visible to the eye only by Tyndall's searching beam of light, or be it dust of a floor or of the wall of a room, or on any other object.

(c) *Light*.—The disinfecting power of the direct sunlight has been mentioned above.

(d) *Chemical Disinfection*.—Koch, his pupils, and others have tested a variety of substances, such as alcohol, iodine, lime, carbolic acid, sulphate of iron, permanganate of potassium, tar, arsenic, perchloride of mercury, and others. As is obvious, according to the nature of the bacteria, according to the strength of the solution and the time of exposure, the result will be found different: for example, bacilli anthracis of the blood, i.e. sporeless bacilli, are killed by 5 per cent. carbolic acid in five minutes, but they are not injured by 1 per cent. carbolic acid in five minutes. Bacilli anthracis of the blood are killed by 1 : 20,000 perchloride of mercury after ten minutes' exposure, but remain uninjured after five minutes' exposure.

The methods used most efficiently for testing the killing power of any reagent are these: (a) exposure of the bacteria to the reagent in solution; either by mixing a trace of the culture with a comparatively large amount of the reagent, or by placing in the reagent silk threads, a piece of blotting-

paper, or other similar material previously charged with the culture, and then inoculating with a trace from this about 8 to 10 c.c. of sterile broth, contained in a test-tube, and exposing this to a temperature of from 85 to 87° C., or making plate cultivations in gelatine.

If growth appears, note the rapidity and character of the growth as compared with control tubes, i.e. tubes inoculated with the non-medicated bacteria; if doubtful whether the growth is of the right character and pure, make fresh plate cultivations.

(β) If the bacteria possess definite pathogenic power, inject, after exposure to the reagent, a small quantity of the medicated culture into a suitable animal and watch the result. We will illustrate this by giving three examples: (1) Koch's cholera spirilla; (2) anthrax bacillus of blood; and (8) spores of bacillus anthracis.

(1) *Koch's Cholera Spirilla*.—From an active culture a droplet is mixed with the disinfectant, contained in a test-tube plugged with cotton wool, or in a sterilised watch-glass covered up with another watch-glass; after the lapse of the desired time, by means of the capillary pipette or the platinum hook inoculation is made from the mixture into a broth tube containing about 8–10 c.c. and into a gelatine tube; the former is placed in the incubator at 85–87° C., the latter is melted, shaken, and a plate cultivation is made. If the broth remains clear after forty-eight hours' and longer incubation, and if no characteristic colonies appear in the plate, the spirilla have been killed by the disinfectant.

(2) A droplet of blood of the heart or spleen of an animal dead of anthrax is mixed as above with the disinfectant; after the lapse of a certain time inoculation is made of broth tubes, of gelatine tubes for plate cultivation, of gelatine tubes in stab cultures, and at the same time a guinea-pig is inoculated with several drops of the mixture. If the bacilli have been killed by the disinfectant, none of the tubes and plates will show the characteristic growth of the bacilli anthracis, and the animal remains perfectly normal; but if the bacilli have not been killed, either of two results follow: (a) either the tubes show normal growth in the normal time, and the animal succumbs to anthrax in the normal time—thirty-six hours to three days—or (b) the growth resulting on inoculation is retarded and smaller in amount, and the animal though showing swelling at the seat of inoculation does not die of anthrax, or dies after a prolonged period (six, seven, or even nine days). In the latter case the bacilli have evidently been injured though not killed by the disinfectant.

(8) A trace of spores—from an agar or broth culture, or from a potato culture—is dried on silk threads, and these are then placed in a solution of the disinfectant for a stated time and then placed in broth or gelatine; or a trace of the spore material is mixed as above in a test-tube or watch-glass with the disinfectant. The testing of the effect is carried out as in (2).

Of all disinfectants hitherto examined, perchloride of mercury solution deserves the first place as a thorough disinfectant, for a solution of 1 : 500 (one grain to about one ounce of water) applied to any material containing spores of even the most resisting kind, e.g. those of hay bacillus, or of tubercle bacilli, sterilises the spores after an application not lasting more than a minute; one in a thousand sterilises most though not all spores when acting from five to ten minutes; one in five thousand kills all non-spore-bearing forms with certainty, even after one-half to one minute's immersion. In the application of perchloride of mercury as of all those agencies which have an affinity to proteids either by coagulating them, as, for instance, alcohol, and perchloride of mercury, or by oxidising them, e.g. sulphate of iron, and permanganate of

potassium, or by decomposing them like chlorinated lime—it must be borne in mind that if any material containing proteids is to be subjected to disinfection, the disinfectant must be added in considerable excess in order to make allowance for the waste necessary in the action on the proteids themselves, so that sufficient of the active disinfectant remains to kill the bacteria. Thus, for instance, in the case of typhoid stools, cholera stools, tubercular sputum, sputum of croupous pneumonia, &c., a considerable excess of the disinfectant must be added, whereas in experiments with pure cultures, taking of these a trace only, the excess need not be so great.

Besides perchloride of mercury, 5 to 10 per cent. carbolic acid, chlorinated lime,¹ iodoform, iodine, bromine, tar (1 per cent.), and lime, are good disinfectants. With this latter the writer has had occasion to make a considerable number of experiments to test its disinfecting power on various bacteria, both as lime milk and as lime with herring brine (the amine process). In the latter case, viz. lime and brine, the amount of lime is effective in smaller proportions than when lime alone is used. Seventy grains of lime and six grains of herring brine to the gallon of water are mixed and then immediately added to the fluid which is to be disinfected; a voluminous precipitate is formed, which soon settles to the bottom of the vessel; while the supernatant liquid is clear and practically sterile. In the case of non-spore-bearing microbes the precipitate is not quite sterile, but the living microbes become in a few days reduced to a large extent.

In the case of sewage the amine treatment yields a practically sterile effluent, and in the sludge the number of surviving microbes diminishes from day to day. The effect of milk of lime has been tested for comparison on a good many species of bacteria, and its disinfecting power when used in the same proportion of strength was found to be considerably less.

SECTION B

CHAPTER XII

SPECIFIC OR PATHOGENIC BACTERIA

Suppuration and Acute Phlegmon.—Acute phlegmon is a disturbance of the connective tissue initiated by vascular alteration. This alteration manifests itself in engorgement; the arteries, capillaries, and veins are dilated and filled with blood, the circulation in the arteries, capillaries, and veins is at first accelerated, then it becomes slower in the capillaries and veins, and the blood in some of these ultimately undergoes stasis. These changes have been observed under the microscope by Cohnheim, and have been graphically described by him in Virchow's 'Archiv,' Band XL. At the same time diapedesis of red corpuscles (in capillaries), particularly emigration of white blood-corpuscles (in capillaries and veins) and exudation of plasma (in arteries and veins), occur, whereby the connective tissue of these parts becomes swollen, the bundles more or less separated one from another, and the lymph-spaces of the connective tissue filled with plasma, red and particularly white corpuscles. As the process continues the matrix of the connective tissue undergoes retrograde changes, both in its fibrous matrix and in the connective tissue corpuscles: the fibrous matrix breaking down, many of the connective tissue corpuscles

¹ Nissen on chlorinated lime as disinfectant, *Zeitschr. f. Hygiene*, VIII. 1.

swelling up, becoming vacuolated, and ultimately degenerating; but active changes, enlargement and division of these are also maintained by some observers (Stricker and Norris). The increase of leucocytes or round cells in the connective tissue both by continued emigration as well as active division of those that have emigrated leads to the formation of at first small cells, then according to the size of the inflamed area to large accumulations of them: these are the pus-cells, which when localised form an abscess.

As this accumulation of leucocytes and plasma proceeds, the connective and other tissues around it become gradually involved in the process; the phlegmon and abscess enlarge at the expense of these tissues, and they themselves undergo degenerative changes and are broken down. In consequence of this, when in the skin or in mucous membranes the surface is gradually reached, the abscess discharges itself and an open sore or ulcer is established; then the process of granulation, healing, and cicatrisation commences from the tissue next to the base of the ulcer.

It is not our object to trace and describe all the details of the pathological changes going on in the connective and other tissues during this process of phlegmon, nor the localisation of the accumulation of leucocytes, as abscess, nor the ulceration and healing, but we will limit our description to their fundamental causes.

In all acute phlegmons, either of the nature of diffuse infiltration with much plasma and relatively few leucocytes (œdema), or with comparatively little plasma and many leucocytes (purulent inflammation and abscess), the leucocytes or round cells form the most conspicuous formed elements; as to other formed elements, notably the micro-organisms present, a clear insight can only be had if the inflammatory matter, exudation, or pus be taken from a phlegmon or abscess which is still removed from the free surface; for the matter taken from an exposed surface, such as an open sore of the skin or mucous membrane, or the exudation of the surface of an inflamed mucous membrane, as in catarrh, contains various species of microbes, as was mentioned in a former chapter, not necessarily connected with the diseased process; besides, an inflamed tissue, particularly one in which there is a good deal of breaking down of the tissue elements going on, is a suitable soil for the growth and multiplication of various kinds of saprophytes not having any relation to the disease.

In *acute abscess*, preparations made of the inflammatory matter or of the pus, taken from the depth, reveal in most cases micrococci (Ogston, Rosenbach) in variable numbers; they are found either singly or in dumb-bells, or even in short chains, or in smaller or larger clumps; as a rule, most are suspended in the plasma; here and there one sees them attached to the surface of a pus cell, or to the surface of a particle of *débris* or other elements not pus cells, e.g. epithelial or connective-tissue cells, muscle fibres, or connective-tissue bundles, &c., or one finds them enclosed within the substance of pus or other cells. In sections through phlegmonous tissue one sees them in more or less continuous masses pervading the tissue, in lymph-clefts and in blood-vessels in which stasis has occurred, particularly in the parts most disorganised. These micrococci stain readily by the ordinary methods, and when cultivations (streak cultures or plate cultivations) are made of such pus, either one of the following species, or sometimes two together, can be isolated in large numbers of colonies (*see* the literature on the subject by Rosenbach, Paltauf, Bonone, and others).

(1) *Staphylococcus pyogenes aureus liquescens*.—This is the most common species in acute purulent inflammation, and in sores following the discharge of abscess. As its name *staphylococcus* indicates, it forms by rapid multipli-

cation masses and aggregations ; its colonies in gelatine, and particularly on agar, have a more or less yellowish-orange tint, and it rapidly liquefies gelatine. In gelatine plate cultivations the colonies appear on the second day as minute whitish yellow dots, which during the third day in transmitted light have a brownish tint, and are situated in a somewhat depressed pit, due to the gelatine surrounding it having become liquefied. During the fourth and fifth days the circular colony increases in size, and assumes in reflected light a more or less orange tint, the liquefaction has become considerable, forming a broad clear circular zone around the colony, which has sunk to the bottom of the liquefied zone, the zone of liquefied gelatine remaining clear and limpid. In stab culture in gelatine the stab is indicated as early as the second day as a line of whitish dots more or less closely placed together. Liquefaction begins from the upper part of the stab ; during the third and fourth days the liquefaction is considerable, the droplet-like dots are deposited at the lower end of the channel of liquefaction, and are of a golden yellow colour, the liquefied gelatine being clear and limpid. In streak cultures on agar the streak is indicated by a broad band, smeary like paint, and of a golden yellow colour ; the band rapidly increases in breadth, is moist looking, and its outline is more or less knobbed ; after a few days (four to six) at 87° C. the band reaches its maximum growth, and its colour then gradually becomes paler, which change may go on till it is only just slightly yellowish tinted. In broth at 87° C. it grows very rapidly (*see* a former page) and produces strong uniform turbidity.

This micrococcus, as stated above, is very common in pus of acute abscess ; it is present also in many other purulent inflammations, primary and secondary, acute osteo-myelitis, catarrhal inflammations of various kinds. There is one important addition to be made to what has been just stated as to its biological character, and that is that there exist several varieties of this microbe, slightly different one from another : the differences consist in the rapidity of the growth, in the rapidity of the liquefaction of the gelatine, and in the tint of the colour of the growth on nutrient agar. The writer has isolated from pus of acute traumatic abscess in the human subject a *staphylococcus aureus liquescens*, exactly like the one described above as the typical *staphylococcus aureus liquescens* ; while from the pus in acute abscess of the udder of the cow a *staphylococcus aureus liquescens* was isolated, that grows more slowly than the former, which liquefies more slowly, and of which the colour on nutrient agar is less golden and fades more rapidly ; then from infectious ulcer of the teats and udder of cows (Wiltshire disease), and from the infectious ulcer of the teats of cows affected with the Edinburgh disease, a *staphylococcus aureus liquescens* was isolated, which in all media except broth compares with the typical *staphylococcus aureus* ; again, from opaque human vaccine a *staphylococcus aureus liquescens* was isolated, which behaved like the typical human *staphylococcus aureus liquescens*, except that it did not fade, and its colour on agar was and remained deep golden ; from the blood of grouse dead of the grouse disease the writer obtained a *staphylococcus aureus liquescens*, which, though the same as the typical *staphylococcus aureus liquescens* of human pus in broth and gelatine, produced on agar only a faintly yellow tinted growth. Finally the writer has several times come across *staphylococcus aureus* (from blood, from air, from dust) which did not liquefy gelatine, but in all other respects was similar to the classical *staphylococcus aureus*.

The typical *staphylococcus aureus liquescens* of human pus and phlegmon, while recent, produces abscess and purulent phlegmon on inoculation under the skin of rabbits and guinea-pigs, but it gradually loses this power on

continued sub-cultures ; besides, the amount necessary to produce purulent inflammation must be always considerable. Bujwid ('Centralbl. f. Bact.,' 1889) has shown that the pyogenic power of this microbe is enhanced by simultaneous injection of a 12 per cent. sugar solution.

Occasionally inflammation and metastatic abscesses are produced in the rabbit in the lungs, in the pericardium and peritoneum, the liver and spleen by intravascular injection of large quantities of this organism. In all these localities the staphylococcus aureus liquescens can be demonstrated in cover-glass specimens and by cultivation. (For its relation to ulcerative endocarditis see a further page.)

Acute purulent abscess of the marrow of tubular bones in the rabbit has been produced by Becker on injecting the staphylococcus aureus liquescens, traumatic injuries of the bones having been previously established; hence it is probable that acute purulent osteo-myelitis also in the human subject is the result of an infection with the staphylococcus aureus liquescens.¹ The writer has made numerous experiments with some of the species of the staphylococcus aureus liquescens above mentioned, but has not been able to produce pathogenic results.

Duclaux and especially Heydenreich (St. Petersburg, 1888) describe the presence of staphylococcus aureus (or a variety of it) as the cause of the chronic purulent disease known as the 'Aleppo bubo' or the 'Pendje tumour'; tumours appear on the skin of the face and extremities, which in the course of a year soften into abscess, open, and then heal. Duclaux found the cocci even in the blood. Cultures of it injected into rabbits produced local gangrene, and in large doses septicæmic infection.

(2) *Staphylococcus pyogenes albus liquescens*.—This organism differs from the former only in the fact that its colonies on gelatine are, and remain, white in reflected light; on nutrient agar it forms a moist-looking whitish uncoloured film. Its power of liquefaction, and the rate of its growth, are the same as in the former. This staphylococcus albus liquescens is common in pus, both of acute abscess as also in various kinds of purulent matter; it is found in air, in dust, and in acute and chronic inflammations; it is also found in secondary inflammation, such as in purulent pyæmic inflammations in the joints, in the glands, in the serous membranes, in the lungs, following infectious diseases like small-pox, diphtheria, scarlet fever, and typhoid fever. In pus of acute abscess, and in purulent catarrhal inflammation (rhinal, bronchial, vaginal, urethral, conjunctival), it is easy to isolate this species by plate cultivation. When its culture is obtained from any purulent matter, and is subcutaneously inoculated in rabbits or guineapigs in sufficient quantities, it produces acute purulent inflammation, either as general diffuse purulent inflammation, at and about the seat of inoculation, or as localised abscess. By continued sub-culture it soon loses this pathogenic power. Several (one to two) cubic centimetres of a recent broth culture injected into a vein of a rabbit produce death in a few days by disseminated inflammation and abscesses in the viscera, in all of which the staphylococcus albus liquescens can be demonstrated by cover-glass specimens and culture. Acute septicæmic infection, and death in twenty-four to thirty-six hours, are occasionally produced in rabbits, the blood of the general circulation containing the microbes in large numbers.

(3) *Staphylococcus pyogenes albus non-liquescens* is another species, occasionally obtained from phlegmon and pus. As its name indicates, it

¹ On the relation of staphylococcus aureus to osteo-myelitis, and the experimental production of the latter by intravenous injection of the former, see Rodet (*Revue de Chirurgie*, 1885), and particularly Lübbert (Würzburg, 1886).

is white in streak cultures on gelatine and agar, and it does not liquefy gelatine; it grows rapidly, but not so rapidly as either of the two previous ones; its colonies are more or less round whitish dots. Also of this there exist various species, differing from one another in some details, e.g. in rapidity of growth, in outline of streak cultures, and in size. In broth, the differences are more pronounced, since one produces more turbidity than another, one causes a greater precipitate than another, and one causes something like a pellicle, the others not. The staphylococcus albus non-liquescens is found in many other materials besides phlegmon and pus, e.g. occasionally in air, dust, and soil, in the secretions of the nose and fauces, also in drinking water and in sewage; from many putrid materials several varieties can be isolated.

Obtained from phlegmon, and inoculated into the subcutaneous tissue of rabbits and guineapigs, purulent inflammation follows in a certain percentage of cases, provided large quantities be injected.

(4) *Staphylococcus pyogenes cereus albus* (non-liquescens) and *staphylococcus pyogenes citreus* (non-liquescens).—The first differs from the previous one in that its colonies and its streak cultivation are of a waxy white appearance; the colonies grow in the course of a week or so into round flat patches, they are thin, flat, and more or less sharply outlined, more or less dry, and look like white wax. This is not often obtained from phlegmonous tissue. It is not unfrequently obtained from vaccine lymph. No result is obtained on inoculation into animals. The second of the two, as its name indicates, forms greenish yellow growths.

(5) *Streptococcus pyogenes* (Ogston, Rosenbach,¹ Krause, Passet).—This occurs frequently in acute phlegmon, though it is oftener found in the fluids and tissues of chronic purulent processes, in pneumonia, in necrotic and purulent matter of secondary infections: in abscess and necrosis of lymph glands, of the liver, in synovial and serous cavities following or associated with ulceration of the skin and mucous membranes. This microbe forms longer or shorter chains when grown in fluid media; on solid media the chains are not very pronounced. Its colonies grow slowly on gelatine; after two or three days they appear as whitish grey dots, which only gradually enlarge; on the surface they grow into irregularly outlined, more or less circular patches, thicker in the middle than at the periphery, which latter becomes gradually serrated or irregularly fringed; the colonies grow better on the surface than in the depth of the gelatine, where they remain small droplets. In stab cultures in gelatine the stab is indicated after two days as a grey or whitish line, which under the microscope is made up of closely placed minute droplets; in streak culture the line of inoculation is indicated after two to three days by a series of minute grey or white dots: these increase in size and number, as growth proceeds, but the streak is always made up of separate dots, which when closely placed become more or less fused one with another. After about a week's growth the streak and colonies are white when seen in reflected light, light brown in transmitted light.

The streptococcus pyogenes obtained from pus or fluid of acute phlegmon or of pus of chronic abscess forms fine long chains in broth or in the condensation fluid in agar tubes. In broth at 35–37° C. it forms a uniform slight turbidity after two to three days; then flakes appear which at the bottom of the tube form a more or less coherent viscid whitish precipitate. In this stage cover-glass specimens show long curved chains of the character shown in fig. 27.

¹ Rosenbach: *Mikroorganismen bei den Wund-Infektionskrankheiten des Menschen*, Wiesbaden, 1884.

When recent cultures are inoculated into the subcutaneous tissue of rabbits, a distinct blush and slight swelling is noticed after twenty-four hours, after forty-eight hours, or at latest after three days, the blush has passed away ; in some instances the swelling at the seat of inoculation increases, and a minute abscess is noticed now or during the next following days. In guinea-pigs a thickening and inflammation are noticeable at and about the seat of inoculation after a day or two, which increases during the next few days, but in most cases has passed away by the end of the week ; in very few instances a purulent abscess is found after four or five days. In white mice, in only a small percentage of cases is an abscess formed ; but if large quantities are injected, necrosis of the subcutaneous tissue occurs, and in some instances acute fatal septicæmia, i.e. death after thirty-six to forty-eight hours. In these cases the spleen is found enlarged and all the viscera greatly congested ; in the blood and the spleen the streptococci can be demonstrated by cover-glass specimens and by culture. In the rabbit intravascular injection of even large quantities produces no result, unless the animal has been previously made ill by the injection of toxic substances ; under these conditions a general infection with the streptococci is produced, and the animal dies in two to five days ; ulcerative endocarditis is noticed, if previous to the injection injury to the aortic valves has been produced (Wyssokowitsch).

It is maintained by many observers that the streptococcus pyogenes, such as occurs in acute, sub-acute, and chronic phlegmon, is always the same species, and is identical with the one isolated by Fehleisen in erysipelas. The writer is confident that the name streptococcus pyogenes does not cover only one species, but is the name of a group of different species, varying in many details, though they all have a number of characters in common ; only on closer observation in the different media and by the result of inoculation of animals can these differences be made out. The streptococcus pyogenes obtained from acute phlegmon is most decidedly not the same as the streptococcus pyogenes which is obtained from chronic necrosis ; it is not the same as that obtained by Fehleisen from erysipelas ; nor is it the same as that obtained by the writer from scarlatina, although they may and do occur together ; e.g. in erysipelas, both the streptococcus pyogenes and the streptococcus erysipelas were found together by Fehleisen, the former in the older or central, the latter in the younger or peripheral part of the erysipelatos patch. In scarlatinal cervical glands the writer found the streptococcus pyogenes together with the streptococcus scarlatinæ ; so also in the ulceration of the teat in the Hendon cow disease, in the superficial part of the ulcer the former, in the depth of the inflamed skin and subcutaneous tissue the latter, was found. In the secondary inflammatory foci following scarlatina and diphtheria, the streptococcus pyogenes has been isolated by Löffler and others, and also in the cutaneous eruption of infantile hereditary syphilis.

To say that, because the streptococcus pyogenes of acute and chronic phlegmon is in its cultural characters extremely similar to the streptococcus erysipelatos, both are identical, would be illogical and contrary to clinical observation. An acute simple phlegmon or ulcer, acute or chronic, is not erysipelas, this latter being as well defined an acute infectious disease as, say, anthrax is, both as to its pathology and symptoms. Streptococcus pyogenes is found in the tissue of many ulcerations, yet no erysipelas follows ; streptococcus pyogenes is obtained occasionally from pustules, yet in these cases there is no sign of any erysipelas. Streptococcus pyogenes obtained from acute or chronic abscess and inoculated as recent cultures into the rabbit's ear does not produce that distinct erysipelas that Fehleisen's true

streptococcus erysipelas does. Some observers find no difference in the result of inoculation into the rabbit's ear between the *streptococcus pyogenes* of acute phlegmon and the *streptococcus* isolated from erysipelas; but this might be due to the fact that they had not got hold of the *streptococcus erysipelas*, although taken from erysipelas, or that they used an attenuated culture. It is, however, right to mention that the *streptococcus erysipelas* by continued sub-culture loses gradually the power of producing erysipelas.

The *streptococcus pyogenes* as obtained from acute or chronic phlegmon is not identical with the *streptococcus* shown by Koch to cause progressive necrosis in mice, although both are extremely alike. The *streptococcus pyogenes* is not identical with some species of *streptococcus* which can be obtained from the fluid of the mouth, from soil, and from ulcers. There are *streptococci* which though similar to the *streptococcus pyogenes* are yet not identical, e.g. a *streptococcus* obtained from ulcers on the teats of cows (Wiltshire disease), which does not grow at temperatures above 25° C.; a *streptococcus* from the fluid of the mouth, which does not grow below 25° C.; Schütz described a *streptococcus* connected with, and causing infectious pharynx-abscess in the horse (Druse), which does not grow below 25° C.; Löffler obtained a *streptococcus articulorum* from diphtheria; Flügge described a *streptococcus malignus* which causes acute septicæmia in mice; Nicolaïer described a *streptococcus septicus* which he found in soil. All these *streptococci* are similar, and only on careful cultural and experimental observation can they be distinguished from one another, though some of them with difficulty, even under these conditions.

(6) *Micrococcus tetragonus* is occasionally found in pus. Gaffky first described it in the purulent sputum of pulmonary tuberculosis, but it has been of late years also found in other localities, notably, purulent inflammations connected with tubercle. The chief character of the coccus is its arrangement in fours. It grows fairly rapidly in gelatine, forming round yellow colonies, on the surface of the gelatine white prominent droplets. This microbe acts virulently on mice and guineapigs, particularly on the former, which on subcutaneous inoculation with small doses succumb in the course of three or four days from septicæmia, the blood-vessels of all viscera containing the microbes.

(7) *Bacillus pyocyaneus* is the microbe found in blue-green pus—in fact, it is the organism which produces the blue-green colour. Gessard and Charrin (Gessard: Thèse de Paris, 1882. Charrin: Communication à la Société anatomique, December 1884) first described the microbe. Gessard particularly isolated the blue pigment produced by it, pyocyanin. When isolated by gelatine plates from blue-green pus the microbe grows as translucent colonies irregular in outline and showing a fine radial striation, the gelatine gradually assuming a greenish colour. The gelatine is liquefied and of a uniformly greenish colour; on agar it forms a white film, while the agar becomes tinted greenish; on potato it forms a brownish film, while the substance of the potato underneath assumes a greenish colour. It has pathogenic action on guineapigs.¹ Under the microscope it is an extremely minute and thin cylindrical rod (see fig. 26).

(8) *Bacillus pyogenes fetidus* of Passet is the bacillus which is found in malodorous pus. This microbe is a short rod, generally arranged as dumb-bells: it shows sluggish motility, and is about 1.45 μ long and 0.58 μ thick.

¹ When a few divisions up to half a Pravaz syringe of the broth culture be injected subcutaneously, the animals become ill and die in from two to four days, showing peritonitis, pericarditis, and pleuritis, with copious membranous and purulent exudation, which contains abundantly the bacilli.

In gelatine plates it forms round white colonies, thicker in the centre than in the periphery. On potato it grows as a brown thick film. Small doses inoculated in animals produce no effect, larger doses sometimes abscess. (Passet.)

Karlinski gives as the result of a large number of observations on purulent matter of man the following list ('Centralbl. f. Bact. und Parasit.,' VII., No. 4, p. 115):—

Disease	<i>Staphylococcus pyogenes aureus</i>	<i>Staphylococcus pyogenes citreus</i>	<i>Staphylococcus pyogenes albus</i>	<i>Streptococcus pyogenes</i>	<i>Micrococcus tetragonus</i>	<i>Bacillus pyogenes foetidus</i>	<i>Bacillus of Friedlander</i>	<i>Bacillus anthracis</i>
Mastitis, 36 cases . . .	22	4	4	6	—	—	—	—
Subcutaneous abscess, 30 cases . . .	10	2	8	6	2	2	—	—
Phlegmon, 24 cases . . .	—	—	—	24	—	—	—	—
Furuncle, 20 cases . . .	9	—	10	—	1	—	—	—
Bubo, 17 cases . . .	8	1	1	7	—	—	—	—
Subperiosteal abscess, 16 cases . . .	6	—	10	—	—	—	—	—
Panaritium cutaneum, 16 cases . . .	7	—	9	—	—	—	—	—
Abscess of gums, 10 cases . . .	1	—	4	1	3	1	—	—
Hordeolum, 10 cases . . .	6	—	4	—	—	—	—	—
Otitis media, 4 cases . . .	2	—	—	—	—	—	2	—
Carbuncle, 4 cases . . .	2	—	1	1	—	—	—	4
Osteomyelitis, 3 cases . . .	2	—	1	—	—	—	—	—
Summary . . . 190	75	7	52	45	6	3	2	4

The three species, *staphylococcus pyogenes aureus*, *staphylococcus pyogenes albus*, and the *streptococcus pyogenes*, from their distribution deserve then the most close consideration, and the question that at once presents itself is this, Are these microbes the cause of the disease? There can be little doubt from what we have seen above, that experimentally the three species can produce primarily inflammation, and there can be also no doubt that, in a variety of secondary inflammations in man, following sometimes other infectious disorders, as scarlatina, diphtheria, variola, typhoid fever, measles, one or the other species of these microbes is found in the foci of those secondary inflammations. Now, it may be admitted that their presence in these secondary foci might not be the primary cause of the foci, but only the result, for it is quite possible to imagine that, from one cause or another, a loss of vitality of a particular tissue having been established, these microbes derived from a diseased surface can pass into such a disorganised tissue, and can here multiply (*see above*, Wyssokowitsch's experiments). That the actual inflammation and suppuration in these secondary foci are due to these microbes, is shown by the fact, that experimentally they can produce suppuration. That also in the primary inflammation and suppuration these microbes play an essential rôle, i.e. are really the cause of the inflammation and suppuration, is extremely probable. It is a notorious fact that in many traumatic lesions of the skin no suppuration follows, while in others neither more nor less extensive, suppuration sets in. Thus, for instance, splinters of wood, glass, wire, &c., inserted under the skin are sometimes followed by a slight reaction, which passes off in a day or two, and is not followed by any suppuration, but on other occasions cause marked inflammation and suppuration.

A traumatic injury even of considerable extent produced under aseptic con-

ditions and treated aseptically, as is the case in Lister's and similar methods, as a rule heals by first intention, and does not lead to suppuration, whereas under other conditions suppuration follows as a rule. A blow by which the surface of the skin is not broken does not, as a rule, lead to suppuration, though in some few instances it is followed by suppuration. These facts seem easily explained on the assumption that in one set of cases the pyogenic staphylococci and streptococci do not happen to enter into the wound, e.g. in some cases of splinters, or are kept out of the wound, e.g. in aseptic treatment, and therefore no phlegmon and no suppuration follow, while in the other set of cases one or the other species finds entrance, and then by its multiplication causes suppuration. From this it must not, however, be concluded that every kind of phlegmon and suppuration is caused by the pyogenic microbes; for it is well known that such is not the case; a burn, or an injection under the skin of chemical irritants (e.g. oleum crotonis, turpentine, alcohol, ammonia, tincture of iodine, tuberculin, &c.) produces phlegmon and suppuration. Besides, various kinds of infectious phlegmons and suppurations are caused by other than pyogenic micrococci, as will be evident by-and-by (e.g. tubercle, glanders, actinomycosis).

A further consideration in connection with the pyogenic micrococci is this: like some saprophytic microbes, the pyogenic micrococci also—at any rate in some instances—produce noxious chemical substances; if large quantities of these are produced at the primary seat of inflammation, and are absorbed into the system, a condition similar to *sapræmia* is set up, manifesting itself by rigors, and fever temperature with or without intestinal disturbances; and it is precisely under such conditions of depressed vitality that an invasion of the whole body by the microbes, from the primary seat of inflammation and suppuration, may occur. As a result of such an invasion, secondary foci of inflammation follow in the lymph glands, internal viscera, bones and joints, with the formation of metastatic abscesses. Such secondary foci of suppuration running a sub-acute or chronic course represent *pyæmia*, or if the invasion is copious and universal an acute *septicæmia* is the result. In the latter case a general congestion of the viscera, and swelling of the spleen, will be found, and on microscopic examination of sections of the lungs, liver, kidneys, and the lymphatic glands, signs of diffuse or localised foci of inflammation—sometimes more, sometimes less abundant—will be apparent. It seems feasible to assume that in those cases in which no *pyæmia* and no *septicæmia* follow a primary inflammation and suppuration, no secondary successful invasion by pyogenic microbes has taken place; and, further, that if a secondary invasion does occur, it is due to the absorption of noxious chemical substances and a general depression of the vitality of the blood and tissues. It cannot be assumed that no absorption of the pyogenic microbes occurs, for there is nothing that would prevent those microbes from finding their way from any primary focus of inflammation or suppuration into the lymphatics and into the blood-vessels; but though this must be assumed to occur under all conditions, their settling down and multiplying does not take place under all conditions, since as a matter of fact not all primary inflammations, be they traumatic or be they associated with specific infectious diseases, are followed by *pyæmia* or *septicæmia*; on the contrary, it is rather the exception that such is the case. The reason for this absence of secondary foci of inflammation and abscess (*pyæmia*) or of a general and acute flooding of the system with the living pyogenic microbes must be sought in the normal power of the blood and tissues to destroy those microbes; but if this power be inhibited or exhausted, e.g. by the absorption of chemical septic substances and injury to the vitality of the tissues, or if

the number of the microbes absorbed be too large, then a secondary infection will be the result.

Pyæmia, i.e. disseminated phlegmons, and suppurations in distant localities (lymph glands, internal viscera, bones and joints), is therefore to be regarded as due to a secondary infection, i.e. an invasion by the pyogenic microbes, settling down and multiplying in various distant localities, and there causing inflammation and suppuration; and it is a sub-acute or chronic process as compared with certain forms of *septicæmia* caused by a copious and general flooding of the system with the pyogenic micrococci. This must be further distinguished from the septic intoxication caused by the absorption of septic chemical substances in sufficient quantities, produced at the seat of the primary inflammation, which in itself may be, and in all probability is, that which initiates and facilitates those *secondary infections*.

In caries of the teeth, various species of micrococci, bacilli, and vibrios occur; the former two have been found to form continuous masses filling the dental tubules of the affected parts and the neighbourhood, but whether they are the primary cause of the disease remains yet to be proved experimentally.

Miller has given in 'Mikroorganismen der Mundhöhle,' Leipzig, 1889, numerous careful observations on the microbes of the diseased teeth and jaw; also David in 'Les Microbes de la Bouche,' Paris, 1890.

Various necrotic processes in the viscera—necrotic foci in the kidneys, liver, cornea, and other organs—have been observed in man and animals (*see* Baumgarten, 'Lehrbuch der patholog. Mycologie,' I.) in which the necrotic foci were clearly due to the presence of streptococci; but all these must not be considered as belonging to a single species of the streptococcus pyogenes, as is done by Baumgarten and others.

A progressive necrosis, leading to death in a few days, has been observed in mice by Koch, the tissue being invaded, and the necrosis caused by a streptococcus.

In the Aleppo bubo, Riehl found numerous cocci in the round cells constituting the tumour, but they have not been cultivated.

ULCERATIVE ENDOCARDITIS

In the experiments by Wyssokowitsch with streptococcus pyogenes, mentioned above, the cultures were injected into the vascular system of rabbits whose aortic valves had been previously injured; the result was ulcerative endocarditis with copious multiplication of the microbe on the endocardium. In human ulcerative endocarditis, the blood-vessels of the heart muscle near the ulcerated endocardium are found plugged with crowds of micrococci; so also the tissue of the endocardium contains numerous continuous smaller or larger masses of cocci, and the ulcerated surface is almost covered with a continuous layer of them; the characteristic papilliform filamentous and fringe-like projections from the endocardium in the verrucous form of endocarditis are themselves sometimes covered with them, and contain numerous cocci in their tissue. Experiments by plate or tube cultivation yield pure cultures of these cocci; in some cases they belong to the species above described as staphylococcus aureus liquescens; in others they are the species described as staphylococcus albus; and in others again they belong to the species of streptococcus, probably streptococcus pyogenes.

Weichselbaum, Wyssokowitsch, Ziegler, Bonone, Hare, and Prudden found in some cases of ulcerative endocarditis the staphylococcus pyogenes aureus, but in others the staphylococcus albus or streptococcus pyogenes.

Netter found the bacillus pneumoniæ of Friedländer; and Weichselbaum describes cases of ulcerative endocarditis, some of which were associated with the diplococcus pneumoniæ, others with the streptococcus pyogenes, and others again with both. F. W. Andrewes made numerous observations and experiments on staphylococcus pyogenes aureus, in connection with ulcerative endocarditis. He found that the staphylococcus aureus taken from acute abscess (mastitis) when injected into the vascular system of rabbits is capable of producing foci of inflammation in the kidney, in the interior of the aorta, and in the myocardium of the ventricles of the heart. In some cases of human ulcerative endocarditis he found the staphylococcus aureus, in others the streptococcus pyogenes.

ERYSIPELAS

In erysipelas, the skin in its whole thickness is the seat of inflammation. In this process the skin appears swollen and red, the redness is more or less sharply outlined, serrated, and it and the swelling gradually spread into larger areas, but as the redness and swelling spread in the periphery, in the middle parts the skin becomes again livid. In microscopic sections the skin is immensely congested in the red parts, the blood-vessels greatly distended and filled with blood, the connective tissue is oedematous and contains leucocytes; these are chiefly present in the superficial layer of the corium, and from here extend into the stratum Malpighii; in the livid parts the number of leucocytes is greatly increased, and they pervade all parts of the corium more or less uniformly. In the peripheral red portions, the lymphatics are greatly distended and plugged by micrococci (Lukomsky), and it is this condition which induces the congestion of the vessels and the exudation of the lymph and leucocytes. As the multiplication of the micrococci increases and spreads, the nearest lymphatics become filled and plugged with them, and this again causes congestion of the blood-vessels of this part, and exudation of lymph and leucocytes, and in this way the redness and swelling follow the multiplication of the micrococci in the lymphatics. In the parts first affected the congestion passes off first, but the lymphatics remain plugged with the cocci, and then many of them are also found outside, i.e. in the connective tissue of the skin (Ziegler); at the same time the number of leucocytes here greatly increases, and they are found to form a diffuse infiltration of the subcutaneous tissue, the corium, the papillary layer and of the stratum Malpighii. In the variety of erysipelas known as erysipelas bullosum, small cavities appear in the superficial layer of the stratum Malpighii; these cavities as in other inflammations of the skin (e.g. foot-and-mouth disease, vaccinia, variola) are due to the accumulation of lymph in the interstices (cement or interstitial substance) of the epithelium; a few leucocytes also find their way into these cavities. As the lymph increases in these cavities the epithelial cells surrounding them become degenerated and break down; the inter-epithelial spaces and lacunæ are smallest in the red part, they increase towards the oldest or livid part, and here they attain such size that they become more or less confluent, separated only by thinner filamentous septa, the remains of compressed streaks and trabeculæ of epithelial cells; all these cavities contain cocci, clear lymph, *débris* of epithelial cells, and leucocytes; the central or largest bullæ extend from the stratum corneum down to the corium, and when the former breaks, the highly infiltrated corium becomes exposed.

Fehleisen has shown¹ that the cocci in the lymphatics of the red marginal

¹ Fehleisen, *Die Ätiologie des Erysipels*, Berlin, 1883.

part, as also from the deep parts of the skin in the central portions, are the *streptococci of erysipelas*, but that besides these, *streptococcus pyogenes* can also be obtained from the superficial layers of the central parts and from those of the peripheral parts. The *streptococcus erysipelatos* can be easily cultivated from the fluid of the marginal red part in the genuine erysipelas, such as occurs sometimes as a special acute febrile disorder, involving the face, neck, and scalp. The writer has had two such cases where genuine erysipelas with high fever occurred in adults; it commenced in the face and spread from here over the upper part of the neck, on the forehead and all over the scalp; the temperature was 102.5° on the evening of the third day, and the erysipelas was of the bullous kind, viz. as the redness passed into the periphery the older livid part became bullous, then the surface dried up into brown scales, which gradually broke and desquamated. From the marginal red part, after well cleaning it with perchloride of mercury solution, by a prick, lymph was received into a capillary glass pipette, and with this plate cultivations were made. Numerous colonies came up, which all proved to be the *streptococcus erysipelatos*.

With the cultures of the true *streptococcus erysipelatos*, Fehleisen has produced distinct erysipelas by inoculation into the skin of the ear of rabbits; after twenty-four hours the skin of the ear shows a deep blush around the place of inoculation, and is slightly swollen; this blush and swelling increase and spread during five or six days over the whole of the ear, on to the scalp and into the neck; the bullous and scaly change is also very marked. The writer has repeated these experiments, and can fully confirm them, as also the other fact stated by Fehleisen, viz. that from the spreading and swollen part of the skin of the ear there is no difficulty in obtaining pure cultures of the *streptococcus erysipelatos*. The cultures and microscopic specimens of this *streptococcus* are difficult to distinguish from the *streptococcus pyogenes*, but inoculation into the rabbit's ear shows this difference. Injection of large quantities into the vein produces in the rabbit in a large percentage of cases acute septicæmia, fatal in thirty to forty-eight hours; the spleen is enlarged, all viscera are congested, and from the spleen, lung, liver, and from the blood of the general circulation the *streptococcus of erysipelas* can be easily recovered by cultivation.

When the *streptococcus erysipelatos* is carried through several generations on gelatine, its virulence suffers a distinct diminution, since the effect on the rabbit's ear becomes greatly diminished. From a twelfth sub-culture, inoculation into the rabbit's ear produces after twenty-four hours a distinct blush; this spreads a little during the next day or two, and the skin is tumid, but after this the redness and swelling again subside.

Whether or not the *streptococcus* isolated from human erysipelas by Passet, Bonone and others, and assumed to be the *streptococcus pyogenes*, was the real *streptococcus erysipelatos*, but representing merely an attenuated form, is a matter of conjecture; the facts brought forward by Fehleisen and others, viz. that two species of *streptococci* can be originally obtained by culture from the erysipelatos skin, and their differences proved by inoculation into the rabbit's ear, seem definitely to contradict the above conjecture. The above observers, in fact, maintain that the *streptococcus pyogenes* of abscess and ordinary phlegmon is identical with the *streptococcus* of Fehleisen, that is, of erysipelas, the latter representing merely an attenuated form of the former. E. Fränkel ('*Centralbl. f. Bact. und Parasit.*' VI., p. 691) is quite sure of the identity of the two microbes; but notwithstanding this, on pathological and clinical grounds, a doubt of this alleged identity may yet be permitted.

An important series of experiments made by Fehleisen was this, that he

produced with cultures of the streptococcus erysipelas distinct and typical erysipelas in the human subject; these experiments were made in order to cause sarcomatous tumours to disappear, in conformity with older surgical experiences; and in this he succeeded in several instances, in which he produced by his cultures true erysipelas of the skin in the part containing a tumour; the erysipelas passed off, and the tumour also disappeared, and became absorbed.

This question of the alleged identity maintained by some observers of the streptococcus pyogenes and streptococcus of erysipelas illustrates better than any other case, on the one hand, the difficulty existing in distinguishing two similar microbes, and on the other the want of appreciation of pathological and clinical facts in drawing conclusions from bacteriological investigations alone. No clinical observer will admit that erysipelas is an ordinary phlegmon, though all bacteriologists may not be able to distinguish the one species from the other. Of course the suggestion is not wanting that owing to an increase in virulence, due to some unknown cause, the streptococcus pyogenes, growing in and causing ordinary simple phlegmon, may become the streptococcus of erysipelas and cause erysipelas, while in a similar manner this latter organism by attenuation, due to some cause or other, may become attenuated and may degenerate thereby into the streptococcus pyogenes. While such an assumption is based on very insufficient evidence, the other alternative, viz. that the two are physiologically different species, though they may not be easily distinguished from one another, is based on the fact that the two species have an altogether different distribution, the one, streptococcus pyogenes, in pus of acute and chronic abscess and in common phlegmon, the other in erysipelas—a type of a specific disease as distinct as any, both pathologically and clinically. It is just as difficult to imagine that an ordinary non-infectious sore throat accompanying an ordinary cold is an attenuated form of diphtheria as that an ordinary phlegmon is an attenuated form of erysipelas; no clinically experienced observer would consider a sore throat associated with an ordinary cold as diphtheritic sore throat, any more than he would admit a common phlegmon, or common abscess, to be erysipelas. Such an assumption of the identity of causes in maladies so utterly different as common phlegmon and erysipelas seems opposed to the known fundamental facts of the specific diseases.

CHAPTER XIII

SEPTICÆMIA

WE have on previous pages pointed out that a distinction must be drawn between septic intoxication, sepsis, or sapræmia, on the one hand, and septic infection, or septicæmia, on the other. Under septicæmia is understood an acute febrile disturbance, due to the presence and multiplication of microbes within the blood,¹ generally ending fatally, between twenty hours (or even less) and two or three days, rarely later. All vessels contain the microbes, the organs of the chest and abdomen are congested,² extravasation of blood

¹ The first systematic experimental work on the relation of micro-organisms to septicæmia was published by Koch in *Die Aetiologie der Wund-Infektionskrankheiten*, Leipzig, 1878.

² The pathology of sapræmia is very much the same as that of septicæmia, since both are probably the result of the action of similar kinds of toxic chemical substances, in the former produced at a given primary focus, in the latter produced in the circulating blood by the microbes distributed in the body.

occurs in the serous membranes, also in the lung, spleen, and intestines; the spleen as a rule is enlarged, its vessels are engorged; the intestinal mucous membrane is in a state of congestion and inflammation. In many small vessels, e.g. in the liver, kidney, spleen, and lung, the number of the specific microbes has so largely increased that veritable thrombi are formed by them; in such places the tissue around the vessels is found in a state of necrosis—coagulation necrosis. This condition is often found in the liver, when the central vein of a lobule and a number of capillaries leading into it are distended and plugged with solid masses of the microbes; the liver cells of such a part are large and opaque; they do not stain in dyes, their nucleus is swollen and clear—such parts correspond to necrotic tissue; in the kidney many of the capillaries of the glomeruli are also plugged with masses of the microbes, also some of the fine vessels in the cortex between the convoluted tubes and in the medulla between the straight tubes; the rest of the glomerulus is swollen and degenerated; in the convoluted tubes and in the straight tubes of the cortex, the epithelial lining of the tubes is swollen, dark, granular, and in a state of disintegration; in the lungs many lobules show great congestion of the capillaries surrounding the alveoli; in these the epithelial cells are desquamating, and blood is occasionally found extravasated into their cavity; pericardial, pleural, and peritoneal exudations occur: the latter is very common.

The specific microbes can be easily demonstrated by cover-glass specimens; and by culture from the heart's blood, and from all congested viscera, particularly the spleen, though there obtain great differences in these respects, since in some the number of microbes in the blood of the general circulation is very limited, while in others it is abundant. From the spleen they can always be recovered in considerable numbers.

We have on a former page pointed out that where, in any locality, extensive inflammation, ulceration, or suppuration is going on, the absorption of chemical poisonous substances, elaborated by microbes at those localities in sufficient quantities, produces *sapremia*, which may then induce, or rather pave the way for, a secondary invasion of the microbes from that primary focus, whereby a true infection is produced, which may, and in some cases does, cause *septicemia*, the microbes multiplying in the blood abundantly. But a *sapremia* may occur without subsequent secondary infection, and the *sapremia* may be in some cases so extensive, owing to the large amount of matter absorbed, that it causes death; the pathological symptoms are the same as those of the *septicemia*, as naturally they would be, but there are none of the microbes of the primary focus to be found in the blood, spleen, or other viscera. We said just now that the pathological appearances would be naturally the same as in the *septicemic* infection: *sapremia* being caused by the absorption of toxic chemical substances produced by the microbes at the primary focus, while *septicemia* following such a primary disease is due in the first instance to a general invasion of the blood and tissues by the microbes and subsequently to the action of toxic substances produced by the microbes in the blood and tissues.

While giving these general outlines of what is usually considered as *septicemia*, we cannot shut our eyes to the fact that the term *septicemia* is applied to a group of diseases in no marked manner or degree well defined from other diseases not strictly coming under this head. At present various diseases of the true type of infectious and contagious diseases fall under this group, e.g. fowl cholera, malignant anthrax, swine erysipelas, swine fever, and human erysipelas. Now all these diseases are as true and typical infectious diseases as small-pox or scarlet fever. There is nothing

fundamental by which septicæmic diseases, anthrax, small-pox, or scarlet fever can be said to be separated from one another. True in the septicæmic diseases, e.g. surgical septicæmia, *post-mortem* wounds septicæmia, various septicæmic diseases in rodents and other animals, infection starts, as a rule, from a wound, i.e. by inoculation; but there are others in which infection is most certainly produced naturally by way of the respiratory and alimentary canal, e.g. in genuine erysipelas of the face, where in the affected individual infection by inoculation is out of the question, or in anthrax of sheep and cattle, where infection is carried out naturally by the alimentary or respiratory tract, or in the form of anthrax in the human subject known as woolsorters' disease, or in intestinal anthrax; again in a septicæmic disease to be described as ragsorters' disease, infection is also effected by the respiratory tract. In fowl cholera, in swine erysipelas, and in swine fever, infection under natural conditions is carried out by the alimentary and respiratory tract; and in these respects there is therefore no difference to be drawn between these diseases and, say, the acute exanthemata. Nor can we establish a difference between them and the acute exanthemata on account of the latter showing some well-marked cutaneous eruption, for in swine erysipelas, in human erysipelas, in malignant pustule, there are just as characteristic cutaneous lesions present. Further, we cannot say that as regards the distribution of the microbes there is a fundamental difference. True, in many septicæmic diseases the microbes are universally present in the blood, but there are marked differences in this respect between the different forms; for while in some the microbes in the blood are very numerous, e.g. anthrax, and certain forms of septicæmia to be mentioned presently (Davaine's and Koch's septicæmia, fowl cholera), they are scarce in the blood in others, e.g. malignant œdema, swine fever, and swine erysipelas: they are not easily found in the blood in human erysipelas. Nor do we find any fundamental difference as regards progress and duration. True, many septicæmic diseases are rapid (duration from sixteen to twenty hours, to two or three days), but there are some belonging to this class which pursue a longer course, e.g. swine fever. We must therefore consider that the term septicæmia is an artificial one, and that the group of diseases indicated by it is not fundamentally different from other acute infectious diseases not generally included in this group. When septicæmic infection occurs after direct inoculation—as in accidental, surgical, puerperal, *post-mortem* infection, or after experimental inoculation of cultures of the septicæmic microbes into animals—the multiplication of the microbes within the blood produces those toxic substances which, when in sufficient quantity, set up the symptoms of the disease and ultimately cause death.

We will now proceed to describe the various specific microbes which have been proved to cause septicæmia in man or in animals, omitting those which have been already mentioned as capable of producing under certain conditions septicæmic infection in rodents, e.g. the pyogenic staphylococci and streptococci, as also certain microbes occurring in saliva (Pasteur and Sternberg) and in normal human faecal matter (Bienstock, Brieger), the typhoid fever bacillus, and the micrococcus tetragonus (on mice and guineapigs) occurring in purulent human tubercular sputum, and the cholera spirilla of Koch and those of Finkler (on guineapigs).

A. Davaine Septicæmia in Rabbits.—This is a septicæmia which Davaine first produced by injecting putrid ox's blood into rabbits. It is now known that a small bacillus (formerly considered as bacterium) is the microbe, which by its great multiplication and universal distribution in the circulating

blood causes the disease and death. The microbe is present in the blood in great numbers, nearly as great as those of the blood-corpuscles; in stained specimens the rods, which are short and oval, show a stained granule at each end with a clear space in the middle; the length of the rods is about $1.5\ \mu$, the thickness about half of the length. The rods are non-mobile, and from the heart's blood, and all other tissues, pure cultures can easily be made. In plate cultures, after about two days, minute white dots are visible; under the microscope they appear as flat circular discs, white in reflected, yellow brown in transmitted light. After several days the colonies are larger, and appear thicker and broader in the centre than at the periphery, which appears more or less concentric owing to regular differences in thickness. At maximum growth the colony does not exceed one to two millimetres. In stab culture the stab is occupied by a whitish line; under the microscope this is seen to be made up of minute droplets and dots, whitish in reflected, yellow brown in transmitted light. In streak cultures the streak is represented by a narrow whitish band of irregular outline, thicker in the middle than at the margin. After two to three months the cultures in gelatine are dead.

Rabbits, mice, fowls, pigeons, and sparrows are very susceptible (Koch) to the inoculation of minute doses of the culture or of the blood of an animal previously dead of the disease; guineapigs and rats are insusceptible (Koch). When rabbits are inoculated with a trace of the blood of a rabbit dead of the disease, or with a trace of the culture, after ten to twenty-one hours they show a distinct rise of temperature; in some cases the animals show spasms and a fall of temperature before the end of the first sixteen hours, and are dead before the day is over; but in some cases, particularly after inoculation with minute traces of culture, death does not take place in less than thirty-six to forty-eight hours. The bacilli are found very numerous in the blood-vessels of all organs. The spleen, liver, lymph glands, and lungs are highly congested, as also the intestines; extravasations are rarely found, and then only in the omentum and lungs; peritonitis is noticed only in a small percentage of cases, and then only when the omentum shows the extravasations; the serous covering of the intestines is greatly injected. As a rule, these symptoms are more pronounced if death does not occur before the second day.

This septicæmia has been minutely studied by Koch, who investigated the susceptibility of various animals towards these bacilli.

B. Septicæmia in Rabbits (Koch).—Oval cocci $0.8\ \mu$ to $1.0\ \mu$ in length, about $0.6\ \mu$. in breadth. Putrid meat-infusion injected by Koch into rabbits produced death by septicæmia; the spleen was found much enlarged and all internal viscera congested. The cocci are numerous present, generally isolated, occasionally in clumps, in the capillaries of all viscera. The cultural characters of this organism have not been studied by Koch. The writer has produced a septicæmia in rabbits by subcutaneously inoculating them with sputum of human croupous pneumonia, which septicæmia bears a great resemblance to the above; the symptoms were the same, the cocci were the same, and their distribution in the organs, particularly in the kidneys, appeared the same as described by Koch. From the blood of the rabbits dead from the disease, cultures were easily obtained; the colonies were whitish, round, and convex; at the end of a week they grew to 4–5 mm. in breadth, and in transmitted light appeared brownish; in streak cultures they formed a whitish, irregularly outlined, moist-looking band, which appeared either of uniform thickness or a little thicker at the margin. Stab cultures several days old appeared in transmitted light to be made up of brownish droplets; during the first few days the stab when viewed with the unaided eye appeared as a whitish line. Mice and rabbits were found susceptible

to the cultures, but comparatively large doses ($\frac{1}{4}$ – $\frac{1}{2}$ c.c. of broth-culture) had to be injected.

C. Malignant Œdema (Koch).—This disease has been produced by Koch in guineapigs by the subcutaneous injection of recently manured garden earth. An extensive cedema occurs at and about the seat of inoculation; the cedema is accompanied by hæmorrhage into the subcutaneous tissue, and is of an offensive odour; it spreads during the second day, leads to gangrene of the subcutaneous and muscular tissues with the formation of gas bubbles, and the animals die in from twenty-four to forty-eight hours: the spleen is found congested, so also are the liver, kidney, lungs, and intestines. In the cedematous exudation and in the spleen long mobile bacilli are present, either singly or in filaments and long chains; their number in the blood is comparatively small; the size of the short bacilli is $2\text{--}3.5\ \mu$ in length, and $1\ \mu$ in thickness; their ends are more or less rounded. Many bacilli are in the form of chains and filaments. The cedematous fluid, and the blood, inoculated into fresh guineapigs produce the fatal disease.

Rabbits are also very susceptible to the disease; and at the seat of inoculation cedema is produced. Mice are very susceptible, and die before the end of the first day, but no cedema is present at the seat of inoculation. All the viscera are congested, and the spleen is enlarged; the blood of the spleen, the exudation of the peritoneum, and the pleura, contain the bacilli. A sure diagnosis, and differentiation from anthrax bacilli, to which the cedema bacilli bear a certain likeness, can with certainty be made by cultures.

The cultural characters of this bacillus show that it is altogether different from that of bacillus anthracis; although in size and general aspect in the fresh state, and in stained cover-glass specimens, it is not unlike bacillus anthracis, yet it is quite different in its action on animals, in the condition of the spleen of the inoculated animals, and in its small numbers in the blood of these. When cultivated it shows the following characters: The cedema bacillus is *anaërobic*, since it does not show growth on the surface of nutritive media; it grows only when planted in the depth; in gelatine (in the depth) it forms characteristic globular colonies of different sizes, opaque and liquefied, their margin more opaque than the centre and finely striated. The growth and liquefaction proceed gradually and slowly, till all the gelatine is liquefied; at the bottom of this is a voluminous greyish white filamentous mass. It grows best in gelatine to which 1–2 per cent. of grape sugar has been added. In solid agar agar it grows well, producing uniform turbidity all through the medium, with floccular condensations and numerous gas bubbles. Solidified blood serum is liquefied by the bacillus. The cultures act virulently, provided comparatively large quantities are injected.

Oval bright spores are formed very rapidly in the short bacilli, either in the middle or at one end; the spores are thicker than the bacilli themselves; and the bacilli in the cedematous fluid contain spores. The cedema bacillus is of great importance, since by the observations and experiments of Chauveau and Cornevin, Brieger, and others, it has been shown that surgical gangrene (progressive gangrenous emphysema) in the human subject is caused by the same bacillus. It seems that many a soil containing putrid animal substances, such as hay dust, rag dust, offensively smelling filth of dust bins, offensively smelling exudations, gangrenous discharges, &c., contain the cedema bacillus and its spores. Horses, pigs, and sheep are susceptible to this malignant cedema, provided large doses are inoculated; cattle are not susceptible. As mentioned above, guineapigs are the best experimental animals, since inoculation produces a typical, emphysematous, spreading cedema, with fatal result.

Pasteur has studied this septicæmia in guineapigs; hence it is also called Pasteur's septicæmia. The bacillus was called by him vibrio septique. Roux and Chamberland have demonstrated, in the broth cultures of this microbe, toxic substances, which, separated from the bacilli and injected into animals, cause a transitory illness proportionate to the amount injected, and thereby confer immunity against the injection of the virulent bacilli themselves. But this immunity does not last long, and is not produced if the quantities used are too small.

With recently manured garden earth, the writer has produced in guinea-pigs, rabbits, and mice a disease which, in its symptoms, is in many respects similar to the malignant cedema of Koch. But the microbe is totally distinct; it is a short, mobile rod, the long cylindrical and filamentous forms being few. It is distinctly *aërobic*, forms also gas bubbles in the depth of the gelatine, but grows well in ordinary nutrient gelatine; does not liquefy the gelatine, and does not form spores. The cedema fluid at the seat of inoculation is crowded with the bacilli, the blood and spleen containing only comparatively few bacilli (fig. 41).

Flügge has isolated a pseudo-malignant cedema bacillus from recently manured garden earth, which resembles Koch's malignant cedema bacillus, but is non-pathogenic.

D. Mouse Septicæmia of Koch.—By inoculation of filthy water into mice, Koch produced an acute and fatal septicæmia, which, owing to the peculiarity of the microbe, is of great interest. At the seat of the inoculation there is found slight hæmorrhage, the internal viscera are greatly congested, and the spleen is not much enlarged; the animals die during the second day. In the blood of all parts are found in very large numbers exceedingly minute bacilli, some longer than others; but all are very fine, and many of the white blood-corpuscles are quite filled with them, and at the same time swollen up. In the lungs there is slight hæmorrhage into the alveolar tissue; everywhere one sees the swollen leucocytes completely filled with the minute bacilli. Some of these become free owing to the disintegration of the leucocytes. Sections through the lung, stained carefully in fuchsin and then in methyl blue, show the nuclei of the tissue and of the leucocytes blue, the bacilli bright red.

Cultivations of the heart's blood, or of the juice of the viscera, yield numerous colonies; pure cultivations in gelatine in test tubes can be made without difficulty directly from the heart's blood. The colonies in plate cultivations appear after two or three days as minute highly translucent, gelatinous, grey, irregularly outlined, angular patches; in the stab culture in gelatine, after two or three days, a very characteristic growth is noticed; the stab is a translucent grey line from which branch out horizontally vast numbers of fine, closely placed, gelatinous, translucent threads; in the streak culture the streak becomes visible after two to three days as a gelatinous, grey, translucent, thin band from which pass out numerous fine grey lines. The growth liquefies the gelatine very slowly; it takes generally some days before liquefaction commences, and it proceeds very slowly, the liquefied gelatine being thick like syrup, fairly limpid, but containing greyish translucent flakes. In agar mixture the growth is slow and very transparent.

Specimens made of the cultures show under the microscope, besides short bacilli, also a great many long threads more or less curved. Inoculation into mice produces the septicæmia with certainty.

This microbe is in so far interesting, as one extremely similar to it, both as to size, aspect, and its peculiar cultural characters, has been isolated by

Schütz from the spleen of pigs dead of swine erysipelas (*see* below). By its action on swine and on rodents this microbe was proved to be the real cause of the swine erysipelas, and it was also shown that its action on mice is extremely similar to that of the microbe of Koch's mouse septicæmia, just described.

E. *Human Septicæmia*.—Several microbes (micrococci, Sternberg) have been described in connection with human (traumatic) septicæmia; the staphylococcus albus liquescens, staphylococcus aureus liquescens, and the streptococcus pyogenes being mentioned amongst them. But in connection with this it must be remarked that the occurrence of these organisms in the blood-vessels of the tissues is possibly only of the nature of a secondary invasion, and may not have been the primary cause of the septicæmia, such as occurs, for instance, in hospital gangrene or in septicæmia after a *post-mortem* wound. The writer has in a case of acute septicæmia, after amputation of the mamma, seen in microscopic sections through the congested lung, liver, kidney, and mesenteric lymph glands, in the capillary blood-vessels, and extending into the surrounding more or less necrosed tissue, particularly of the liver, of the glomeruli of the kidney, and of the alveolar walls of the lung, numerous short bacilli, which formed in some places veritable emboli; their distribution clearly indicating that they stood in an intimate relation to the diseased process; but no cultures having been made, nothing further can be said of them. Chauveau and Arloing described the occurrence of bacilli in cases of gangrene, but here no culture experiments were made, although experiments on animals made it probable that these were the œdema bacilli. (On the relation of the bacillus of malignant œdema to surgical gangrene *see* a former page.) Human septicæmia is therefore a subject which at present is still awaiting solution, as far as the causing microbes is concerned; though from what has been said in a former chapter it is quite probable that some forms of human septicæmia may be due to a general invasion by the above-named micrococci. That some forms of septicæmia, not of surgical antecedent, are caused by specific bacilli is illustrated by the acute forms of disease known as ragsorters' disease, which is caused by the bacillus proteus hominis.

Ragsorters' Disease.—In a very important research carried out by Bordoni Uffreduzzi ('Zeitsch. f. Hygiene,' III., 2, p. 888) it was shown that a bacillus distantly related to the *proteus*¹ of Hauser (figs. 10, 11, 12), and called

¹ Hauser's proteus is a motile bacillus occurring in putrid meat, in gangrenous wounds, and in many putrid animal substances. Two species were isolated: (a) *proteus vulgaris*, and (b) *proteus mirabilis*.

(a) *Proteus vulgaris*. Motile rods 0.6 μ thick, 1.25–3.75 in length, some short like cocci, others much longer like leptothrix threads; the threads vary in length, and are aggregated in bundles and twisted as in a cable; it grows rapidly in gelatine plates (gelatine 6 per cent.). In from six to eighteen hours the colonies are noticeable as whitish dots situated in a pit, because surrounded by liquefied gelatine. Thence rapidly extend threads and processes (swarmers), by which the growth gradually spreads over the rest of the gelatine. (Figs. 10, 11, 12.)

(b) *Proteus mirabilis*. In size, and in its polymorphous nature, the same as *proteus vulgaris*. The rods show slower movement, the gelatine is liquefied slower than by *proteus vulgaris*. The *proteus mirabilis* is also distinguished from the former by numerous 'involution forms,' i.e. longer or shorter threads, swollen at one end, having either at or near one end a spherical or flask-shaped knob.

The name 'proteus' has been chosen by Hauser on account of the polymorphous nature of the microbes; some being coccus-like, others short oval rods, others again longer cylinders and threads; even vibrio-like and spirilla-like forms occur.

A third species of proteus is known as *proteus Zenkeri*: this resembles in many respects the *proteus mirabilis*, but does not liquefy the gelatine.

by Bordoni Uffreduzzi *proteus hominis capsulatus*, on account of the bacilli being surrounded by a gelatinous capsule, produces a septicæmic (acute) fatal disease in man. The disease resembles to a certain degree anthrax in man (wool-sorters' disease): the lungs, trachea, bronchial glands, and mesenteric glands are congested and inflamed, the spleen is enlarged, the serous membranes and lymph glands show hæmorrhages. Cover-glass specimens made of the blood of the general circulation, the lungs, spleen, and kidney show bacilli not unlike anthrax bacilli, though there are certain well-marked differences between the two: the proteus bacillus having rounded ends, and on the whole being smaller. Sections through all organs, particularly the mesenteric and peribronchial lymph glands, show the bacilli in great numbers. The cultural characters of the proteus hominis at once show a striking difference between it and the bacillus anthracis.

The proteus hominis does not liquefy gelatine, and grows equally well in acid, neutral, and alkaline gelatine. Further, the proteus grows very much more rapidly than the bacillus anthracis. After eighteen to twenty-four hours it forms round dots, which after forty-eight hours have much enlarged, and under the microscope appear granular, and threads can even be distinguished in them. In the streak culture it forms a white glistening waxy band; in the stab culture it grows similar to Friedländer's bacillus pneumoniae, i.e. like a nail composed of the nailpin and nailhead; in the recent cultures the bacilli form threads; later on many very short forms are noticed. In agar cultures this proteus hominis is always well capsulated.

Experiments were made on animals with the cultures; the result of these shows a distinct difference between the proteus bacillus and the bacillus anthracis. Mice and dogs are very susceptible, rabbits and guinea-pigs are less so. In the dog the injection of culture into the jugular vein produces an acute fatal disease, very similar to that which Bordoni Uffreduzzi observed in the human subject; also the distribution of the bacilli in the organs is the same. This disease of human beings was observed to occur in persons occupied in the sorting of rags, the same disease had previously been considered as anthrax, owing to a certain similarity to wool-sorters' disease and its microbes; but there can be no doubt that there are two kinds of rag-sorters' disease: (a) real anthrax¹ due to a general and fatal infection with the bacillus anthracis, as in wool-sorters' or hidesorters' disease; and (b) a septicæmic infection, due to the proteus hominis.

Kolb ('Arbeiten aus dem Kais. Gesundheitsamte,' VII., p. 60) describes the occurrence in the organs, but not in the blood, of persons dead from *purpura hæmorrhagica*, of thick oval rods, non-motile, chiefly occurring as diplobacteria; in gelatine their growth resembles that of the typhoid bacillus. Dogs, mice, and rabbits inoculated with cultures die in from one to three days, showing hæmorrhages in the various organs, the bacilli being abundantly present in the blood and viscera. These bacilli do not form spores.

Puerperal Septicæmia.—In a number of cases of puerperal septicæmia investigated by Dr. W. R. Smith under the writer's direction, the presence of streptococci was shown in the circulating blood. From a droplet of the heart's blood of the dead body, and from a droplet of the blood of the finger of the living, we obtained without difficulty in pure cultures large numbers of colonies of this streptococcus. Its cultural characters are in many respects similar to those of other streptococci described on previous pages. When tested by inoculation into the skin of the ear of the rabbit a distinct blush and swelling are noticed after twenty-four hours; these increase after

¹ Paltant (*Wiener kl. Woch.*, 1888, Nos. 18-26) describes such cases of rag-sorters' disease which are anthrax.

another day or two, and then pass off, without leading to erysipelas or to suppuration; minute traces of the cultures injected into mice produce acute fatal septicæmia. The animals die in from thirty to forty-eight hours; in the blood of the heart and in that of the other congested organs—lungs, liver, kidney, and spleen—the streptococci can be demonstrated easily by cover-glass specimens and by culture. The cultures retain their virulence on rabbits and mice through many sub-cultures, even when injected in minute doses, and in this respect it materially differs both from the streptococcus of erysipelas and the streptococcus pyogenes.

The writer obtained a pathogenic bacillus causing acute *septicæmia in guineapigs and mice* from the pleural exudation of mice and guineapigs that had died spontaneously from septicæmia, that is to say, in which no primary cause could be assigned, and in which the *post-mortem* appearances showed the symptoms of septicæmia—viz. great congestion of the lungs, liver, and kidney, inflamed peritoneum, pleural and pericardial exudation, the spleen dark and slightly enlarged in the mice, the intestines relaxed, congested in the mucous and serous coats, the cavity of the small intestine filled with sanguineous mucus. Inoculation of guineapigs or mice with the gelatine cultures proved fatal in the mice within one, two, or three days; in the guineapigs larger doses had to be used to produce death in a day or two. When small doses are used there is noticed already in twenty-four hours, about the seat of inoculation, a firm thickening which gradually extends into wider areas; and death ensues after several days to a week. In all cases the bacilli can be easily demonstrated in the heart's blood and in the congested organs by cover-glass specimens and by culture. In sections through the liver and kidney the bacilli are found in masses occluding like emboli the capillary blood-vessels; in the liver the central vein of a lobule and numerous capillaries leading into it are found filled with and distended by continuous masses of the bacilli, the surrounding liver tissue being in a necrotic state; in the kidney numerous capillaries between the convoluted tubes of the cortex and in the glomeruli are found occluded by the bacilli. The bacilli taken from the blood are rounded at their ends, and motile; in cultures, notably in broth or other fluids, some of the bacilli are short like cocci, others are oval, others again cylindrical; there are also numerous longer and shorter chains, which show active motility; in these chains the joints or elements are of all shapes—cylindrical, oval, or coccus-like. That all these forms belong to the same species can be easily proved by plate cultivation; for in these all colonies are of exactly the same kind.

In gelatine plate cultures the microbe forms white round flat dots, which make their appearance in the course of a day or two. These colonies gradually enlarge, their outline becoming irregular; in stab cultivation they form a white line; on the surface of the stab is a flat white plate, with a very irregular crenated outline, extending rapidly over the surface of the gelatine; in streak cultivation a white band appears with knobbed or uneven outline. The gelatine is not liquefied. No spore formation can be demonstrated, since the microbes of recent or old cultures, when dried in a thin film, or when heated to 65–70° C. for five minutes, are killed; but the culture tubes preserved at the temperature of the room remain living for many months.

In broth at 37° C. they grow very rapidly, and form a uniform turbidity during the first day; during the second and third days a copious whitish sediment appears, and the fluid contains numerous flakes and granules.

Wooldridge has shown that when these bacilli are grown in alkaline albumen solution (Brazil nuts) they form chemical substances which, when separated from the bacilli and injected into the animal, give immunity to this animal against the virulent bacilli, since a subsequent inoculation of even large doses of the active bacilli causes no disease. This is then a parallel instance to that of Roux and Chamberland, in which protection against the virulent bacilli is conferred by a previous injection of the chemical products of the bacilli.

Fowl Cholera.—This disease causes great devastation amongst poultry. The malady, well known by the researches of Perroncito, Toussaint, Pasteur, Kitt, and others, affects fowls, pigeons, and rabbits. In the fowl after an incubative period, varying between sixteen or eighteen hours to twenty-four hours, the disease declares itself by diarrhœa of fluid, greenish evacuations, great drowsiness and sleepiness of the animal. In about twenty to forty-eight hours the animals are found dead; the blood in the heart and general circulation, and in the vessels of all organs, the intestinal contents, and the evacuations teem with short, oval, non-motile bacilli, measuring 0.5–1.2 μ in length, 0.2 μ in thickness. In stained

preparations they show at each end a stained granule, while the middle part is clear and unstained. On *post-mortem* examination the viscera are found greatly congested and containing hæmorrhages; the mucous membrane of the upper part of the intestine is found congested; often small hæmorrhages occur in its mucous membrane; the contents are fluid fæces; the spleen is enlarged. Fowls, rabbits, and pigeons inoculated with a droplet of the blood of a fowl dead of the disease, or inoculated with the artificial culture of the bacilli, die of the disease in from twenty to forty-eight hours, the blood teeming with the bacilli. By feeding other fowls with the intestinal contents of diseased fowls the disease can be reproduced in them. From this the conclusion is justified that also under natural conditions infection is caused by the healthy fowls picking up the contagium with the food from soil tainted with the evacuations of diseased animals (fig. 53).

The cultures of the bacilli show the following characters: In plate cultivations the colonies appear before forty-eight hours as minute yellowish white dots, irregularly outlined or round; seen under the microscope they are faintly granular discs; the centre is yellow and transparent, then follows a brown zone, and then a transparent marginal part. In stab culture the line of inoculation becomes marked as a white line made up of more or less confluent yellowish white droplets; on the surface of the stab is a small irregularly outlined plate; in streak cultures the growth appears after two or three days as a yellowish white band with irregular or knobbed outline, thin in the centre and margin, thicker and brownish in the intermediate parts; on potato the microbe grows only at higher temperatures, 28°–38° C. It grows slowly, and forms a waxy, greyish white film.

By inoculation of minute quantities, a drop of broth culture, into the subcutaneous tissue, or by feeding fowls, rabbits, mice, or pigeons with culture, the disease is easily reproduced. In guinea-pigs and sheep it produces a local abscess at the seat of inoculation.

By keeping broth cultures for some months Pasteur has succeeded in producing by inoculation of fowls a local cedematous inflammation; the animals became only slightly affected, but recovered and showed themselves refractory against a second inoculation. Pasteur thought that the influence of the oxygen of the air produced the attenuation; it is now proved, however, that this is not so (Kitt), but that Pasteur had impurities (accidental microbes) in his broth cultures, which at first attenuated the bacilli of fowl cholera, and as time went on altogether suppressed them; hence the broth cultures of Pasteur after the lapse of some months proved barren of all pathogenic action.

Pasteur has shown that by injection of large quantities of broth cultures, from which the bacilli of fowl cholera have been previously removed by filtration, a transitory illness can be produced, and that the animals show themselves afterwards refractory against inoculation with virulent material. Marchiafava and Celli showed that the microbe passes from the mother to the fœtus, probably owing to ruptures (hæmorrhages) in the vessels of the maternal placenta.

Eberth and Schimmelbusch ('*Fortschritte d. Medicin*,' Bd. VI., No. 8, p. 295) described an acute infectious disease in *Mustela furo*—*Frettohenseuche*—showing itself chiefly as pneumonia with enlarged spleen; in the heart's blood, in the inflamed lung, the liver, and the enlarged spleen there are present numerous motile bacilli, similar in many respects to the bacillus of fowl cholera, swine fever, and Wildseuche. The cultures act very virulently on sparrows, less virulently on pigeons; fowls are refractory; in rabbits the inoculation produces only a local inflammation of a temporary character, and the same results, only milder, are produced in guinea-pigs.

Duck Cholera.—As such, Cornil describes a fatal infectious disease affecting ducks, which in its symptoms and causation is similar to fowl cholera; but there is this difference between them, that the disease of the duck is not transmissible to the fowl. The bacilli are, however, similar in many respects to those of fowl cholera.

On account of the morphological and cultural similarities presented by a series of pathogenic microbes—bacillus of fowl cholera, bacillus of septicæmia of rabbits, derived from human saliva (Pasteur, Sternberg), bacillus of Davaine septicæmia, bacillus of *Frettohenseuche*, bacillus of swine fever, bacillus of Wildseuche and others—Hueppe and also Baumgarten consider them to be identical, and they call the disease produced by them septicæmia hæmorrhagica. But this view cannot be accepted, since the experimental results produced by these different microbes in different animals are utterly different one from another; besides, this assertion is based on insufficient observation even of the cultural characters of these different microbes. Owing, however, to their being similar in size, aspect, and general character in culture, they certainly belong to the same family, but each represents a well-defined species.

Fowl Enteritis.—This is an acute fatal infectious disease affecting fowls, but not pigeons and rabbits, and by this alone its differentiation from fowl cholera is established. Besides the microbe and its distribution, the course and symptoms of the disease are

quite distinct from fowl cholera. The writer has met with the fowl enteritis on a poultry farm in England, where it caused great mortality. After an incubation of three to four days, the affected fowls show diarrhoea of fluid greenish evacuations, are quiet, but never show sleepiness or drowsiness. A day or two after the diarrhoea has set in they are found dead. The mucous membrane of the intestine is found congested, but without hæmorrhage, the internal surface of the mucous membrane coated with grey or yellowish mucus, which under the microscope contains numerous leucocytes and detached epithelial cells; the liver is congested and brittle, the spleen much enlarged, the lungs normal. In the heart's blood are present relatively few bacilli, which are a little longer and thicker than those found in fowl cholera; the spleen contains the bacilli numerous, and also the vessels of the liver; the mucus of the intestine contains the bacilli in almost pure culture. In culture the microbe distantly resembles the bacillus of fowl cholera, except that its colonies are more plaque-like when growing on the surface; the microbe is non-mobile, does not form spores, and is killed by exposure to 60° C. for five minutes, as also by drying. Pigeons are insusceptible, rabbits only very slightly susceptible. By feeding of fowls with the contents of the intestine, the disease can be produced; by subcutaneous inoculation the disease can be produced, both with the blood or spleen tissue of a fowl dead of the disease, as also with artificial cultures of the microbe. In all cases the animals do not show any illness till the fourth day (this is also an important distinction from fowl cholera), or more generally till the fifth day; they suffer then from diarrhoea and are quiet; on the sixth or seventh day most of them are found dead, rarely do they survive till the eighth day, nor do they die before the fifth day. This course of the disease, the symptoms, and the pathological appearances after death are very uniform, and definitely distinguish it from fowl cholera.

By heating recent broth culture to 55° C. for twenty minutes, the writer has succeeded in so attenuating the microbe that when inoculated into fowls these become slightly ill, but they survive and prove themselves refractory against a second inoculation with virulent material. The writer has also found that continued sub-cultures on gelatine, kept up for many generations, attenuate the microbe, so that inoculations with such cultures do not produce death, but only a transitory illness, and the animals are hereby protected against a second inoculation.

Grouse Disease.—The fatal disease which affects red grouse, and known as the *grouse disease*, is an acute infectious disease, of which the chief, and we may say the essential, pathological character is that of a severe pneumonia, the lungs being greatly congested, and sometimes one or the other portion almost in a state of red hepatisation with engorgement of the blood-vessels and extravasation of blood into the air-spaces; the serosa and mucosa of the intestine show patchy redness; the liver is greatly congested and dark; the spleen is not enlarged. In the diseased lung and in the liver there occur in the vessels and in the extravasated blood numerous bacilli singly, or more commonly in larger or smaller groups, sometimes forming emboli in the capillary blood-vessels. These bacilli belong to one and the same species; they are motile, either oval or even coccus-like, and some few are rod-shaped. By cultivation on gelatine they can be easily obtained in numerous colonies from the sanguineous juice of the lung and liver; only in few cases are they to be seen in the heart's blood, both in cover-glass specimens and in culture. The morphological characters of the microbe are shown in figs. 47–49.

The microbe when examined from a cultivation is often rod-shaped—more often than in the tissue of the grouse. The motile forms are common in recent cultivations; in cultivations some days old most of the microbes are non-motile. Cultures inoculated into mice and guineapigs produce general infection, and speedy death, mice being more susceptible than guineapigs; in both animals the disease produced is a double-sided pneumonia. Sparrows are also susceptible, but less so than the common bunting and yellow-hammer, which birds are highly susceptible; also in these the disease produced is a double-sided pneumonia. The microbe is present in numbers in the heart's blood, but particularly in the diseased lung.

Fowls, pigeons, and rabbits are insusceptible to the disease. These two species—the bacillus of fowl enteritis and the bacillus of grouse disease—owing to their size and cultural characters, also belong to the group or family of bacilli mentioned on a former page, which include fowl cholera, septicæmia of rabbit, Frettohenneuche, swine fever, and Wildseuche.

Swine Erysipelas (Mal rouge, Rouget, Red Soldier).—An acute infectious disease, to which swine are very susceptible, and of which about 60 per cent. of those attacked succumb. The affected animals are quiet, their voice is hoarse, and the temperature is much raised; on the skin of the neck, chest, abdomen, and thighs extensive red patches of swollen oedematous skin are noticed; under convulsions—occasionally paralysis of the

hind extremities—the animals die in from twelve hours to three or four days after the first symptom. On *post-mortem* examination hæmorrhage is found in the affected patches of the skin, the lymph glands are swollen and much congested, the peritoneum is inflamed, the mucous membrane of the intestine is much injected and œdematous, the Peyer's glands are swollen, the spleen and liver are much congested and slightly enlarged.

The blood of the heart, and particularly the juice of the lymph glands and the spleen, contain fine bacilli very similar to those of Koch's mouse septicæmia (Schütz), 0.6–1.8 μ long. In microscopic sections through the liver, spleen, kidney, and lymph glands the bacilli are easily demonstrated in the capillary blood-vessels, either isolated between the blood-corpuscles or enclosed in the swollen leucocytes. As regards cultural characters, they completely resemble those of the mouse septicæmia (Koch) described on a former page.

Swine fed or inoculated with the blood or tissues of a swine dead of the disease become also affected. Mice¹ and pigeons are very susceptible to the disease; guineapigs and fowls are refractory; rabbits show generally only a local effect; mice die in two or three days, pigeons in three to four days; in all, the blood of the general circulation and of the organs contain abundantly the bacilli. In the pigeon numerous white blood-corpuscles in the vessels of the viscera are filled with the bacilli.

Pasteur has shown that the virus in its passage through a series of pigeons increases in virulence, both as regards the pigeon as well as the pig; on its passage through a series of rabbits, it increases in virulence as regards the rabbit, but decreases in virulence as regards the pig. Pigs inoculated with blood of the last rabbit of the series become ill, but recover, and are then found protected against the virulent disease.

Swine Fever.—This disease prevails largely in this country; in America it is known as hog cholera, on the continent of Europe as swine plague. It is a highly infectious disease, spreading from animal to animal by way of air, food, water, the lungs and bronchi and the intestines being the chief seat of the disease, and containing the virus. The infection is, under natural conditions, attributable to the virus being derived from and spread by the expectoration of the lungs and the evacuations of the bowels. Both by feeding, respiration, and by inoculation with particles of the diseased lung and intestine, the disease is easily reproduced in healthy swine. After an incubation period varying from between two days and six to seven days the animals are quiet and refuse food, the body temperature shows slight rise, red patches of a transitory nature are noticed on the belly and thighs; cough and occasionally diarrhoea of fluid evacuations soon declare themselves; the inguinal lymph glands appear enlarged. In severe cases the diarrhoea increases, the fever continues, the cough becomes more pronounced; this state lasts for a few days, seldom more than a week, and under general prostration the animal succumbs. In a large percentage (50) of cases the animals recover. In a considerable percentage of cases, the disease is very mild and diagnosed only with difficulty: the rise of temperature is but slight and transitory, lasting only a day or two; the animals feed fairly well, and have a slight cough occasionally and at long intervals; the inguinal glands are slightly enlarged. These symptoms are so slight and so little marked that it requires careful examination to diagnose the disease; nevertheless, on auscultation of the chest, distinct lung disease may be recognised. On *post-mortem* examination of such slight cases the disease of the lung is easily confirmed.

In the well-pronounced cases terminating in death the *post-mortem* examination shows the following appearances: the lungs of both sides show severe extensive lobar pneumonia, involving sometimes the greater part of the lung; in recent cases the lobules show all stages between congestion (punctiform hæmorrhages) and hepatisation; the lobes that are longer affected show more consolidation, and the older this is, the more grey, solid and necrotic, dry and friable, is this part of the lung; the septa between the lobes are œdematous and well marked; the bronchi and trachea contain grey and sanguineous muco-purulent matter; the endocardium of the left, and occasionally of the right ventricle near the atrio-ventricular valves, and also these latter show patchy and punctiform hæmorrhages; the liver is congested, and occasionally shows dark red patches, due to hæmorrhage; the spleen is enlarged and dark; the colon and cæcum, particularly the former, contain punctiform hæmorrhages; in many cases also prominent, round or oval isolated, or in severe cases, more or less confluent ulcers (necrosis), showing an infiltrated base, and being stained greenish black by altered bile pigment; between a few small round ulcers near the ileo-cæcal valve to very numerous extensive ulcerations, comprising occasionally extensive areas of the mucous membrane of the cæcum and colon, all intermediate

¹ Mice die with congested and enlarged spleen and greatly congested lungs; the intestines are relaxed and filled with sanguineous mucus; the kidney and liver are enlarged and congested.

stages can be noticed (*see* Klein, in the 'Report of the Medical Officer of the Local Government Board for 1878').

In the stomach hæmorrhagic patches can occasionally be seen. The lymph glands along the bronchi, and the mesenteric and pelvic glands, are swollen, juicy, dark red, in part or wholly, and contain hæmorrhages. The peritoneum is inflamed, and on its surface are clumps of solid lymph composed of leucocytes. Owing to the lungs and intestines being found constantly affected, the disease has been designated by the writer pneumo-enteritis; but in Germany (Schütz) and in America (Salmon), it is asserted that the above disease is really two—one a disease of the lung, the other of the intestine; but from experiments made on a large scale with diseased lung, and with diseased intestine, and from the *post-mortem* appearances in well-defined localised outbreaks that the writer has made, he is of opinion that this division cannot be maintained, but that the swine fever in this country is one single disease, viz. pneumo-enteritis. Microscopic examination of the lung and intestine shows that the disease really commences with congestion, stasis, and hæmorrhage, leading to infiltration and necrosis of the affected parts. The cause of the disease is a bacillus, which, in the affected tissues of the pig, appears, as a rule, as a short rod, often constricted in the middle; in fluid cultivations (broth) and in animals (rabbits, mice) as a cylindrical rod, singly or in dumb-bells, occasionally growing to considerable length, and forming longer or shorter chains; but there can be always found short forms almost like oval cocci, rods, and cylindrical bacilli. Cover-glass specimens and cultures of the lung, spleen, lymph glands, and the sub-mucous tissue of the affected intestine, demonstrate the presence of the bacilli. These bacilli are motile, though the motility is observed in a minority; in cultivations in broth, gelatine, and agar, many of the bacilli are motile during the first few days, but lose their motility later.¹

In gelatine plate cultivations the colonies, when first noticed, i.e. after about twenty-four hours, are greyish dots; in two to three days they are conspicuous as whitish round specks of about the size of a large pin's head; in transmitted light they appear brownish, granular. In stab cultures, the stab of inoculation becomes marked as a white line made up (when seen under a glass) of minute globules closely placed side by side; on the surface of the stab is a small irregularly outlined whitish patch. In streak culture the line of inoculation is occupied in a few days by a grey band, knobbed or crenated in its outline. On agar the growth (at 37° C.) is a greyish brown smeary film, rapidly spreading over the surface of the agar. In alkaline broth at 37° C. uniform turbidity is produced; after a few days a voluminous greyish white precipitate is noticed at the bottom of the tube. No distinct pellicle is formed on the surface.

Inoculation of swine with cultures produces the disease, but this does not lead to death, and such animals after recovery show themselves refractory against inoculation with material of the diseased lung or intestine.

Inoculations into guineapigs with material from the diseased swine produce at the seat of inoculation hæmorrhagic infiltration and thickening, sometimes leading to death in two or three days; often, however, the thickening passes off in a week or so; cultures injected subcutaneously in guineapigs produce thickening at the seat of inoculation but rarely death.

Inoculation into mice of minute particles of material of the diseased lung, or intestine, or of gelatine or broth-culture of the bacillus of swine fever, causes disease and death in four to eight days: the spleen is found enlarged and dark; the liver is mottled with grey dots, streaks, and patches of necrotic tissue; the peritoneum is inflamed, and so are the kidneys, as also both lungs. Cover-glass specimens and cultures from the heart's blood, liver, kidney, and particularly the spleen, demonstrate the presence of the bacilli in large numbers (*see* fig. 56). Among the bacilli in the spleen, numerous long cylindrical rods can be seen. In the kidney many of the capillaries of the glomeruli are plugged by the bacilli, so also in the liver.

In the rabbit, inoculation produces disease and death in a few days: the spleen is slightly enlarged, the lungs are inflamed, the kidney is much congested in the cortex. Here also the bacilli can be easily demonstrated in the heart's blood, the liver, and the kidney; in this latter many Malpighian corpuscles show the capillaries of the glomeruli plugged with masses of the bacilli.

As we mentioned above, although the microbe of Davaine's or Koch's septicæmia in

¹ It is curious to find Flügge (*Die Microorganismen*, 2nd edition, 1886, p. 249) maintaining that, because the writer stated that the bacillus of swine fever is motile like a bacterium *termo*, he could not have had before him the bacillus of swine fever. Flügge evidently considers swine erysipelas and swine fever as one disease.

the rabbit, the microbe of fowl cholera, and others (Fretschensseuche, Wildseuche, fowl enteritis), and the microbe of swine fever are in many microscopic and cultural respects similar, it is erroneous to suppose that they are identical; there are sufficient differences amongst them in cultural respects alone to distinguish between them; but in their action on animals they are distinctly different. The microbe of Davaine's or Koch's septicæmia produces a disease in rabbits, and particularly in mice, different from that of the microbe of swine fever; the microbe of fowl cholera and of rabbit septicæmia act differently on swine from that of swine fever. But all these, no doubt, belong to the same group or family of species as mentioned above; their microscopic and cultural characters, and to a certain degree also their action on animals, being similar though not identical.

A disease amongst cattle (Binderseuche) and horses, and amongst deer (Wildseuche), manifesting itself in diffuse pneumonia and hæmorrhagic enteritis, but without necrotic change (consolidation and dryness) of the lung, and without ulceration of the intestine, was first recognised by Bollinger. Kitt has shown that this affection is caused by a bacillus in many respects (morphological and cultural) similar to that of fowl cholera, rabbit's septicæmia, and swine fever; and Kitt and Hueppe maintain, indeed, the identity of all of these microbes; but the evidence brought forward by them is not satisfactory. True, rabbits inoculated with the microbes obtained from Davaine's septicæmia, fowl cholera, swine fever, or Wildseuche succumb under the symptoms of Davaine septicæmia; it is likewise true that pigeons inoculated with cultures derived from either of these diseases die with symptoms similar to fowl cholera; still a great deal remains yet to prove the identity, as regards the action on swine, of the bacteria of rabbit's septicæmia, fowl cholera, and Wildseuche. To mention only one series of difficulties. Fowls, as mentioned above, are highly susceptible to the microbe of fowl cholera, but they are insusceptible to the microbes of swine fever or Wildseuche.

Billings ('Texas Fever,' Lincoln, Nebraska, 1888) describes a species of small motile bacilli, closely related to the bacilli of swine fever, both as to morphology, cultural and pathogenic characters, as the cause of the cattle plague in Texas and other southern parts of the United States.

Charbon symptomatique.—This disease, affecting young cattle and sheep, occasionally produces great mortality amongst them, particularly amongst the former. Owing to its involving chiefly one of the hind extremities in the form of a large subcutaneous tumour, which, on incision, shows a quantity of sanguineous, discoloured, almost black fluid, the disease is called *quarter ill* or *black leg*. Owing to its slight resemblance to anthrax (large tumour containing serous sanguineous fluid) it is called in France *charbon symptomatique*; in Germany it is called *Rauschbrand*, on account of the emphysematous nature of the tumour, and on account of the gangrenous nature of the infiltrated tissues. The disease, when it appears, rapidly spreads amongst young cattle and sheep, seldom amongst horses; it is unknown amongst swine or poultry. The animals affected are quiet, do not feed, and show high temperature; on one or other of the quarters—generally one of the hind—there appears a large diffuse swelling, on account of which the animal is lame and cannot move that extremity. In the course of thirty-six to forty-eight hours death takes place. On *post-mortem* examination the tumour is seen to be located subcutaneously; here the connective and muscular tissues are dark, almost black, gangrenous, and contain a large quantity of sanguineous serum and a large quantity of gas bubbles (said to be CO₂ and methane). The infiltration with sanguineous serous fluid extends for some distance into the adjacent parts of the muscular tissue; there is congestion of the liver, spleen, kidney, and particularly the subcutaneous lymph glands: these, beginning from near the tumour, are found swollen, dark, and on incision a sanguineous fluid oozes out from them. The spleen is only very slightly enlarged. Cover-glass specimens of the subcutaneous and muscular infiltration at or near the tumour, particularly of the subcutaneous lymph glands, show in considerable numbers small motile bacilli 8–5 μ long, and about 0.5 μ thick: they are rounded at their ends, and some con-

tain terminally a bright oval spore ; others possess a terminal enlargement without a spore (Bollinger, Arloing, Cornevin, and Thomas).

Immediately after death the bacilli are not easily demonstrable in the heart's blood, being present only in small numbers, though they can be shown to be present in the liver, spleen, and kidney, but always more numerously if some hours are allowed to elapse after death.

The exudation of the tumour or of the surrounding muscular tissue injected subcutaneously into guineapigs in comparatively large quantities ($\frac{1}{2}$ –1 Pravaz syringe) produces the same kind of emphysematous gangrenous infiltration with sanguineo-serous exudation at or near the place of inoculation ; the animals die in from twenty-four to sixty hours, the internal viscera show great congestion ; in the subcutaneous tumour, in the blood of the heart, and in the juice of the viscera, the bacilli can be easily demonstrated ; in the blood and viscera they are fairly numerous if some hours have elapsed after death (*see* fig. 71).

If only a drop or two be injected, the guineapigs, though they become affected with the local disease, do not succumb, but show themselves refractory against infection with large quantities, such as in control animals would invariably produce death. Rabbits are only slightly susceptible.

Arloing, Cornevin, and Thomas have brought to light various important facts connected with the action of the bacilli. These authors cultivated the bacilli in broth, but they found that the bacilli grow best in chicken broth, glycerine and sulphate of iron, provided oxygen (air) is excluded ; they are, therefore, true or obligatory anaërobic bacteria. They grow well in grape sugar gelatine, but must be inoculated into the depth of it. The character of the growth is similar to that produced by Koch's malignant cedema bacillus, except that the former forms fewer gas bubbles, grows much faster, and afterwards does not form such voluminous fluffy masses in the liquefied gelatine as the bacillus of malignant cedema.

Arloing, Cornevin, and Thomas have shown that if small quantities of the fluid of the natural tumour be injected subcutaneously into cattle, only a local, though typical tumour is the result ; the animals recover, and then are possessed of immunity against further inoculation with otherwise fatal doses.

Further, they found that three to five drops of the tumour fluid injected into the vein of cattle—but without inoculating the subcutaneous tissue around the vein—produce only a transitory febrile disturbance ; the animals quickly recover, and show themselves refractory against subcutaneous fatal doses. A safe mode of protective inoculation used by these observers successfully on a large scale is this : The tumour fluid is rapidly dried at 82–85° C., then it is rubbed up with water, and heated to 100° C. Another lot is treated in the same way, but heated only to 85° C. ; the first lot represents a first vaccine (*premier vaccin*), the second lot a second vaccine (*deuxième vaccin*) ; both can be dried and sent to distances ; when required for use the dried matter is rubbed up in 100 parts of water, and of this 1 c.c. per animal is subcutaneously injected. The *premier* (weaker) vaccine must be used first ; after the lapse of about ten or twelve days the *deuxième* (stronger) vaccine is injected. Animals thus twice vaccinated proved themselves completely protected against a fatal and virulent dose taken from the natural tumour.

Though there exists, both as regards the pathology and the microbes, a certain resemblance between the malignant cedema and the charbon symptomatique, this resemblance is only superficial, and there can be little doubt that the two diseases in their pathology, in their microbes, and their transmissibility or non-transmissibility to certain animals are *totally different* diseases.

CHAPTER XV

ANTHRAX

ANTHRAX is a fatal acute disease affecting naturally sheep, cattle, horses, goats, deer, and man. A conspicuous symptom on *post-mortem* examination is the dark, greatly congested and enlarged spleen, the enlargement amounting to three or four times the natural size of this organ; hence the name Milzbrand, or splenic apoplexy. In sheep and cattle there occur hæmorrhagic exudations under the skin of various regions, the exudation forming tumours of a dark to black gelatinous nature; hence the name anthrax, charbon: in human beings affected by accidental inoculation of the skin, a large pustule or swelling with black core is found; hence the name malignant pustule, or carbuncle. This disease, when naturally contracted by sheep, cattle, goats, and horses, declares itself by rise of temperature, and rapid loss of muscular power; the animals are very quiet, do not move and do not feed; twenty-four hours after, or even sometimes before, they are found dead. In some virulent epidemics of anthrax the animals have been seen to die in a few hours after the first symptoms set in; such cases are known as fulminating anthrax. On *post-mortem* examination, gelatinous sanguineous tumours are found in various parts under the skin; the spleen is dark, soft, full of blood, and several times its natural size; the liver, lungs, kidney, stomach, intestines and lymph glands are greatly congested, and often show hæmorrhages; the serous membranes are congested and show petechiæ; the blood has not much tendency to coagulate. Examined under the microscope, in all these organs the capillaries and veins are found distended and filled with blood; in many places, e.g. in the alveoli and bronchi of the lung, in the spleen, in the vessels of the liver, kidney, omentum, intestinal mucous membrane, and subcutaneous tissue, there are ruptures of the capillaries and small veins, in consequence of which blood *en masse* is effused into the surrounding tissue. In sheep, goats, horses, and cattle there are always sanguineous effusions from the stomach and lungs by the mouth and nose, from the kidney and bladder by the urethra, and from the intestine by the anus.

Brauell and Davaine, about twenty-five years ago, showed that in the blood of the general circulation, in the spleen, and in the subcutaneous effusions there always occur straight, non-motile rods (*bacteridiæ* of Davaine), which were considered to be probably connected with the cause of the disease. Zürn, Klebs, and others have shown that the blood of animals dead of anthrax—which blood is very virulent when inoculated into fresh animals, for it produces the fatal disease—fails to produce the disease after the rods are removed from the blood by filtration.

Koch in 1876 carefully studied these rods, and has shown that they are bacilli, which except for their non-motility, and certain small differences—to be mentioned presently—grow and multiply, and form threads and spores after the manner of the *bacillus subtilis* of Cohn. Koch studied directly under the microscope bits of spleen removed from a mouse dead of anthrax after inoculation, and he saw how the rods elongate and divide, how they gradually grow out into smooth threads in which after a day or two bright glistening oval spores appear, how in the meantime the threads swell up and disintegrate and the spores become free; he further observed how such spores germinate again into the bacilli (see a former chapter), how these again multiply by division, grow into threads, and finally again form the bright oval spores. With the spores, by inocula-

tion of mice and guineapigs, he readily produced anthrax, and death in thirty-six to forty-eight hours; the spleen of these animals was found greatly enlarged, dark, full of blood, every drop of the blood of the spleen and the general circulation teeming with the bacilli. In microscopic sections he found the capillary blood-vessels in many organs almost filled with the bacilli.

The bacilli are found in the blood and spleen of every animal dead of anthrax, more numerous after than before death, though there is no difficulty in demonstrating them an hour or so before death; a few hours after death they are found as a rule in the blood of the spleen in great crowds, and to a lesser degree in that of the general circulation.

In fine sections through the spleen a very striking difference is noticed between the pulp and the Malpighian corpuscles, the former being full of the bacilli, the latter containing them either not at all or only sparingly.

The bacilli examined fresh are non-motile rods, homogeneous-looking, more or less truncated at their ends; their length differs very greatly from cylindrical rods to short, uniform threads or chains of bacilli, the latter 5–20 μ in length; their thickness amounts to 1 μ or a little over. There exist certain differences in so far as most of the bacilli in the blood and spleen are shorter when death ensues rapidly than when it ensues late, say after three days or later; in the guineapig dead from inoculated anthrax, in between thirty-six and forty-eight hours, the bacilli are longer than in the sheep dead in the same time; the bacilli are also a little thicker in the guineapig than they are in the mouse or sheep. Koch was the first to show in cover-glass specimens of the blood or spleen juice (dried and stained) that the bacilli, though in the fresh state they appeared as uniform cylindrical rods, are really made up of short, cubical, or more or less elongated protoplasmic elements, all with square cut or even with concave ends,¹ and this appearance has a certain diagnostic value, since it is not generally seen, or at any rate it is not so marked in other bacilli; but it must be remembered that the above character is not invariably shown by the anthrax bacilli; in the mouse many bacilli do not show it, since many of the longer forms are made up, not of short cubical, but of long cylindrical elements, and further the writer has seen bacilli (e.g. the *bacillus filamentosus*) in which the above appearance was as pronounced as in typical anthrax bacilli.

The longer bacilli or their chains, taken from an animal dead of anthrax, or from an artificial culture, and examined in dried and stained cover-glass specimens show within a common sheath cubical or rod-shaped cylindrical, square cut, stained masses of protoplasm; these are the real bacillary elements; the cylindrical elements often show a middle constriction more or less deep, indicating the division into two elements: these will be of course cubical if the original cylindrical element is short; between adjoining elements a transverse septum can be occasionally seen. In specimens made of artificial cultures these appearances are more easily noticed, since they are more pronounced; in some anthrax-threads of cultures all elements constituting a thread are separated one from another by a transverse septum.

The mode of infection of animals under natural conditions is effected by their grazing on land previously infected with the spores of bacillus

¹ The illustrations given by Koch and others as to the concave ends of the bacilli seen in dried and stained cover-glass specimens, though of diagnostic value, are not pre-formed, but are due to the methods employed; in sections through the properly prepared or fresh tissues the bacilli do not show this appearance.

anthracis. As mentioned above, in sheep, goats, cattle, and horses there are always discharges from the mouth, nose, anus, and urethra, full of the bacillus anthracis: these discharges soak into the soil where the animal falls dead of anthrax. If the superficial soil is favourable for the multiplication of the bacilli and the formation of the spores—and most soil in the spring and summer, owing to temperature and an abundance of proteid nutritive material and on account of its containing air, is favourable both for the multiplication of the bacilli and for the spore-formation, particularly when owing to the presence of a small amount of lime the reaction of the superficial layers is only neutral or even slightly alkaline—very soon a large and abundant crop of spores will be the result. Add to this the circumstance that when an animal falls dead of anthrax in a field, it is not at once buried; nor is the earth surrounding the animal and saturated with the infective discharges disinfected; but, on the other hand, the animal is generally carted away to a distant point in the field, perhaps a corner where the dead animals are buried; and in this way blood and fluids full of the bacilli are dropped about; besides, before being buried the animal is generally skinned, and hereby a lot of blood full of the bacilli is smeared about the ground. Every copious watering, as by the spring and autumn rains, will subsequently distribute the spores over wide areas; these spores, owing to the favourable temperature during the spring, summer, and early autumn, will be able to germinate, and by multiplication of these bacilli to produce an abundance of further bacilli in which spore-formation will again proceed. Thus within a few seasons an amount of infective spores will be produced capable of giving infective power to large areas. Sheep or cattle feeding or grazing on such land are liable to infect themselves with the spores. Feeding on bacilli alone does not produce infection, as has been proved by numerous experiments, but spores may pass unscathed through the stomach and be absorbed into the system, and thus produce anthrax; likewise inhalation of spores produces anthrax. Water infected with spores of anthrax from such a field, or from other sources, is infective for sheep and cattle.

Infection through cutaneous inoculation (abrasions, ulcers, &c.) is not common in sheep or cattle, though in horses, infection and local malignant pustule is not uncommon. Men that have handled the carcasses of sheep, cattle, and goats dead of anthrax have, by subsequently grooming horses, been known to inoculate these latter with anthrax, in consequence of which malignant pustule has been produced at the seat of inoculation, leading occasionally, if not cut out, to general fatal infection with anthrax.

Bollinger has shown that infection of animals may be effected by the instrumentality of flies: these, having been previously sitting on the carcass of an animal dead of anthrax, carry matter (blood, oral, nasal, and other discharges), containing the bacilli anthracis, to another animal on which they alight, particularly on sores or abrasions of such an animal, and hereby inoculation with the anthrax bacilli may be easily effected.

Pasteur maintained that in the body of sheep, dead of anthrax and then buried, the bacilli form spores, which latter in the course of years may be brought up to the surface by earthworms, and become a source of infection for animals grazing here. Koch has shown that the bacilli anthracis do not form spores unless well exposed to air; they do not form spores within the body of an animal dead of anthrax. The statement of Archangelski, that in the blood of the living animal affected with anthrax there occur 'spores,' which under certain conditions of cultivation can be shown to divide and to multiply as spores, cannot be accepted as correct, as the writer has shown in his Report to the Medical Officer of the Local Government Board

for 1882. Further, the writer has there shown (*ibid.* 1882) that if an animal dead of anthrax is buried or left unopened, all bacilli anthracis in the blood and organs of such an animal degenerate in the course of several days, so that even large quantities of blood or spleen taken after this period and injected into a suitable animal do not produce anthrax. Mice and guineapigs dead of anthrax are buried in earth or left unopened, after a week the spleen is used for inoculation of mice and guineapigs, but it fails to produce anthrax (*ibid.* 1882);¹ the bacilli, not having been able to compete with putrefactive organisms, and no free oxygen being present, have all degenerated.

Anthrax in man takes place after inoculation of a cutaneous wound by persons handling the carcasses of animals dead of the disease, e.g. grooms, shepherds, cattle drovers, butchers, knackers, &c. The place of inoculation becomes a large, more or less firm, discoloured, painful tumour after sixteen to twenty-four hours; in its centre, i.e. at the point of inoculation, there is a black core; the connective tissue of the tumour is greatly infiltrated with serum and blood, the whole infiltration being of a sanguineous, gelatinous nature. In the exudation there are numerous bacilli anthracis; the infiltration and phlegmon gradually extend into wider areas, with constitutional disturbance, i.e. fever and pains all along the infected part. In few cases a general infection, i.e. invasion with bacilli from the carbuncle, takes place; in these cases the bacilli are found in the blood, but only rarely does a fatal result ensue.

Another mode of infection is that with general anthrax, and this in a large percentage of cases leads to a fatal issue; in all cases it is associated with severe febrile disturbance; the general infection occurs either by the alimentary canal or by the respiratory tract; in both instances the bacilli anthracis enter in the form of spores. In woolsorters' disease, in anthrax of rag-sorters, and in anthrax of hide-sorters it has been conclusively shown by Mr. Spear (*see* his Reports to the Medical Officer of the Local Government Board for 1881 and 1882) that in one case the dust of infected wool, in the other the handling of the infected hides, was the source of the contagium. In localities in which anthrax occurs almost endemically amongst sheep or goats, e.g. certain parts of Russia, Armenia, Turkey in Europe and Asia, the fleeces of animals dead of anthrax are preserved, their wool being as a matter of course smeared with blood and other excretions containing the bacilli anthracis. In these materials there is every opportunity for the bacilli to form spores, which resist drying and other processes fatal to the bacilli as bacilli. These spores remain adhering to and buried within the wool. The wool brought to a factory is first of all turned out from the bales and sorted; hereby a large amount of dust is separated which during the sorting and other subsequent processes is sure to become mingled with, and suspended in the air of the work-rooms; the workers in such rooms are therefore particularly liable to infect themselves with the spores, either by mouth and alimentary canal, or by the respiratory organs. The disease which ensues has been carefully described by Mr. Spear: it resembles in a marked degree the general virulent form of anthrax in animals, the congestion and enlargement of the spleen, the extreme congestion of the lungs, and the hæmorrhage in the air cells and bronchi, the congestion of, and hæmorrhage in other viscera being the most prominent symptoms. The blood of the general circulation and all the vessels of the organs contain the bacilli anthracis. The water which is used for the washing of the infected wool has been known to contain the spores and to produce anthrax in cattle that had drunk it. Hides coming from a country where anthrax occurs amongst

¹ Esmarch in 1889 arrived at the same results (*Zeitschrift f. Hygiene*).

cattle (China, Russia, Turkey) are in the same position as wool; when the hide is taken off from the animal that has died of anthrax there is also on the inside of the hide a large amount of blood smeared over and adhering to it, besides there is blood naturally contained in the subcutaneous tissue. All this blood teems with the bacilli anthracis. Such hides are exposed to drying, but as this process always takes a good many hours there is every opportunity for the bacilli, that are exposed to the air, to form spores. As the subcutaneous tissue dries, these spores become well fixed on and in it; the drying does not, of course, interfere with the life of the spores. In addition to this, the hides are subjected to a process of curing, i.e. certain chemicals perfectly harmless to the spores are coated over the inner surface of the hide by which the spores become only more shielded from being lost. So that when such hides are afterwards handled, dust and particles containing the spores coming off from them readily produce infection either through the alimentary canal or through the respiratory organs, or a local carbuncle is produced by inoculation. Infection through the alimentary canal of human beings generally declares itself by prominent symptoms on the part of the alimentary canal, *mycosis intestinalis*: great congestion, inflammation, and hæmorrhage of the bowels; congestion, inflammation and hæmorrhage of the mesenteric lymph glands. The intestinal contents, the lymphatics of the intestine, and the mesenteric glands contain abundantly the anthrax bacilli. A general invasion of the system and fatal anthrax is the result.

Fatal anthrax in ragsorters has been observed several times—see Paltauf ('Wiener klin. Wochenschrift,' 1888, Nos. 18–26)—but not all acute infectious diseases contracted by the sorting of rags is anthrax, as has been proved by the research of Bordoni Uffreduzzi mentioned on a former page in connection with septicæmia in man caused by the proteus hominis.

While sheep and goats, cattle and horses, white mice and guineapigs, are easily infected with anthrax by inoculation, rabbits, although fairly easily susceptible to it, are a little less so than mice and guineapigs; pigs are difficult to infect, though they can be infected (Crookshank); adult rats, dogs, and cats are almost insusceptible; fowls in the healthy state are insusceptible, but Pasteur found that by cooling their bodies they can be infected with fatal anthrax. Chauveau has shown that also amongst sheep those of Algiers are to a large extent refractory against anthrax.

By subcutaneously inoculating sheep, cattle, and guineapigs with a drop, or a fraction of a drop, of the blood of sheep or cattle dead of anthrax, or with virulent culture of anthrax bacilli, there is found at the seat of inoculation in from sixteen to twenty-four hours a tumour due to œdema; after inoculation with spores somewhat later. This increases during the next day to a very considerable degree and involves extensive areas of the subcutaneous tissue. In cutting into the tumour it is seen that it is due to a gelatinous infiltration of the connective tissue and slight hæmorrhage, the infiltration abounding in bacilli anthracis; from the gelatinous infiltration, on incision, clear serum containing bacilli and a few red blood-corpuscles easily and spontaneously separate; the infiltration has no odour. The temperature of the body is greatly raised, to 41° C. The animals die in between thirty-six and fifty-four hours. Some hours before death the temperature rapidly falls to 36 or 35° C. The animals are weak, and appear paralysed. On *post-mortem* examination the spleen is always found greatly enlarged, the lungs, liver, kidney, and peritoneum much congested, and often showing hæmorrhagic spots; the blood has not much tendency to coagulate. In the heart's blood, the spleen, and in all vessels of the organs the anthrax bacilli

are found in great numbers. The inoculated disease is therefore identical with the one occurring naturally.

There exist considerable differences in the degree of virulence shown on inoculation of different animals with the blood of anthrax derived from different sources. Thus blood of mice that die from typical anthrax within forty-eight hours produces on inoculation typical and fatal anthrax in mice, guineapigs and rabbits, but in sheep and cattle it does not do so; it produces a local oedematous tumour with fever; but this, after two to three days or later, again disappears and the tumour gradually vanishes. In sheep the effect is more severe than in cattle, but such animals are refractory to a second inoculation with virulent anthrax, which otherwise in healthy sheep would produce fatal anthrax. Blood of guineapigs dead of typical anthrax within forty-eight hours, or so, inoculated into mice, guineapigs, rabbits, and sheep, produces as a rule local tumour and general fatal anthrax in forty-eight hours or so; but in cattle a local tumour follows, sometimes severe and extensive, with constitutional disturbance, but as a rule no fatal result, and such animals are refractory to a second inoculation with virulent material. Blood of sheep and of cattle dead of typical anthrax produces on inoculation in all animals typical and fatal anthrax. So that we conclude from this that the virulence of the bacillus anthracis growing in the body of a mouse is of a lesser degree than that derived from the body of a guineapig; that the virulence of the bacillus anthracis derived from the body of a guineapig is of a lesser degree than that of the sheep or cattle; further, to produce fatal anthrax in the mouse, anthrax of a lesser degree of virulence than for the guineapig is sufficient; for this, again, a lesser degree of virulence than for the sheep, and for this again a lesser degree of virulence than for cattle. Or, to put it in a different form: anthrax bacilli from a virulent source, say of sheep or cattle, on their passage through the mouse suffer attenuation of virulence, but on their passage through a sheep, or still more through the body of cattle, do not lose in virulence.

That human beings contract local anthrax (*carbuncle*) by inoculation from blood of sheep, cattle, or horses is well known, and has been mentioned above; that human beings contract general anthrax from spores of anthrax of sheep or cattle is proved by woolsorters' and hidesorters' disease, but there is no record that anthrax, local or otherwise, has been contracted by human beings from mice or guineapigs in the very many instances in which in laboratories the bodies of these animals dead of anthrax have been handled. The writer has seen a good many instances in which the *post-mortem* examination of the bodies of these animals had been carelessly performed by assistants and others, who sometimes had abrasions and wounds on the fingers, yet no infection has resulted from it.

Various degrees of virulence are also noticed of artificial cultivations of anthrax, though all yield growths which in morphological and cultural characters do not differ one from another. Thus anthrax bacilli grown for some weeks at 37° C. on distinctly alkaline agar are less virulent than when grown on neutral or faintly alkaline agar or on potato; anthrax bacilli grown in faintly alkaline broth or on neutral or faintly alkaline gelatine are more virulent than when grown on distinctly alkaline gelatine; the spores found early in cultures of broth and gelatine cultures are more virulent than the bacilli of the same cultures. The different degrees of virulence are estimated by this: cultures that kill every guineapig of about the same age (young animals are more susceptible than old ones) with typical anthrax within forty-eight or fifty hours are virulent; if only a percentage of them die within that time, others during the fourth or fifth day or later, the virus

is of a lesser degree ; further, the less resistant to perchloride of mercury or other agencies the bacilli or spores are, the less virulent they are found on inoculation into guineapigs, and the less virulent also their sub-cultures. More than that, the less virulent the anthrax is at starting, the less virulent (*cæteris paribus*) also the bacilli when taken from a guineapig infected with such culture (see the writer's Report to the Medical Officer of the Local Government Board, 1885 ¹).

The bacillus anthracis offers some very characteristic features in cultivations. In gelatine plate cultivations made of the blood (previously well diluted with neutral salt solution or broth, on account of the large number of bacilli, present in the blood), already after twenty-four to thirty-six hours the first signs of colonies can be made out in the form of translucent grey angular dots ; after forty-eight hours to three days they are conspicuous by their size, and by their margin being distinctly made up, to the naked eye, of filaments, either straight or bending like loops. Under the microscope the filamentous nature of the colonies is distinctly seen ; the filaments looked at under a magnifying glass are more or less in bundles twisted like cables, and extending sometimes like radii from a centre ; at the margin this is particularly conspicuous. At the same time the colony is seen to be sunk in the middle, being situated in a slight depression of the gelatine due to commencing liquefaction. Looked at obliquely, the gelatine looks pitted by the colonies. As growth proceeds the colony enlarges, the marginal loops and bundles of twisted filaments project more or less irregularly, some project for longer, others for shorter distances, sometimes not much beyond the margin of the colony, and the gelatine surrounding the colony becomes more and more liquefied, but remains clear in the liquefied part. In stab cultures made from a culture or from the blood, the stab is noticeable after a day or two as a whitish line made up of closely placed dots ; in another day or two, from each dot a lot of fine whitish filaments are seen extending, often like rays from a centre. When the dots are closely placed in linear series, the white filaments projecting mostly in horizontal direction from them give to the stab a characteristic appearance, like the vane of a grey feather, the stab being the middle rib ; liquefaction has by this time set in on the surface, i.e. on the upper end of the stab, and there is here a more compact plate-like mass of filaments ; the liquefaction gradually proceeds into the depth while the surface patch of the growth increases in bulk ; the liquefied gelatine is clear, and the original surface growth occupies always the deepest part of the liquefied gelatine. When the surface patch while spreading remains adhering to the glass wall of the test tube, spore formation is observed in the threads of the bacilli, but when the growth is in the depth of the liquefied gelatine no spore formation ever takes place. After ten to fourteen days at 19–20° C. the upper half of the gelatine in the tube is quite liquefied, the liquefied gelatine is clear, and the whole growth is at the bottom of the liquefied part in the form of whitish grey fluffy masses ; when shaken the mass breaks up into whitish nebulous flocculi.

In streak culture on gelatine, the streak of inoculation is marked after twenty-four to forty-eight hours as a whitish grey line ; then a number of fine whitish threads shoot out horizontally from this line, liquefaction at the same time commencing and proceeding slowly and gradually ; the line thickens and broadens, and after a week is made up of masses of threads twisted and convoluted and forming a thick white filmy patch, which as liquefaction proceeds sinks to the bottom of the liquefied gelatine, forming here a whitish grey fluffy mass.

¹ Esamarch some years later arrived independently at the same results.

In neutral or faintly alkaline broth kept at 86–88° C. there is, if the broth be thin, uniform slight turbidity after thirty-six to forty-eight hours : flakes small and large then appear at the bottom of the fluid, while this latter remains fairly clear. As growth proceeds, about the end of the week, there are contained at the bottom of the fluid characteristic greyish fluffy loose nebulous masses, which are masses of anthrax threads matted together : these masses increase in bulk, and extend as it were from the bottom of the fluid towards the upper parts. If during the first few days some of the flakes remain adhering to the glass at the surface of the fluid, these flakes enlarge and form on the glass, on a level with the surface of the fluid, a sort of whitish ring, somewhat like a pellicle ; in this, copious spore formation takes place ; but in the tubes, in which all the growth is limited to the deeper parts of the fluid, no spore formation occurs at any time, since for the formation of spores a free and copious supply of oxygen is required.

On agar mixture at 86–88° C. a thick greyish film is noticed after two days along and beyond the line of inoculation. This rapidly increases in breadth till the whole surface of the agar is covered with a sticky pasty greyish layer ; this after some days shows some patches thicker than others, is light brown, and in some patches even dark brown.

On potato at 85–87° C. a thick cohesive layer like paste is formed : this is of a brownish colour, the growth is extensive after a few days. Both on nutrient agar and on potato, the film is a mass of threads matted together, and after two to three days copious spore formation is noticed in many threads ; at the end of ten days to a fortnight the whole of the film is a mass of spores ; little of the original bacilli is recognisable.

Whenever spores are formed in a culture (their mode of formation has been described on a former page), they remain active and living for indefinite periods, i.e. the spores when at any time sown into new suitable medium are capable of germinating and producing new crops of bacilli, and are capable of producing anthrax when introduced into an animal (mouse or guineapig) by spray or inoculation. But when no spores are developed, e.g. in gelatine and in broth tubes in the depth of the medium, and when none of the growth has remained adhering to the glass on the surface of the medium, the growth proceeds till all nutriment is used up ; but before this point is reached, numerous bacilli, parts of the threads, and whole threads undergo degeneration ; the protoplasm in the bacilli and threads becomes granular, and gradually disappears till only the empty sheaths—many of them collapsed—are left. This process of degeneration is noticeable in gelatine and broth already after a few days. In broth cultures in which the growth occurs in the depth of the fluid, in the form of the characteristic fluffy masses above mentioned, degeneration once set in rapidly extends to the whole mass ; by this the mass diminishes in bulk and becomes more transparent (*see the Report of the Medical Officer of the Local Government Board for 1882*). From such cultures after some time—several weeks to several months—no new sub-culture can be established, and no anthrax can be produced in animals. In agar, which has strong alkaline reaction, this process of degeneration is also noticeable without spores being formed ; the growth on the surface of the agar after several weeks to several months shows no longer any good anthrax threads, but is made up of granular *débris*.

The writer has described this degeneration as occurring occasionally in the bacilli while in the blood and in the spleen. It has been noticed within forty-eight hours in guineapigs and in sheep that died of typical anthrax, in which the number of the bacilli in the blood and particularly in the spleen were enormous ; in well-stained and well-washed cover-glass specimens large

numbers of the bacilli showed the degeneration distinctly—either the protoplasm of the whole bacillus was in a state of granular degeneration, or part of a chain was in that state (*see* fig. 60).

A change observable in the bacilli of cultures (gelatine, broth, agar, potato) and generally taken to be due to involution consists in the fact that one or the other elementary individual of a chain or thread appears swollen up, thickened, spindle-shaped, club-shaped, or even spherical. In the threads, in longer or shorter portions, all the elements may thus be changed. The writer has described and figured these changes (fig. 70) as the torula forms of bacillus anthracis; they were particularly abundant in neutral gelatine after two to three days. He has had cultures where some of the anthrax threads were thus transformed into chains of torula-like large elements—many times thicker than the typical bacilli—strung together into beaded necklace-like strings by thin short connecting bridges; there were other threads present, in which some elements were spherical, others oval; the appearances here described were exactly like those occurring in *saccharomyces mycoderma*. He has also shown that those torula-shaped bacilli are very virulent, and he cannot for a moment agree with the general assumption that this change always indicates involution and degeneration. We have observed this change in other bacilli, e.g. bacillus filamentosus, bacillus tuberculosis, bacillus of diphtheria, and bacillus of cholera, under conditions and at periods in which an involution and degeneration is not probable. On the contrary, we are inclined to think that these changes mean a reversion to a former type—atavism, in fact—from which the bacilli at an early phase of the life-history of the species have sprung; so that the typical bacilli, as we generally know and see them, are merely one form set in permanency, as it were, while the conditions of growth remain of a particular kind, e.g. in the animal body they always remain bacilli. But under altered conditions, e.g. in cultivations, many of the bacilli, though the single bacilli and their threads are of the typical kind, while the cultures are recent, soon revert or at any rate have a tendency to revert to the atavistic forms.

We have in the foregoing described the peculiar characters of the bacillus anthracis in microscopic specimens and in cultures; now, though they are peculiar and characteristic, they are nevertheless not exclusive, for there exist bacilli which resemble them in a marked degree, e.g. the bacillus filamentosus repeatedly mentioned; though this latter grows considerably quicker than, and is different in streak culture from, the bacillus anthracis, yet a mistake between the two is not inexcusable, owing to the similarity of the appearances presented by both in gelatine plates and in gelatine stab cultures; morphologically, i.e. under the microscope in the fresh or dried and stained specimens, they are quite indistinguishable. Hueppe also describes a bacillus obtained and isolated from earth which in morphological and cultural respects bears a very close resemblance to the bacillus anthracis; but neither the bacillus filamentosus nor the bacillus of Hueppe inoculated into animals produces anthrax. We have above mentioned certain fatal acute forms of human septicæmia in man (Bordoni Uffreduzzi) in which the proteus hominis was found in the blood and the organs. Here a mistake for anthrax is not easily made if cultures with the bacilli are instituted; but if from the pathological appearances at the *post-mortem* examination and from the presence of the bacilli in the blood and spleen alone, the diagnosis be made, a mistake, i.e. for anthrax, is also here possible. As regards animals, the cases are not at all rare where such a mistake has actually occurred, e.g. in sheep, cattle, pigs, dying under symptoms and pathological appearances resembling anthrax. Take, for instance, malignant

oedema, symptomatic charbon, and possibly other diseases : the appearances at the *post-mortem* may greatly resemble those of anthrax, the bacilli resembling to a certain extent, in size and general appearance under the microscope, those of anthrax ; the diagnosis by culture would, no doubt, throw great light on the nature of the bacilli, but a simple and decisive—in fact, the best and only reliable—diagnostic means is inoculation of mice or guineapigs with the blood or spleen of the questionable case. The appearances in the mouse and guineapig dead of anthrax are very characteristic, the spleen being dark and many times enlarged, the blood of the general circulation and of the spleen being crowded with the bacilli ; cultures from the blood of these experimental animals and further inoculations of mice and guineapigs can now be easily made, and the diagnosis fully established. The writer remembers the death of two sheep having been brought to his notice, where it was important, for taking preventive measures, to decide at once whether or not the sheep had died of anthrax. The *post-mortem* appearances were certainly very like those of anthrax, but there were only a few bacilli found in the blood. The spleen was enlarged, but not to a very conspicuous extent : it was dark, the lungs were congested and inflamed, in the juice of the lung and spleen numerous bacilli were found, and in sections through the lungs many capillaries were almost filled with the bacilli. In stained cover-glass specimens, and in stained sections, it was extremely difficult to say the bacilli were not anthrax bacilli. The question was, however, soon settled. Several mice and several guineapigs were inoculated, each with several drops of the blood of the spleen of the sheep ; none of these animals became in the slightest degree affected. From this of course there was justification for saying that those sheep did not die of anthrax, and the diagnosis was fully confirmed by the fact that, though no preventive measures of any kind had been taken on the farm on which those two sheep had died, no further cases of illness or death occurred either amongst the sheep or cattle, of which a considerable number were kept on the farm.

In all cases of doubt as to anthrax, inoculation of mice and guineapigs, particularly the latter, with a few drops of the blood of the spleen is the quickest and surest means of diagnosis ; if in these inoculated guineapigs typical anthrax with the typical bacilli (shown by cover-glass specimens and cultivation) in the blood and enlarged spleen results, then the diagnosis is clear ; if this result does not follow, no diagnosis of anthrax can be made safely.

Buchner maintained that by successive cultivation in broth he succeeded in changing the bacillus anthracis, virulent at starting, into an inactive bacillus in morphological respects identical with the motile hay bacillus ; and further that he was able by successive cultivations in certain broth cultures to change the motile hay bacillus, inactive on animals, into a non-motile bacillus, identical in morphological respects with the bacillus anthracis, and capable of producing typical anthrax in mice. These assertions have been proved to be erroneous, and Koch ('Mittheil. aus d. k. Gesundheitsamte,' I.) and the writer (Report of the Medical Officer of the Local Government Board for 1881) have shown the probable sources of these errors. All observers agree in this, that, through however many generations the bacillus anthracis be cultivated in broth, in gelatine, in serum, in agar, on potato, it always remains bacillus anthracis morphologically and physiologically. It may, owing to peculiar soil and other conditions of culture, diminish in virulence, and even lose its virulence altogether, as will be presently shown, but it nevertheless always remains morphologically the bacillus anthracis ;

but bacillus anthracis under ordinary conditions of cultivation, such as used by Buchner, does not lose its virulence.

Hueppe, as mentioned above, isolated from earth a bacillus which in morphological respects coincided with the bacillus anthracis; inoculated into mice it did not produce any disease, but, strange to say, protected the mice against inoculation with virulent anthrax. Hueppe concludes from this that his earth bacillus is real anthrax bacillus, which in course of time, and owing to its habitat and perhaps other conditions, has lost its power to produce anthrax, but nevertheless, though not apparent, has retained some kind of chemical action like anthrax. It is difficult to judge of and criticise these results; but without altogether declaring them improbable, this much can be said, that very clear evidence will have to be brought forward to support such assertions, which at present are not, it seems, forthcoming. *A priori*, it seems improbable that in the soil the true active bacillus anthracis should change into a non-active bacillus, or *vice versa*, seeing that the bacillus anthracis can thrive well in the soil and remain virulent for generations (e.g. the countries in which anthrax is endemic and in which infection is carried out by the soil). Further it is extremely improbable that a bacillus not causing any diseased condition should be capable of protecting against virulent anthrax; and lastly there is reason to doubt whether in mice protection against virulent anthrax can be at all or at any rate so easily produced as would seem from Hueppe's assertion.

The first observations that bacillus anthracis can become attenuated in its action without losing its morphological and biological characters were recorded by Toussaint, who found that heating anthrax blood up to 55° C. for a few minutes incapacitates such blood from producing anthrax on inoculation. Chauveau then found that the same attenuation and destruction of virulence occur when the virulent bacillus anthracis, e.g. the blood, is subjected to the action of 5 per cent. carbolic acid for a few minutes. But Pasteur was the first who showed that when bacillus anthracis is cultivated in broth at a high temperature (42·5° C.) it gradually loses its full virulence, and when such cultures are inoculated into sheep and cattle a mild and transitory form of anthrax is produced; animals so treated withstand successfully the further inoculation of virulent materials: they are therefore protected by the inoculation with the attenuated cultures. Pasteur has shown by a large number of experiments carried out in France and elsewhere that, by inoculation of such attenuated cultures, protective inoculation can be effected on sheep and cattle. He used two kinds of culture, for protective inoculation: (a) first vaccine: this is a culture of anthrax bacillus in chicken broth kept at 42·5° C. for fourteen days; when inoculated into sheep or cattle it produces only a slight local tumour. After about twelve days the animals are inoculated with (b) second vaccine: this is chicken broth culture kept at 42·5° C. for a week only. This culture produces also a local effect with slight constitutional disturbance, more pronounced than after the inoculation of the first vaccine; but the disturbance is only transitory and the animals recover. Up to nine months, such animals are refractory against inoculation with virulent anthrax blood.

If the second vaccine is used for the primary inoculation, the effect is more severe, and may lead to fatal general anthrax; this second vaccine having been grown for one week only at 42·5° C. is therefore stronger, and is of a higher degree of virulence than the first vaccine, which had been grown at the high temperature for a fortnight. Though theoretically correct, and fully confirmed by numerous experiments, there are several difficulties in the way of the practical application of this protective inoculation: (1) Since it is a

question of making such vaccine on a large scale—and this must be of course the case if it be used in practice, e.g. for whole flocks of sheep and herds of cattle—the exact method and way of getting the right degree of virulence is not easily achieved. Some years ago (1882, 1888) the writer bought some Pasteur's vaccine made in Pasteur's laboratory, and sold in tubes by Pasteur's agent, M. Boutroux, and it was used on guineapigs and sheep. Of the guineapigs inoculated with first vaccine a certain percentage died of typical anthrax; with second vaccine the percentage of death was greater. Of the sheep that had been inoculated exactly after Pasteur's prescribed method, one of four died with anthrax from the second inoculation, i.e. with second vaccine; of five sheep treated with the two Pasteur's vaccines, and afterwards subjected to inoculation with virulent anthrax blood, one died after two days, a second one after a week, and three proved refractory, i.e. were protected. Such or similar results were recorded also by other experimenters. (2) The vaccine manufactured thus by Pasteur on a large scale is not always pure anthrax; there are present other organisms besides anthrax bacilli. Vaccine thus contaminated may therefore harbour bacilli—e.g. those of malignant cedema—which are not desired, since these themselves may produce in sheep and cattle undesired results. But it ought to be in fairness stated that the published statistics on the greatly decreased mortality amongst sheep and cattle, since Pasteur's protective inoculations have been used in the localities in which great devastations by endemic anthrax (France, Russia) had previously occurred, bear out Pasteur's contention as to the practical use of the protective inoculation with his anthrax vaccine. (8) A third serious difficulty is this: Koch and Gaffky have shown that animals (sheep) protected by Pasteur's vaccines against further *inoculation* with virulent anthrax prove unprotected, or almost wholly so, if they are infected with spores by feeding; for by this method Koch and Gaffky have shown that such sheep become infected with typical anthrax; and it is precisely this method of infection, viz. by spores taken in by the alimentary canal, that obtains amongst sheep and cattle under natural conditions, as has been mentioned on a former page. Other modes of protective inoculation have been recorded by Perroncito, Kitt, and others. The writer has shown (Report of the Medical Officer of the Local Government Board, 1882) that mouse's anthrax blood is attenuated anthrax, and can be used for protective inoculation of sheep; guineapig's anthrax blood mixed for a few minutes with a large quantity of solution of perchloride of mercury, of the strength of 1 : 25,000, is sufficiently attenuated for sheep (*ibid.* 1885). It produces a slight illness and protects against further inoculation with virulent material.

Wooldridge ('Proc. Royal Society,' 1887) has shown that in cultures of bacillus anthracis—in a particular alkaline albumen—chemical substances are present which have a powerful protective influence, inasmuch as when injected into the vascular system of rabbits they provide at once protection against inoculation with virulent anthrax. Hankin then showed that in certain proteid material the bacilli anthracis produced a poisonous albumose, to which the pathogenic action of the bacilli is due; and Sidney Martin further proved that these albumoses give further origin to a 'poisonous alkaloid,' which he isolated, and obtained as a salt in a crystalline form.

The writer has shown that anthrax bacilli, derived from virulent disease (blood of a guineapig dead of typical anthrax), when grown in gelatine medicated by the admixture of perchloride of mercury, 1 : 40,000, suffer an attenuation of their virulence; though the growth is apparently normal, and when transferred to new normal nutrient gelatine yields normal typical

anthrax growth : nevertheless it either does not produce death at all, or only after many days.

This mode of producing attenuated anthrax depends, however, to a large extent on the primary degree of virulence of the bacilli at starting. While anthrax bacilli which possessed at starting a high degree of virulence suffer slight attenuation by this method, others not of a high degree of virulence at starting are easily attenuated in their virulence by this method (*see Reports to the Medical Officer of the Local Government Board, 1885-86*).

While sheep, cattle, and rabbits that have once passed through a mild form of anthrax are, as a rule, refractory against further inoculation with virulent anthrax, this is not the case with some other animals, for instance, guinea-pigs. Of these the writer has shown that only in a very small percentage (less than 4 per cent.) can a real protective inoculation be effected ; the rest, though sometimes protected against one, two, or even three successive inoculations with less virulent anthrax, eventually succumb to virulent degrees of anthrax (*Report of the Medical Officer of the Local Government Board for 1887, p. 291*).

As mentioned in a former chapter, Ogata and Jasuhara have shown that a previous or simultaneous injection of the blood of an insusceptible animal (rat, dog) protects from, and neutralises the action of virulent anthrax in mice. Hankin obtained a glycerine extract from the spleen of a rat, which also protected mice against anthrax. Normal frogs and rats, insusceptible to anthrax, become susceptible to it if previous to, or simultaneous with, or immediately after, inoculation with anthrax blood or anthrax culture (spores or bacilli) they are subjected to narcosis with ether-chloroform.

The fact that frogs kept at the temperature of a warm-blooded animal are hereby rendered susceptible to anthrax (*Petruschki*) has been mentioned in a former chapter.

The disease of silkworms known as *pébrine*, and showing itself in minute grey or black patches all over the body of the silkworms, has been shown experimentally to be contagious, and generally communicated to the worms by food (mulberry leaves) which had been previously tainted with the contagium. This latter, known as '*Cornalia corpuscles*,' first discovered by *Cornalia*, is composed of oval microscopic corpuscles which by some are declared to be oval cocci, hence also termed *micrococcus ovatus* ; *Lebert*, *Naegeli*, and others have shown them to be the living contagium of the disease. These corpuscles enter by the alimentary canal of the worms, multiply there enormously, and spread thence into all the tissues ; they are transmitted by the moth to the eggs, multiplying within the body of the chrysalis, and reaching their maximum development in the worm, in which the disease declares itself as *pébrine*. Pasteur has shown experimentally that the time at which the silkworm, during its existence as worm, becomes infected has a very important bearing on the period at which the disease shows itself in the worm, moth, or chrysalis respectively.

The *Cornalia* corpuscles, also called *nosema bombycis*, are by some observers, amongst them *Naegeli*, declared to be a form of bacteria ; others, like Pasteur, *Balbani*, and *Leydig*, declare them to be *psorospermia*. Pfeiffer has studied them carefully and extensively, and has shown that they are a species of sporozoa (*see his paper in 'Zeitschr. f. Hygiene, III., p. 469*).

Another infectious fatal disorder of silkworms is the one known as *flacherie* : it is not associated with the cutaneous spots of the *pébrine*, but the worms affected with the disease show a typical limpness and relaxation, become very weak, and ultimately die. The corpuscles that are present in the worms, and believed to be the cause of the disease, are called *microzyma bombycis* of *Béchamp* ; they are oval cocci, forming longer or shorter chains ; hence are also called *streptococcus bombycis*. They have been found by *Cohn* in enormous numbers in the intestinal canal of the affected worms. By mixing with the mulberry leaves the intestinal contents of affected worms, and feeding thereon healthy worms, these latter contract the disease, and their alimentary canal, previously free of the streptococci, becomes now full of them.

Foulbrood of Bees.—A fatal infectious disease of bees has been investigated by *F. R.*

Cheshire and Watson Cheyne, and has been shown by the latter to be caused by a slightly motile bacillus, the *bacillus alvei*. The single bacilli measure 2.5–5 μ in length, 0.8 μ in thickness, forming in cultures longer or shorter threads, and in these oval spores, 2 μ long, 1 μ thick, make their appearance. The growth of the bacilli in gelatine plate cultivations is very characteristic: in their first appearance the colonies are round or oval discs, next they become pea-shaped, and more or less drawn out at one or more points into longer or shorter processes: these become branched. Under the microscope the branches are straight or circular, or possess irregular thickenings; the same projections are seen to arise in the streak cultures as well as stab cultures in gelatine; the branches, projections, and filamentous processes are seen under the microscope to be bundles of bacillary chains or filaments. That this bacillus is really the cause of the fatal disease has been proved experimentally by Cheshire and Watson Cheyne by feeding healthy bees with spores, or bacilli and spores, of the artificial cultures. ('Journal of the Royal Micr. Soc.' March 1885.)

CHAPTER XVI

EPIDEMIC PNEUMONIA—INFLUENZA

By the researches of Leyden and others it has been shown that the disease known as lobar or croupous or fibrinous pneumonia belongs to the group of infectious diseases. It has this in common with infectious diseases, that it occurs simultaneously in a number of persons living under the same conditions of place and surroundings; that it runs a definite course; that its symptoms and pathology are the same or very much alike in all cases; and last, but not least, that in several instances the disease has been communicated within the same household from one person to another. Only during recent years has bacteriological investigation shown that not all acute croupous pneumonias are of exactly the same kind. Though in all there is the viscid gelatinous sputum, more or less rusty or prune-juice-coloured during the first three or four or five days, giving way to a less viscid, more greyish, more purulent expectoration, and though in all of them there is an early or red solidification or hepatisation, leading gradually to a more grey infiltration (grey hepatisation) of one or more lobes of one or both lungs, associated with more or less extensive pleuritis: yet there are certain differences of detail which seem to show that croupous or epidemic pneumonia is a name which ought to designate more than one species of acute infectious pneumonia.

In the genuine croupous pneumonia, the lobar fibrinous pneumonia generally involves but one lung; the sputum is, as is well known, of a characteristic gelatinous consistency; it cannot be poured out of a vessel, since it forms a very tenacious viscid material; it is of a gelatinous transparency, contains a number of air bubbles, and in the early stages is scanty. During the first few days (three to five days) it is streaked with rusty or reddish streaks and patches, which colour is due to the presence of red blood-corpuscles; the sputum is transparent and contains under the microscope red blood-corpuscles, a few leucocytes and swollen alveolar epithelial cells; fibrin threads are seen to permeate the whole. This condition is, however, only observed during the first few days; after the fifth or sixth day the sputum becomes more copious, less rusty, and more grey; under the microscope few red blood-corpuscles, but crowds of leucocytes, are seen and few threads of fibrin; and when the disease has passed its height (sixth to ninth day) the sputum becomes semi-fluid and greyish white, and under the microscope appears almost uniformly made up of leucocytes.

On *post-mortem* examination of cases dying during the first four or five

days, the most prominent symptom is the pneumonia; generally one lung, rarely both, shows in one or more lobes well-marked-off intense inflammation, the tissue being in a solid state, intensely congested, dark purple red—red hepatisation. When an incision is made, copious almost fluid blood oozes out of the cut surface; pieces of such a lobe sink in water. The pleura is opaque and injected, and its cavity contains in some cases more, in others less, sanguineous serous exudation: the right heart contains clotted blood. In cases that die after the seventh to ninth or tenth day the inflamed lobes are less red than in the former stages, but solid, and portions of them sink in water; on incision the surface is less juicy, is grey—grey hepatisation. Congestion of the liver, spleen, and the cortex of the kidney, and inflammation of the pleura and pericardium, with serous and fibrinous exudation are occasionally present, particularly of the pleura, which latter condition is generally observed at the *post-mortem* of early cases, i.e. those that die in the stage of red hepatisation. Ecchymoses into the mucous membrane of the stomach and intestine are sometimes observed in such cases.

On microscopic examination of the red hepatised lung, one finds all the blood-vessels of the inflamed lobes intensely congested, the blood-vessels, including the capillaries around the air cells, engorged with blood; the alveolar cavities, infundibula, and minute bronchi are distended by, and filled with a dense network of fibrin threads, in whose meshes are situated numerous red blood-corpuscles and a few leucocytes, isolated or in groups; the epithelial cells lining the alveoli are swollen and detached from the wall, and are contained within the fibrinous mass. The substance of these epithelial cells is granular and contains vacuoles; in the alveolar wall, and in the peribronchial and interlobar connective tissue, blood is present *en masse*. Around the branches of the pulmonary artery, and around the small and large bronchi, are found numerous leucocytes. In the stage of grey hepatisation the air cells and infundibula and small bronchi are filled with a network of fibrin, and in their meshes very numerous leucocytes, a few red blood-corpuscles, and a few epithelial cells are discernible; in this stage leucocytes are found also in the alveolar walls and in the peribronchial tissue (figs. 83 and 84). Examined under the microscope the liver appears congested; the liver cells are slightly swollen and granular, and in most lobules many of the cells contain fat globules, i.e. show fatty degeneration.

In the kidney the epithelium of the convoluted uriniferous tubules shows opaque swelling and granular degeneration.

The first statements that a definite microbe is present in the sputum and in the exudation, contained in the air cells, were made by Friedländer. Though Klebs, Koch, and others had previously described cocci as occurring in the exudation, Friedländer¹ and Frobenius first demonstrated by the culture test the presence of a definite species of *capsulated oval cocci*, called by them pneumococci, but now known as the *bacillus of Friedländer*, since it has been shown that the coccus is only a phase in the growth and multiplication of the microbe, and that this is really a rod-shaped organism, a bacillus. It occurs as oval cocci, as single rods, as chains of oval cocci or rods. In the sputum and in the exudation of the lung it is characterised by a hyaline capsule, each coccus or rod when isolated being surrounded by it, or it forms a common capsule round a dumb-bell of the oval or rod-shaped elements; in cover-glass specimens of the sputum or lung exudation the capsules can be stained in a different colour from the rods, either by over-staining the whole, e.g. with carbol-methyl blue or Weigert's gentian violet,

¹ *Fort. d. Med.* vol. i. 1883, No. 22.

and then well washing with spirit. By this means the capsule remains faintly stained while the cocci or rods retain a deep colour : or by Gramm's method—first stain in gentian violet, then in eosin ; or by double staining first with methyl blue or gentian violet and then with eosin. In sections through the hardened lung, the capsulated rods and cocci can be shown by well staining with carbol fuchsin (half to two hours), then washing in alcohol for some time (half to one hour).

This bacillus of Friedländer, when it occurs, appears in the stage of red hepatisation, and disappears during the further stage. It shows the following characters in cultivation, whether derived from the sputum or the lung juice : in stab cultures in nutritive gelatine it forms a whitish line, which gradually thickens, and which, on the surface of the stab, forms a convex, rounded, greyish, or greyish-pink knob, the culture thus resembling a ' nail,' in which the linear growth in the stab is the nail pin, the convex knob at the upper end of the stab the nail head. In plate cultivation it forms small round greyish-white colonies, those situated on the surface of the gelatine being convex. The bacilli and cocci of the cultures have no distinct capsules.

Friedländer and Frobenius and other observers afterwards tried to show the pathogenic action of this microbe on rodents—mice and guineapigs. They did not succeed in producing any pathogenic action by injecting the cultures subcutaneously, but on inoculation into the pleural cavity and the lung tissue (by means of a Pravaz syringe, the cannula of which pierces the wall of the chest) those observers produced definite results : pleuritis and pneumonia, and in the pleural exudation and inflamed lung the capsulated bacilli were found in great numbers. The writer has in 1883 (*see* Report of the Medical Officer of the Local Government Board for 1884) shown that by subcutaneous injection of sputum of croupous pneumonia (from the stage of red hepatisation) into rabbits and mice, acute septicæmia is sometimes produced ; in the blood, pleural and pericardial exudation, and in the congested lung of such animals the capsulated bacillus of Friedländer can be demonstrated in large numbers. The writer has in that paper taken exception to the assumption made by Friedländer as to the true pathogenic nature of the so-called pneumococcus of Friedländer ; that is to say, from the inconstancy, scarcity, and unequal distribution in the human lung of this microbe, and owing to the septicæmic action on mice and guineapigs, and to the method of experimentation used by Friedländer and others in order to demonstrate that this microbe is the true pneumonia microbe, the writer has differed from the proposition that the bacillus of Friedländer is the microbe of croupous pneumonia. Passet has shown that a capsulated microbe in every respect identical with Friedländer's pneumonia bacillus occurs elsewhere and independently of croupous pneumonia—is, in fact, probably a normal inhabitant of the fluid of the mouth and throat, and is present in the normal sputum. A. Fränkel,¹ and independently at the same time Weichselbaum,² have by a very large series of observations, made on very numerous cases of genuine lobar or croupous pneumonia in the stage of red hepatisation, shown that the bacillus of Friedländer occurs only in a small percentage of cases ; but that almost invariably there is present in large numbers, both in the sputum and the lung juice, an oval capsulated coccus, and they justly conclude that Friedländer's bacillus cannot be causally connected with pneumonia.

The coccus which Fränkel and Weichselbaum independently discovered is capsulated, and occurs as a rule as a diplococcus ; occasionally the

¹ *Deutsche med. Wochenschrift*, 1885, No. 81.

² *Wiener med. Jahrbücher*, 1886, p. 483.



diplococcus is arranged in shorter or longer chains. This diplococcus does not grow on gelatine at 20° C.; it requires higher temperature, at least 22° to 24° C., i.e. it cannot grow at ordinary temperature, but nutrient agar or blood serum has to be employed. In agar plates or serum plates it forms rounded white colonies; on the surface of the agar or serum, in streak culture, it grows as a whitish film; on potato the growth is thin and white, dry and difficult to see. Recent cultures of the microbe inoculated subcutaneously, or better still into the peritoneal cavity, produce in rabbits and mice a general septicæmic infection; cultures carried on for several generations gradually lose all virulence. Owing to the fact that this diplococcus pneumoniae of Fränkel and Weichselbaum has been found by them and by others as a constant and numerous inhabitant of the sputum and lung juice of true croupous pneumonia in the acute stage, it is considered as the real microbe of this pneumonia. These facts have hitherto been confirmed by a large number of independent observers.

Notwithstanding the very extended observations of Fränkel, Weichselbaum, and of many others in relation to the diplococcus pneumoniae and its distribution, it is perfectly justifiable to question whether this is the microbe of all cases of croupous pneumonia, and whether it is the only essential etiological factor in the croupous pneumonia. Croupous pneumonia is in its pathology and clinical aspects a well-defined disease, and it is curious to find that the identical microbe should also be the cause of such widely different diseases as acute purulent otitis, cerebro-spinal meningitis, and acute peritonitis (*see below*). Further, Weichselbaum ('Wiener med. Blätter,' XIII., No. 6), examining the sputum derived from twenty-one patients affected with bronchitis after influenza, found the diplococcus pneumoniae constantly present in considerable numbers, yet only in two or three cases did croupous pneumonia actually supervene; again, Besser (Ziegler's 'Beiträge zur path. Anat.,' Band VI., 4) found in the normal secretion of the nasal cavity in fourteen out of eighty-one cases, the presence of the diplococcus pneumoniae in many instances so numerous as to form a pure culture. He further met with the diplococcus pneumoniae in the bronchial mucus of cases of fracture of cervical vertebrae, in one case of tuberculosis of the peritoneum and in one of enteric fever; and lastly Neumann ('Centralb. für Bacteriologie und Parasit.,' VII., No. 6) points out that the micrococcus pyogenes tenuis of Rosenbach, which has been described by Rosenbach and others in connection with various kinds of purulent processes (empyema, abscess on the neck, on the leg, and in the processus mastoideus, &c.), and considered to be one of the species of microbes capable of causing suppurative changes, is identical with the diplococcus pneumoniae. All these facts would point to the conclusion that a particular condition of the lung is essential for the diplococcus to cause the pneumonia.

Finkler in an exhaustive treatise on Acute Pneumonia ('Die acuten Lungenentzündungen als Infections-Krankheiten,' Wiesbaden, 1891), summarises, p. 286, his own observations and those of others thus: (1) Fibrinous pneumonias are caused particularly by the diplococcus pneumoniae; (2) broncho-pneumonias are bacteriologically not a single disease; (3) cellular pneumonias are caused chiefly by streptococci.

There have occurred cases of pneumonia in batches and in epidemics in which a direct infection from person to person has been demonstrated, pneumonias which in many respects resemble the ordinary croupous pneumonia: the symptoms are similar and the course is only slightly different; the sputum is of the same viscous consistency but less prune-juice-coloured, more grey; in the later stages the sputum is whitish grey and more fluid.

Under the microscope few red blood-corpuscles, but numerous leucocytes, are seen in the early stages, but otherwise the sputum looks very much the same as in the above croupous pneumonia; in the later stages it is also crowded with leucocytes. The lung shows also a stage of red and a stage of grey hepatisation, the latter gradually following the former. The naked eye appearances of the lung differ very little from the true croupous pneumonia, except that the demarcation of the inflamed lobes from those not inflamed is not marked, that the pneumonia is generally double-sided, and that the lung is more flaccid; sections of the lung in the stage of red hepatisation examined under the microscope show only a few red blood-corpuscles in the exudation of the air cells, but very numerous leucocytes and a few detached epithelial cells in a network of fibrin. In this respect there exists a difference between this kind and the true croupous pneumonia in the stage of red hepatisation; in the latter the network of fibrin is more copious, it contains few leucocytes and chiefly red blood-corpuscles; whereas in the pneumonia under consideration, even in the earliest stages, the alveolar cavities already contain numerous leucocytes—in fact, appear crowded with them. Now in these pneumonias neither the bacillus of Friedländer nor the diplococcus of Fränkel and Weichselbaum can be demonstrated. The cases of infective pneumonias of this kind that the writer has seen were those investigated by Dr. Ballard in Middlesbrough, and described by him in the Report of the Medical Officer of the Local Government Board, 1889. The pathology and clinical history of these cases have been described by Dr. Ballard, and on comparing them with what is known of the ordinary croupous pneumonia, there are only slight differences to be noticed.

Cover-glass specimens made from the sputum or the bloody juice of the lung show bacilli in large numbers; they are neither Friedländer's nor are they the capsulated diplococcus of Fränkel-Weichselbaum occurring in genuine croupous pneumonia; these bacilli are present in the juice of the inflamed lungs in pure culture. They are short, oval rods, some are constricted in the centre, they occur singly or more commonly as dumb-bells, or also, but rarely, as short chains, but there are always rods amongst them which are longer and more cylindrical. Cultivations made on the surface of nutrient gelatine (in plate or in test tubes) with such lung juice bring forth pure cultures of the microbe; and it is hereby seen by the large number of colonies that come up, if even only a trace of the lung juice be used, that those bacilli must be present in the lung in very large numbers. In preparations made of such colonies many of the microbes are short and oval rods, but there are now a good many longer bacilli visible, and after several days' growth even long filaments made up of cylindrical bacilli can be found. The size of the bacilli prepared from the sputum or the lung juice or from cultures is this: the thickness 0.8 to 0.4 μ , length 0.8 to 1.8 μ and more. The bacilli in the fresh state are motile, though these are only few in number; a good many individuals do not show it. In cultivations the motile forms occur only if the culture is on the surface, e.g. on gelatine, on agar, or potato, and in these only while the culture is of recent date; in broth after two days' growth at 37° C. motile forms are missed. The motility consists in the bacilli darting through the field of the microscope or spinning round very rapidly. These facts as to the bacilli showing motility only when growing on the surface of the nutrient medium show that a free supply of oxygen is required to maintain such motility. But the great requirement of oxygen during the growth and multiplication of the bacilli, i.e. that they are *aërobic*, is also shown by the fact that they do not grow well and rapidly in the depth, though they grow and multiply

with enormous rapidity on the surface of the nutritive media. When growing in a gelatine plate cultivation, those colonies that develop on the surface grow incomparably faster than those which develop in the depth ; while the former in a few days reach the breadth of several millimetres, the latter at the same distance of time are only minute points. The colonies when first appearing (twenty-four to forty-eight hours) on the gelatine are minute grey round dots ; soon they spread out into translucent plaques, of which the margin is thinner than the centre, being at the same time irregularly crenated ; under a glass the colonies are uniform and light brownish in colour. In about ten to fourteen days the diameter of the plaques reaches as much as half an inch. The nutrient gelatine does not become liquefied at any time. In streak culture on gelatine the streak is already after two to three days occupied by a translucent greyish white band, 2-3 mm. wide, thicker in the middle than in the margin, which latter is very irregular and crenate. In the stab culture in gelatine the stab becomes indicated by a thin grey line made up of minute grey droplets, the size of which always remains small, whereas on the upper or free end of the stab there is present a thin greyish translucent patch, rapidly expanding and of irregular and crenate outline. On agar mixture at 36-37° C. the growth forms in a few days a thin whitish-brown pasty pellicle ; in alkaline broth at 37° C. the bacilli grow very rapidly, after twenty-four hours producing strong turbidity, after two to three days yielding a whitish floccular sediment ; no real pellicle is formed on the surface of the broth. On potato at 37° C. the growth after a few days is a light brown-coloured, pasty, moist layer.

The cultures as also the lung juice act virulently on mice and guineapigs, on the former more than on the latter. Subcutaneous inoculation produces disease and death in the course of thirty to 100 hours. On *post-mortem* examination both lungs are found intensely inflamed, some portions in a state of red hepatisation ; generally there is present pleurisy and pericarditis and peritonitis, with more or less sanguineous exudation. The spleen is enlarged in mice, but not in guineapigs. The bacilli can be easily demonstrated in very large numbers both by cover-glass specimens and by cultures in the heart's blood, the lung juice, and the spleen of the mice, and in the lung juice of the guineapigs.

The lung juice, or cultures derived from the tissues of the infected mice or guineapigs, inoculated into further mice or guineapigs, produce the same disease and death with the symptoms just described.

While working with cultures of these bacilli on mice and guineapigs there occurred amongst normal mice and guineapigs, kept in the same stalls as the experimental animals, an epidemic of pneumonias, leading to death of a great many of them ; on *post-mortem* examination all showed exactly the same appearances as the above experimental mice and guineapigs, and the juice of the inflamed lungs contained the same bacilli in crowds.

Three monkeys, kept on the same premises and which most probably became accidentally infected by food, died of pneumonia. In the inflamed lungs the bacilli could be easily demonstrated by cover-glass specimens and by culture.

It may be added here that in two out of five cases of croupous pneumonia following after influenza, the writer has found the same bacillus in large numbers, almost in pure culture ; its cultural characters and its pathogenic action on mice were the same as in the Middlesbrough cases.

Amongst a number of pneumonias not connected with influenza, the same bacillus has been demonstrated in a small percentage of the cases, but in these the bacilli were present in pure culture.

Foà ('Deutsche mediz. Wochenschrift,' 1889, No. 2) describes cases of croupous pneumonia which were neither associated with the bacillus of Friedländer nor with the diplococcus of Fränkel-Weichselbaum. In the sputum, in the fluid withdrawn from the inflamed lung during life by means of a syringe, and in the hepatised lung after death, he met with a micrococcus, which, owing to its shape and being capsulated, he called micrococcus lanceolatus capsulatus. This by its morphological and biological characters was recognised as a definite and separate species. Cultures of it in broth could easily be made, also on agar and on blood serum; it did not grow on potato or on gelatine. On mice, guineapigs, and rabbits it acts virulently, on the latter always if inoculated in large quantities; the animals dying in two to three days. Injected into the peritoneal cavity, it produces peritonitis with serous fibrinous exudation; subcutaneously, it either produces rapid septicæmia and death, or a severe local œdema and death.

Mosler ('Deutsche mediz. Wochenschrift,' 1889, Nos. 18 and 14) describes cases of croupous pneumonia occurring in one family, and clearly communicated directly from one member to the other. On *post-mortem* examination both lungs were found affected with fibrinous pneumonia, the character of the hepatisation being of a hæmorrhagic kind, and there was also hæmorrhagic fibrinous pleuritis. No pneumococci could be found in the sputum or lung. Löffler examined the lung fluid withdrawn by a Pravaz syringe from the living patient, and in it he found very numerous examples of bacteria, belonging to one single species of bacilli which differed from Friedländer's bacillus, and from Fränkel's and Weichselbaum's diplococcus pneumoniæ. From the description of the characters in cover-glass specimens, and on agar and gelatine, the writer finds a very great resemblance to the bacilli described by himself of the Middlesbrough croupous pneumonia. However, no motility of the bacilli was noticed; but this need not surprise, since striking motility of the Middlesbrough bacillus is noticed only in recent cultures; after two to three or more days the bacilli do not show any conspicuous motility. Mosler did not succeed in producing any disease in mice, guineapigs, or rabbits, nor were the bacilli found in the lung at the *post-mortem* examination.

Various acute inflammatory diseases are associated with, and ascribed to the diplococcus pneumoniæ of Fränkel and Weichselbaum. Weichselbaum describes ('Wiener klin. Wochenschrift,' 1888, Nos. 28-32) several cases of acute meningitis: three cases of cerebro-spinal meningitis pure, one case of acute internal hydrocephalus and spinal meningitis, one case of acute encephalitis and circumscribed cerebro-spinal meningitis, one case of pneumonia of long standing, there being present at the same time ulcerative endocarditis of the aortic valves, embolism of the right arteria fossæ sylvii, and circumscribed meningo-encephalitis. In all these cases the diplococcus pneumoniæ could be demonstrated in the exudation by cover-glass specimens and cultures. In the cases of primary cerebro-spinal meningitis and meningo-encephalitis Weichselbaum constantly found exudations in the tympanic cavity and the secondary nasal cavities, and in four such cases he found in the exudations of the tympanic cavities, maxillary cavity, and the ethmoidal labyrinth an inflammatory exudation which contained the diplococcus pneumoniæ. From this Weichselbaum concludes as probable that this microbe has found its entrance into the cranium from those cavities and set up the meningitis. Weichselbaum found also, at the *post-mortem* examination of cases of croupous pneumonia, exudations in the tympanic and secondary cavities of the nose, and of the ethmoidal labyrinth, which contained the diplococcus pneumoniæ; in one case of croupous pneumonia, dead on the

eighth day, he found that microbe even in the cavity of the sphenoid bone. In one case of primary pleurisy with slight pericarditis and puerperal endometritis, the diplococcus pneumoniae could be demonstrated in the pleural, pericardial, and uterine exudations. Banti (*ibid.*) describes cases of pericarditis, and also of pleuritis, associated with the diplococcus pneumoniae.

Foà and Bordoni Uffreduzzi likewise describe cases of epidemic cerebro-spinal meningitis associated with the diplococcus pneumoniae ('Zeitschr. f. Hygiene,' IV., 1, p. 67).

Bonone ('Centralbl. f. Bact. und Parasit.,' IV., No. 11, p. 821) describes a case of pleuro-pericarditis and cerebro-spinal meningitis in which he found, and from which he cultivated (from the fibrinous exudation of these membranes), a microbe which, though in many respects resembling the diplococcus pneumoniae, was, nevertheless, found to offer slight differences; for this reason Bonone calls it the pseudo-diplococcus pneumoniae.

Weichselbaum has further observed ('Centralbl. für Bact. und Parasit.,' 1888, Band V., No. 2) cases of primary acute peritonitis, followed by pleurisy, in the exudation of which the diplococcus pneumoniae was found in large numbers.

The same author ('Monatsschrift f. Ohrenheilk.,' 1888, Nos. 8, 9) describes a case of acute rhinitis and otitis media, followed by pneumonia of one lobe of the left lung and acute parenchymatous nephritis; in the exudation of the nasal cavity and in the pus of the tympanic cavity and mastoid cells, the bacillus of Friedländer was present in large numbers.

Levy and Schrader ('Archiv f. exper. Path. und Pharm.,' XXVI., 1889, and 'Centralb. f. Bact. und Parasit.,' VII., No. 8, p. 98) found amongst ten cases of otitis media thrice the diplococcus pneumoniae, thrice staphylococcus pyogenes albus, once streptococcus pyogenes, once diplococcus pneumoniae and streptococcus pyogenes, once diplococcus pneumoniae and staphylococcus albus, and once staphylococcus pyogenes albus and cereus albus.

Zaufal, on the other hand, mentions cases of genuine acute otitis media caused by the diplococcus pneumoniae ('Centralb. f. Bact. und Parasit.,' Band V., p. 617). Kanthack ('Zeitschr. f. Ohrenheilk.,' XXI., 1890, p. 44) found in the exudation of seven cases of otitis media the staphylococcus pyogenes albus, the bacillus saprogenes of Rosenbach, and the diplococcus pneumoniae.

INFLUENZA

Maximilian Jolles ('Wiener med. Blätter,' 1890, XIII., No. 4) asserts the constant presence of the capsulated bacillus of Friedländer in the sputum of persons who had passed through influenza, and is inclined to ascribe to it an etiological importance; but for this there is not sufficient ground, since this bacillus is present also in normal sputum, and further the pneumonia following influenza is sometimes of the character of a complication or secondary infection. With this accord Weichselbaum's observations (*ibid.* 1890, No. 6), who found in cases of pneumonia following influenza the diplococcus pneumoniae; but he found this same microbe abundantly in the sputum of other cases of influenza, twenty-one in all, though only in two of them croupous pneumonia supervened. Weichselbaum arrives at the conclusion that the diplococcus pneumoniae is not the microbe of influenza.

Ribbert and Finkler describe the streptococcus pyogenes as the cause of the pneumonia following influenza, and the first-named author thinks that microbe also the microbe of the influenza itself.

Professor Klebs, of Zürich ('Centralbl. für Bact. und Parasit.,' VII., 5) maintains that during the febrile stage in influenza the blood contains, 'in very large numbers, flagellate protozoa,' either free in the plasma, or adhering to or contained within the red blood discs. More recently Pfeiffer

('Deutsche med. Woch.' No. 2, 1892) described the constant presence in the bronchial sputum and in the pulmonary exudation in all cases of influenza, mild or severe, of minute bacilli, about half the length but the same thickness as the bacilli of mouse septicæmia; in stained specimens the bacilli show a characteristic bipolar granule with intermediate clear part, hence resemble a diplococcus. They are aggregated in smaller or larger clumps, occur in the leucocytes of the sputum, and also form short chains, which in stained specimens resemble streptococci. These bacilli disappear with the cessation of the disease. Kitisato (*ibid.*) cultivated the bacilli; they grow in a characteristic manner in broth and agar kept at 37° C. The broth remains clear, while a growth at the bottom of the fluid appears as whitish grey granules, and fluffy nebulous masses. On agar the growth forms minute translucent droplets which have no tendency to coalesce; the bacilli do not grow on nutrient gelatine kept at 20° to 22° C. In broth and in agar the bacilli form long threads (*see* fig. 6). Pfeiffer did not meet with the bacilli in any but influenza cases; Kitisato did not succeed in cultivating the bacilli from any but influenza sputum; Canon (*ibid.*) asserted the presence of bacilli, supposed to be morphologically the same as the above, in the blood of all cases of influenza, their number varying in cover-glass specimens between 5 and 20, occurring singly and in small groups. The writer had the opportunity of confirming Pfeiffer's and Kitisato's discovery as to the presence of the bacilli in the bronchial sputum of cases of influenza, as also, to a limited degree, the assertion of Canon as to the presence of certain bacilli in the blood ('Br. Med. Journal,' January 28, 1892). From an extensive series of observations carried out during February, March, and April, 1892, the writer has convinced himself that while Pfeiffer and Kitisato's assertions are correct, those of Canon cannot be upheld, since in over fifty cases of true uncomplicated influenza, in which the blood was examined (prolonged staining of cover-glass specimens after Canon's method with a mixture of eosin and methyl blue), in only two cases were bacilli present in appreciable numbers (*see* fig. 95), in a third case there were present but very few, and in the rest no microbes could be detected. Cultivations of the blood yielded, except in one case, no organisms. The bacilli present in the blood of the above three cases appeared larger than the influenza bacilli of the bronchial sputum.

Important observations on the different species of bacteria found in influenza are recorded by Babes, 'Centralbl. f. Bact. und Parasit.,' IX.; Friedrich, 'Arbeiten aus dem Kais. Gesundheitsamte,' VI., 2; Kirchner, 'Zeitschrift f. Hygiene,' IX., 8, p. 528; Bein, 'Centralbl. f. Bact. und Parasit.,' IX., No. 5; Fränkel, Fraser, Kartulis, and others, *ibid.*

As to pleuro-pneumonia of cattle, various species of microbes have been described as the cause of it, but none can be said to have been at all satisfactorily proved to be so (*see* the assertions of Lustig, Poels, and Nolen).

CHAPTER XVII

GONORRHOEA

IN this acute infection, be it of the conjunctiva, or of the urethra, or vagina, the inflammation is essentially of the character of a severe catarrh: the mucous membrane is congested, and its tissue is infiltrated with leucocytes; these migrate into the epithelium of the surface, and through this on to the

free surface. The lines occupied by the cement substance of the epithelial cells are at first the paths through which the leucocytes and exudation pass towards the surface; as these, with the progress of the disease, increase in number, they burrow also into the substance of the epithelial cells, numerous epithelial cells at the same time becoming swollen and detached. In the earlier stages the exudation passing from the surface of the mucous membrane is thin and more of a serous or thin purulent character; later on it becomes more of the character of thick pus. In the former we find numerous free epithelial cells and leucocytes; some of these epithelial cells contain vacuoles, and in them one or more leucocytes; occasionally one meets with a large epithelial cell containing in a cavity a number of leucocytes, the protoplasm of the epithelial cell being reduced to the periphery and including the compressed oval nucleus. Such epithelial cells with vesicles in which one or more leucocytes are contained, have been occasionally interpreted as epithelial cells in which pus cells are being formed by endogenous formation; but in sections through the epithelium it is clearly seen that leucocytes everywhere pass from the mucosa into the epithelium, and here they invade the epithelial cells themselves. In the later stages, when the exudation is of thicker consistency, the relative number of epithelial cells in the exudation is not so great as at first, but the number of pus cells is very great, and, indeed, causes the consistency of the exudation. Neisser was the first who pointed out the constant presence, in the exudation in gonorrhœa, of peculiar micrococci, which occur as dumb-bells and as masses of dumb-bells either free in the serum, or frequently within the protoplasm of the pus cells, or adhering in smaller or larger numbers to the epithelial cells: these cocci he called gonococci (fig. 96). The length of a coccus is $1.25\ \mu$, the transverse diameter $0.6-0.8\ \mu$, and they occur, as just stated, in the form of diplococci and as groups of four; the cocci are crescentic, and in this respect do not differ from many other species of cocci (see Section A of this article). Besides these diplococci, cocci often occur in the pus of gonorrhœa which are spherical and probably belong to the staphylococcus species (*liquescens albus* and *liquescens aureus*).

The gonococcus does not grow on nutrient gelatine, on agar mixture, or potato, and herein differs materially from the ordinary cocci occurring in pus. Bumm has proved that the gonococcus grows only on blood serum, and Löffler and Krause have also succeeded in growing it on serum. In streak cultures on moderately solid blood serum and kept at 32°C ., well moistened, the gonococcus, according to Bumm, grows in the form of a thin, narrow, greyish-yellow film, 1-2 mm. in breadth, with smooth and moist-looking surface. The growth does not proceed for more than a few days, and then dies. Animals are refractory against the gonococcus or the gonorrhœal secretion; dogs, rabbits, monkeys, horses show no reaction, either on the conjunctiva or on the urethra. Bumm has, however, succeeded in producing in the human subject real gonorrhœa by inoculating, from a culture of the gonococcus, the urethral mucous membrane.

There can be no doubt about the fact that the gonococcus which, as mentioned above, grows well on serum, is peculiar to gonorrhœa, and cannot, therefore, be confounded with other pus micrococci. Probably Neisser's gonococcus was only a pus coccus, since it grew also on other media.

Koch ('Cholerabericht,' 1888) has shown that what is spoken of as 'Egyptian ophthalmia' is really two kinds of infectious ophthalmias: one is an acute blennorrhœa or purulent ophthalmia, and does not differ from that known to occur in consequence of infection with gonorrhœal exudation.

Catarrhal Conjunctivitis (Koch).—A second, the true Egyptian ophthal-

mia, is, however, of an altogether different etiological character, though in its symptoms and pathology it is similar to, but not identical with, the blennorrhœa. This second one, the '*catarrhal conjunctivitis*,' is associated, not with the gonococcus, but with a minute fine bacillus, very similar in morphological respects to the bacillus of Koch's mouse septicæmia mentioned in a former chapter. In this ophthalmia the bacillus is present in the purulent exudation of the conjunctiva as isolated rods, but more commonly enclosed within the pus cells, whose protoplasm is sometimes found crowded with them (*see fig. 97*), in the same way as we saw the leucocytes in the mouse septicæmia crowded with the small bacilli. The cultivation of these bacilli carried out by Kartulis (*see below*) shows that there exist definite differences between this and the septicæmia bacillus of mice.

Kartulis, besides describing minutely the symptoms and course ('*Centralbl. f. Bact. und Parasit.*,' Band I., No. 10, pp. 289-298), and the differential characters existing between blennorrhœa of the conjunctiva and the catarrhal ophthalmia, succeeded in cultivating the bacilli of the catarrhal or true Egyptian conjunctivitis. He showed that they do not grow on nutrient gelatine; on blood serum or on agar they grow well between 28-36° C., forming in thirty to forty hours small white punctiform colonies, prominent over the surface of the medium; when closely sown (e.g. in streak culture) they soon coalesce into a whitish grey band of a fatty glistening appearance; the margin of the band is wavy or crenated. Animals inoculated on the conjunctiva with the conjunctival secretion or with the culture prove refractory; but Kartulis succeeded in producing with the culture the typical catarrhal conjunctivitis in one out of six cases in the human subject. The pus corpuscles resulting in this case were crowded with the characteristic bacilli. This one case was that of an individual twenty-five years old.

Xerosis Conjunctivæ.—Under this name is understood an inflammation of the conjunctiva associated with the secretion of whitish, fatty scales and clumps, consisting of fatty, degenerated epithelial cells with a few leucocytes. In these scales certain minute bacilli have been demonstrated by Kuhnbert and Neisser, Leber, Schatz, and others, in large numbers, almost in pure cultures; these were assumed to be the microbes of the xerosis. Schleich, Sattler, Fränkel, and Franke found them, however, in the secretion of the most varied inflammations of the conjunctiva and cornea, in trachoma, in corneal abscess, and in suppurations of the eyeballs; and Schreiber has definitely established that, not only do they occur in many diseased conditions of the eye, but also on the normal conjunctiva: this view is in harmony with that expressed by Weeks and also Fick, viz. that the xerosis bacillus is a common saprophyte, which owing to the pathological conditions in the eye finds an excellent medium and suitable conditions for its copious multiplication.

CHAPTER XVIII

ACUTE EXANTHEMATA

THE diseases belonging to this group are: (1) foot-and-mouth disease; (2) small-pox and vaccinia, and chicken-pox; (3) scarlet fever; (4) measles and rubeola; and (5) typhus fever. Strictly speaking, we ought to include here also erysipelas, but this has been treated already in a former chapter, together with the septicæmic diseases.

FOOT-AND-MOUTH DISEASE

This acute infectious disease affects cattle, sheep, pigs, and man. In cattle, after a sudden febrile rise of temperature and general constitutional disturbance (falling-in of the flanks and falling-off in feeding) there appears great salivation of a mucoid viscid saliva, at the same time on the mucous membrane, and about the lips, the mouth, the gums, and also the tongue, there are visible red papules of irregular shape, which in a day change into vesicles or blebs; these blebs have only a very short duration, since by the breaking of the superficial layer—the cover of the bleb—due sometimes to mechanical causes, they are changed into ulcers; the mucous membrane surrounding the ulcer is injected and the base of the ulcer is slightly depressed and often bleeding; in a few days the ulcer becomes covered with a thin cuticle, and then begins to heal; the whole is slightly depressed. While this eruption appears in the mouth a similar change occurs in the skin of the cleft between the hoofs and in the soft skin near the margin of the hoofs, the skin being at first swollen and injected, then a distinct vesiculation taking place, this by bursting becoming changed into a sore. These sores are very extensive, comprising in many instances the whole of the skin of the cleft between the hoof, and also extensive areas at the side and above the hoofs; the margin of the sore is tumid; the base of the sore dark red and bleeding. These sores produce, of course, lameness of the extremity; the animals move with difficulty and with pain; when both hind or both fore extremities are involved, movement is well-nigh impossible. These ulcerations last for several days, during which time the animals do not feed well, do not ruminate, and do not move; sometimes the extent of these ulcerations is very great, and the animals are then in a very helpless and pitiful condition.

In the sheep the eruption in the mouth is not so common as in cattle; in most cases it is limited to one extremity; here it is of the same nature as in cattle.

In pigs the eruption is most intense about the snout; sometimes sores of the size of a shilling and a florin being observed; the eruption is present on the feet at the same time.

In the human subject, foot-and-mouth disease shows itself as a febrile disorder associated with and followed by a red papular eruption of the skin on the hand and fingers, more rarely the toes and dorsum of the feet, and on the lips and the mucous membrane of the mouth, fauces, and pharynx. The papules are situated on an injected basis and become small blisters, then dry up into a thin crust, underneath which is a sore, which in a few days, as a rule, heals up; in rare cases the ulceration in the mouth is more of a diphtheritic character, and then runs a much longer course.

In respect of the intimate pathology of the eruption of the skin and mucous membrane, the following have been shown:¹—

Sections made through the entire vesicle and the skin adjacent to it show the following (*see* fig. 81): In the central region of the vesicle, the superficial corium and the papillæ are densely and almost uniformly infiltrated with round cells, and similar cells surround all vessels throughout the entire thickness of the corium. In the peripheral portions of the vesicle the infiltration is less marked, and is not found at all in the skin bordering the vesicle. In some of the enlarged (thickened) papillæ, besides the round cells, isolated red blood-corpuscles are met with, and even ruptured blood-vessels with hæmorrhage. The vesicle is loculated and made up of a number

¹ *Report of the Medical Officer of the Local Government Board, 1885, p. 122 et passim.*

of smaller and larger cavities, extending through the whole thickness of the rete Malpighii; in the centre the cavities are largest and longest, reaching in a vertical direction from the superficial corium to the stratum corneum of the epidermis. The septa between the cavities are strands of compressed epithelial cells of the rete Malpighii. As the disease progresses these septa are broken, and the cavities enlarging ultimately break through and open on the free surface; in consequence of this an ulcer is established. The contents of the vesicle are a fibrinous fluid: in it are numerous round cells, detached epithelial cells and their nuclei, many in a state of degeneration.

The whole disease is then an acute inflammation of the corium and papillæ, leading to the exudation of round cells and fluid into the corium, the papillæ and the stratum Malpighii, in which latter cavities are established, gradually enlarging and becoming confluent.

In the mouth the eruption has the same character, except that the cavities formed within the epithelium are not so large and well marked; hence the vesicle is not so conspicuous; but there is here the same dense infiltration of the mucous membrane and of the epithelium, with lymph and leucocytes, and after the rupture of the superficial epithelium an ulcer of the mucous membrane is established.

That the disease is transmissible from the cow to human beings by the milk has been proved by various outbreaks, investigated and described by Dr. Thorne Thorne (Reports of Medical Officer, Local Government Board), and that it is also transmissible by direct contact is known to veterinary surgeons, who, by inspection of, and attendance upon diseased cattle, have been known to have contracted the disease on the skin and in the mouth. The way, however, in which the disease spreads from animal to animal is not well understood; that it does so spread easily and sometimes with great rapidity, of this there is unfortunately abundant evidence, and for this very reason it is so much dreaded by farmers. If in a herd of cattle an animal shows the disease, there is little chance that after a few days many will be found free of the disease; in a flock of sheep, one diseased animal means in many instances the majority of the flock. That the saliva of a diseased animal, that is, an animal affected with the eruption in the mouth, produces the disease by contact with the healthy mucous membrane of the mouth in another animal, for this there is but fragmentary experimental evidence. Mr. Duguid and Dr. Sanderson in 1874 made a number of direct experiments by rubbing hay, moistened with the saliva, taken from cattle affected with the eruption in the mouth, into the mucous membrane of the mouth of healthy cattle, but only in one single instance did they succeed in producing the disease. In this single instance the infection may have been produced, not by the direct contact at all, but by way of the stomach or intestine, or the respiratory tract. It is more feasible to assume that infection is carried out by animals while feeding on grass or hay, or licking articles which have been tainted with the diseased matter of the mouth or feet of affected animals and thereby introducing the virus into their system by the alimentary canal or the respiratory tract; this would be in harmony with what actually occurs in the human subject, e.g. by milk. The eruption being in the mouth and on the feet does not and cannot of course mean that the contagium has been introduced at those places, either in animals or in man; this is no more the case in foot-and-mouth disease than in small-pox, but like this latter is a result of the general infection of the system with the virus. In experiments which the writer has made with the microbe of the disease presently to be described, the typical disease on the feet of sheep was produced by feeding.

When fine sections through the vesicle (at its full development, and before it is broken) of the eruption in sheep are made and well stained, one finds in the contents of the vesicles amongst the leucocytes and the fibrinous and the albuminous material, cocci arranged as diplococci and as chains, streptococci: their diameter is about $0.2-0.4\ \mu$; similar cocci can be demonstrated also amongst the epithelium cells below the vesicle, i.e. those in contact with the corium. From the contents of such vesicles, at their full development, and before they are broken, the writer has isolated by cultivation these streptococci. In the manner in which they form chains when growing in fluid medium (broth) they resemble other streptococci, e.g. streptococcus pyogenes, except that the cocci in the foot-and-mouth microbe are very much smaller and the chains are more wavy; also in the way in which they grow on the surface (streak and plate cultures) and in the depth (stab culture) of nutrient gelatine, on and in agar, and on solid blood serum, the streptococci of foot-and-mouth disease resemble other kinds of streptococci, e.g. the streptococcus pyogenes, the streptococcus of scarlet fever, &c.; but on nutrient gelatine the streptococcus of foot-and-mouth disease grows much more slowly than the others mentioned just now: the colonies are smaller, and are conspicuously more transparent than those of other streptococci. On making a streak cultivation on the surface of gelatine, the line of inoculation becomes noticeable after a few days, as consisting of very minute dots, not confluent; the whole growth is very transparent as compared with a similar growth of streptococcus pyogenes.

With broth cultures of the streptococcus of foot-and-mouth disease the writer has by feeding sheep been successful in producing the typical disease, viz. typical vesicles on the feet.

The same streptococci as were used for the feeding were again recovered by culture from the vesicle of the artificially infected sheep. So that it may be considered as established that the streptococcus present in the lymph is the actual microbe of the foot-and-mouth disease.

Schottelius quite recently (1892) observed in the lymph of the vesicles the presence of streptococci, which he, however, calls streptocytes, but from his description there is no necessity for considering them other than streptococci.

HUMAN VARIOLA

Pathology.—The eruption of the skin known as small-pox commences as a small localised swelling of the congested skin, in fact, as a minute firm papule, the swollen skin forming the papule, as well as the skin immediately surrounding it, being much congested and red. Next, the papules enlarge in thickness and breadth, and at the same time show in their marginal part clear evidence of vesiculation; the centre is less prominent than the marginal vesicular portion, hence a characteristic 'umbilication' is apparent. Next the vesiculation extends over the whole pock, which hereby loses its umbilicated aspect and becomes convex, the centre as convex as the marginal part, that is to say, the whole pock is converted into a flat or slightly acuminate vesicle. The vesicle at first contains clear lymph, which soon becomes turbid owing to the presence in it of numerous leucocytes or pus cells. Next, the vesicle dries up, the covering and contents becoming converted into a brownish crust; at this time the injection of the skin surrounding the pock becomes more marked, thus forming a more or less broad areola. In a few days the crust becomes loose, the parts underneath being slightly moist (purulent), and red; finally it contracts and heals, and the place of the former pock is indicated by a slightly tumid

reddish mark. This course is, however, only noticeable in the milder cases ; in severe cases the pocks are so closely placed—face, arms, and legs—that when they enlarge and become changed into vesicles these become in many places confluent, and such confluent patches sometimes measure an inch to two inches or more in breadth in one or the other diameter. Such confluent small-pox cases are always marked by a deeply congested skin surrounding the vesicles, and also by this, that when the vesicle dries up into a yellowish or brownish scab, and after this has become loosened and detached, an extensive ulcer is established, the surface of which is entirely denuded of the epithelium and the superficial or papillary layer of the cutis, and is covered with, and infiltrated by purulent matter ; when this ulcer heals it always leaves behind a reddish cicatrix, at first raised beyond the surface of the surrounding skin, but by-and-by shrinking and becoming converted into a pale whitish pitted cicatrix. In malignant forms of small-pox the lymph of the vesicle at its first appearance is not a colourless, transparent fluid, but is more or less deeply stained by blood, the skin itself surrounding the vesicle containing blood effused in its tissue. This is the hæmorrhagic form, and is due to rupture of blood capillaries in the superficial parts of the corium both in the region of the pock as well as that surrounding it. Sections through the pock in its various stages show the following features : (a) in the stage of the papule : the blood-vessels of the corium and papillæ are distended and injected, more in the corium of the papule itself than in the corium immediately surrounding it ; the papillæ themselves are enlarged, being both thicker and longer ; in their tissue numerous lymph cells are already noticeable ; the stratum Malpighii of the epidermis is thickened, owing to a rapid multiplication of its cells, and also to distinct swelling of the cells of the middle layers. In a vertical section the stratum Malpighii in the region of the papule is twice the thickness of that of the surrounding skin ; the interstitial spaces, i.e. between the epithelial cells, particularly those in the upper layers of the stratum Malpighii, are very conspicuous by their breadth. (b) In the stage of the vesicle, the first indications of vesiculation are shown by the appearance of microscopic cavities, round, irregular, or elongated in the upper layers of the stratum Malpighii *in the marginal region* of the papule, hence the pock appears umbilicated ; these spaces and cavities are entirely due to the enlargement of the interstitial spaces between the epithelial cells. Owing to the congestion of the vessels of the superficial corium and the papillæ we are justified in assuming that a considerable amount of lymph exudes, not only into the tissue of the corium—hence the swelling of the corium—but also into the epithelium, and for this reason the intercellular spaces become more apparent ; and, further, as this exuded lymph passes outwards, it will accumulate between the cells of the more superficial layers of the stratum Malpighii of the marginal part of the pock, the dense horny layer above, i.e. the stratum lucidum and corneum, forming a barrier to the further passage. Owing to this accumulation of lymph, the intercellular interstices change into cavities of various sizes, and the epithelial cells themselves become involved, some being compressed, and others gradually swelling up and disintegrating. As the exudation of lymph proceeds, so the superficial layers of the stratum Malpighii of the marginal part contain more and more of those cavities, while the epithelial cells themselves become greatly disorganised ; the cavities while enlarging become closer and longer, and ultimately become more or less confluent, separated only by streaks and septa which are the remnants of the former epithelial cells. The presence and increase in numbers and size of these marginal loculi, filled with lymph, produce the marginal vesiculation,

the normal skin forming to a certain extent a barrier to the progress of the vesicle; the further progress of the exudation will produce accumulation of lymph also toward the central part of the pock; hence cavities, filled with lymph, become established in the superficial parts of the thickened stratum Malpighii, and this causes the disappearance of the central umbilication of the pock. The exudation of the lymph proceeds from the engorged vessels of the corium and papillæ of the pock, and thereby the cavities in the stratum Malpighii become more and more enlarged, so that almost the whole of the middle and upper layers of the stratum Malpighii become converted into larger and smaller cavities, some confluent, others separated only by thin trabeculæ and septa: these latter are composed of the remnants of the former epithelial cells, some of them compressed into non-nucleated strands, others still showing a remnant of a nucleus, but their substance opaque or breaking down into granular *débris*. The cavities constituting 'the vesicle' contain 'lymph,' which in the hardened preparation is in a coagulated state: it contains numerous threads of fibrin, and in its first appearance, in the marginal part, a few lymph cells, and also more or less *débris* of broken-down epithelial cells are to be noticed. As the central part of the pock becomes also vesiculated, the number of lymph corpuscles present in the cavities is greatly increased, and this gives to the lymph the opaque or purulent character, and constitutes the stage of the pustule. (c) During the formation and progress of the cavities in the marginal, as also in the central part of the pock, there is great emigration of leucocytes from the blood-vessels, so that the deepest layer of the epithelium, the tissue of the papillæ and of the superficial corium all become infiltrated with crowds of leucocytes. Their passage from the papillary layer between the cells of the deepest layer of the epithelium, and further into the cavities in the middle and superficial layers of the stratum Malpighii, can be easily traced; at the same time they invade the epithelial cells themselves and help towards their destruction.

(d) The stage of the crust. When, as is the case in mild forms, the vesicle dries up as a whole and is converted into a crust, we find that beginning with the margin a new stratum lucidum is formed underneath the cavities of the vesicle; that is to say, the deeper layers of the stratum Malpighii in the region of the pock become gradually separated from the middle and upper layers, those containing the cavities, by a layer of horny epithelium, so that the vesicle in the narrower sense becomes enclosed within two horny layers, one on the surface, the former stratum lucidum and corneum, and one in the depth, the new stratum lucidum and corneum. This condition it is which has given rise to the statement that the vesicle is developed within the layers of the stratum lucidum, an incorrect interpretation, in which the new horny stratum separating the lower layers of the stratum Malpighii from the vesicle is mistaken for a portion of the original stratum lucidum. The vesicle is now separated by a horny layer from the deeper parts, and its contents, therefore, dry up and become converted into a crust, which ultimately loosens and falls off, leaving no scar behind, the deeper layers of the epithelium and the papillæ having been preserved underneath the new horny layer. At the same time the congestion abates, and the exudation of lymph and leucocytes gradually decreases, and finally disappears altogether from the epithelium and the papillary layer.

But when, owing to the severity of the process, the enlargement of the vesicle proceeds and extends beyond the middle layers of the epithelium, and the infiltration of the epithelium and papillæ with pus cells continues, then the whole thickness of the stratum Malpighii becomes involved in the

vesicle and the crust, and the tissue of the superficial layers of the corium becomes destroyed; a typical ulceration is the result, of which the final result is seen in a pitted, well-marked scar.

VARIOLA OVINA

In sheep-pox the pathology is the same as in variola of man, both in cases caused by inoculation with lymph taken from a previous vesicle produced by inoculation—so-called vaccine vesicle of sheep—and in cases of a general eruption, natural as well as artificial, as after injection of lymph into the vein of a healthy sheep. In the papular stage, the vesicular stage, the pustular and ulcerative stages, the microscopic appearances are the same, the duration of the different stages being, of course, different from those of human small-pox. The local pock produced by inoculation of lymph is slower in its development, and is much larger than that of the general eruption, reaching a size of one-half to an inch in diameter. The vesicles of the general eruption run a much quicker course, and are much smaller; those on the lips containing purulent lymph as early as the second or third day, so that from the first appearance of the cavities in the stratum Malpighii, in the marginal part of the pock, to the formation of the cavities in the middle part of the pock, not more than some hours intervene.

VARIOLÆ VACCINÆ

The spontaneous eruption on the teats and udder in cows of papules, leading to vesicles, and these again to brown or black-brown thick crusts, known since the days of Jenner, Ceely, and others as cow-pox, shows a pathology identical with human variola. During the last two or three years the writer has had the opportunity of studying such spontaneous cow-pox in Gloucestershire and in Surrey. The first symptoms consist in the presence of red papules on a tumid injected skin; the size of the papules is in some places not more than that of a split pea, in others that of an ordinary pea; the skin is tumid and much injected. In a day or two after their appearance the marginal part of the papule begins to vesiculate, and is of light colour, while the centre is dark and umbilicated; the vesicle is distinctly surrounded by a sort of areola. In another day or two the whole papule is a vesicle, at first loculated, but soon continuous, the whole eruption having at the same time become much enlarged. The lymph which can now be squeezed out of the vesicle after breaking the superficial horny cover is clear, and under the microscope contains few lymph cells. But soon the lymph becomes purulent as the lymph cells increase in numbers; then the whole vesicle dries up into a brownish black crust which gradually thickens, the pock enlarging all the time, the corium itself being tumid and infiltrated; the scab remains firmly adhering, and continues to increase in thickness for about a week; the surrounding skin is at first much injected, but gradually becomes paler. Not till the beginning or middle of the third week does the scab show any signs of loosening; after this it begins to loosen, and finally becomes altogether detached, leaving a scar behind; the time of falling off differs in the same animal considerably, some extending over five to six weeks. Underneath the crust is a sore, covered with purulent matter; this ulcer only slowly and gradually contracts and dries up, becoming finally converted into a scar, at first reddish, but gradually becoming pale. If the crust is forcibly taken off while it thickens, a new crust is formed on the ulcer. As to the microscopic appearances, they are essentially the same as in human and sheep variola: the microscopic character of the papule, the first formation of the

loculated vesicle, the extension of the vesicle over the whole pock, the purulent change of its contents due to the presence and increase of leucocytes, the condition of the epithelium, of the papillæ and corium in their different stages, the changes leading to the formation of the ulcer, and the character of the scab, all these are essentially the same in both processes.

By inoculation of lymph taken from the vesicle during the first week, or by using a young scab not later than the tenth day, and inoculating it into fresh cows or calves, typical vaccinia is produced, such as is well known since the days of Jenner, Ceely, Chauveau, and many others. For illustration of the appearances of the various stages in the calf, *see* the writer's paper in the Report of the Medical Officer of the Local Government Board, 1889.

Likewise inoculation of susceptible milkers into a cutaneous wound—generally the skin of the hand—produces at the place of inoculation an eruption known as human vaccinia, which in all essentials is identical with that of the cow or calf: the formation of the papule (third day); the gradual enlargement of this and the formation of an umbilicated loculated vesicle with dark centre (sixth to eighth day); the formation of the surrounding areola; the extension of the vesiculation over the whole of the enlarging pock (ninth or tenth day); the purulent change; the drying up of the vesicle into a dark brown gradually thickening crust, and the presence underneath it of a deep red sore; the loosening and ultimate falling off of the crust; the contraction and healing of the sore, and the leaving behind of a pitted mark, at first reddish, gradually becoming pale or white; all these are the same as in the calf.

Using lymph or crusts, after the purulent stage has set in, an eruption is produced both in the calf and in man, which runs a more rapid course, and is of a milder character than the above. Retrovaccination of the calf with lymph taken from a human vesicle not later than the eighth day produces typical results. The microscopic nature of the inoculated vaccinia is identical both in the calf and man: the nature of the papule, the formation of the vesicle, or rather loculi of the vesicle, the gradual enlargement of this and extension over the whole pock, the purulent change, the microscopic character of the changes in the epithelium, corium, and papillæ, are in all respects the same.

Various eruptive diseases on the teats and udder of the cow have been described by veterinary surgeons as true cow-pox, but on careful investigation, they do not bear this interpretation. Jenner and Ceely have already distinguished some such eruptions as 'false' cow-pox. In all of these eruptions it can be shown that they are contagious and spread from animal to animal, generally by the instrumentality of the milker's hands in the act of milking, being as it were transferred from the infected teat of one cow to the teat or udder of a healthy animal, a chap or any abrasion of the epidermis in the latter animal being the seat of the inoculation, which is generally followed by the appearance of a red pimple. In some instances this pimple, while enlarging, vesiculates more or less perfectly, and further changes into a brown crust, underneath which an ulcer of the corium is present. But the duration, general character, and course of the disease in the cow are not the same in all these eruptive diseases: in cow-pox it is of one kind, in the Hendon disease of another, and in the Edinburgh disease of a different kind.¹ The duration of the different stages, the nature and

¹ A further eruptive contagious disease in the cow may be added, which was produced artificially by the writer with the bacillus diphtheriæ. *See* a later chapter.

aspect of the ulcer underneath the crust, the period of healing, and the general condition of the cow, are different in these several diseases. Inoculations with matter taken from one kind of eruption or another produce different results both in man and in calves. The lymph or the crust of real cow-pox before the end of eight days, when accidentally inoculated into the hand of a susceptible milker, gives rise to typical vaccinia, i.e. a red papule on the third day, a well-marked umbilicated loculated vesicle with dark centre and surrounded by an areola on the sixth to eighth day, and a pitted white mark after the sore has contracted and healed. Lymph or crust taken from such a vesicle during the sixth to the eighth day, and inoculated into calves or susceptible human beings, always produces true and typical vaccinia. If lymph or crusts are used after the tenth day, an accelerated eruption is produced: the pocking and redness are marked as early as the second, vesiculation on the third day, purulent change very soon after; the crust is thin and becomes loose by the end of the week; the corium underneath is only slightly infiltrated, and soon heals: no distinct scar is noticed several weeks after.

If lymph or crust be used from the eruption on the milker's hand, which eruption had been contracted from natural cow-pox, and if such lymph be retrovaccinated on calves, the result is typical vaccinia (Jenner, Ceely). The illustrations given in the Report of the Medical Officer of the Local Government Board for 1889 show such a result. Calves vaccinated successfully, i.e. in which the process takes the typical course, are refractory to a second or revaccination.

Two other eruptive diseases on the udder and teats of the cow, which the writer had the opportunity of investigating, proved themselves to be different from cow-pox, though veterinary surgeons and others were and still are inclined to consider them all as 'cow-pox.' One of these was a contagious malady in cows at a Hendon farm. It was proved by Mr. Power and the late Dr. Cameron that the milk of certain cows of this farm had produced human scarlet fever in the north of London about the end of 1885 (*see* a further chapter). These cows were affected with an eruptive disease on the teats and udder, which though offering a certain resemblance to cow-pox was, nevertheless, distinctly different from it; the similarity consisted in the fact that the eruption appeared on the teats and udder, that it commenced as red papules, that it showed vesiculations, and that these dried up into brown-black crusts covering the sores; but here the resemblance terminated. The difference was manifested by the conspicuously more rapid course of the eruption than in cow-pox, by the greater infiltration of the corium at the eruption, by the fact that unlike cow-pox the eruption was not communicated to the milkers' hands, and by the important visceral lesions that the Hendon cows suffered from: these will be described in a further chapter. Inoculations with matter from the eruption into calves produced in them results which were also different from those produced in calves by vaccination.

A second eruptive disease in cows which was investigated was very similar to the one observed at the Hendon farm; this disease affected cows in Edinburgh, the milk of which had been proved to have produced febrile sore throat in the consumers. The eruption passed through a more rapid course than cow-pox, was not communicated to the hands of the milkers, produced on inoculation into calves a disease different from vaccinia, and was also associated with definite visceral disease in the cow. This will be more fully described in the chapter on SCARLET FEVER.

Towards the end of 1887 Dr. Crookshank described, before the Pathological Society, cases of eruptive disease on cows' udders and teats, occurring in Wiltshire, and which

owing to the fact that the disease was communicable to the milkers' hands, on which it had produced vesicles resembling vaccinia, he felt convinced to be the 'true Jennerian cow-pox.' The proof which he then adduced as to the Wiltshire disease being true cow-pox rested on the facts (1) that the disease communicated to the milkers' hands resembled vaccinia; (2) that calves retrovaccinated from such a milker's vesicle developed at the insertion 'typical vaccinia;' (3) that these calves failed on revaccination with true vaccinia; and (4) that the milkers (at the Wiltshire farm) who had contracted the eruption from the cows failed afterwards to take 'vaccinia.' It is quite possible, or indeed probable, that the Wiltshire cow disease was true cow-pox; but neither at the time Dr. Crookshank first asserted it to be so, nor later on, did he furnish satisfactory or sufficient evidence for it. The assertion by Dr. Crookshank that the calves which were inoculated from a milker's vesicle did really develop 'typical vaccinia' turned out afterwards to be unreliable, since out of four of such calves three, on subsequent vaccination with real cow-pox a few weeks afterwards, at the Government Animal Vaccine Station, took typical vaccinia; a result which could not occur had these calves really had vaccinia previously. From the vesicles¹ produced at the animal vaccine establishment in one of these three animals children were vaccinated with typical results.² After the success of this second true vaccination of the calves had been made clear, they were declared by Dr. Crookshank not to have been sufficiently well vaccinated in the first instance, and therefore were not protected thereby against a second vaccination.

It must be obvious that evidence adduced by Dr. Crookshank in *subheads* 1, 2, 3 and 4 is not direct experimental evidence, and cannot therefore be considered as unassailable; that mentioned in *subhead* 3 was the only direct evidence that could be experimentally tested: this was done, and, as we have just seen, broke down completely.

The writer is inclined to think that the Wiltshire disease was probably true cow-pox; but that Dr. Crookshank in inoculating the calves from the milkers' hands failed to produce in them this disease.

Jenner from his own and previous observations has shown that natural cow-pox bears a definite relation to human variola: the first protects against the latter, in the same way as one mild attack of variola protects against a second virulent attack, or as in some other infectious diseases one mild attack protects against a second attack; all protective inoculations, such as in anthrax, charbon symptomatique, hydrophobia, fowl cholera, &c., are well founded on experiment, and often practised with great success. The facts observed by Jenner and Ceely, confirmed by a host of other observers, well qualified to judge from special knowledge and a life-long experience, viz. that natural cow-pox protects the cow from a second vaccination; that one successful vaccination protects the calf against a second inoculation; that persons (milkers) who have contracted the eruption on their hands or other parts from handling the teats of cows affected with the natural cow-pox become insusceptible to small-pox; and, above all, the fact that persons efficiently vaccinated and revaccinated from the calf or with humanised vaccinia are protected against small-pox, require little additional proof. The intimate relation that has thus been proved to exist between cow-pox and human small-pox suggests that the two eruptions are two forms of the same disease, the former in its natural occurrence as natural cow-pox being the small-pox of the cow and of a mild character as compared with the human form, which is more virulent, and in unprotected or in insufficiently protected persons generally assumes a grave character.

The great similarity in the pathology of the two eruptions also offers considerable support to this. Ceely has shown by direct experiment that cow-

¹ The companion calf was reinoculated with humanised vaccinia by Dr. Crookshank. Result doubtful. It failed on being inoculated afterwards with animal vaccine lymph.

² In a paper published by Dr. Crookshank in the *British Medical Journal*, July 1888, he states that this calf had practically failed on reinoculation at the Government Animal Vaccine Station. This statement is shown to be incorrect by the subsequent successful vaccination of children from this calf.

pox is really small-pox in the cow, and he and Badcock, as is well known, have in several instances—the latter in a large number of instances—succeeded in producing the eruption of vaccinia by direct inoculation of lymph or crusts taken from human variola into the skin of heifers. The lymph of the latter was then used for producing in the human subject typical vaccinia, which has been subsequently propagated as vaccinia for many generations of vaccinifers. Chauveau in his experiments, on the other hand, has failed; he maintains that he produced in the cow a papule, but the matter taken from such papule produced again in the cow only papules: in the human being, in some instances, it produced variola. But these negative experiments do not bring us nearer the decision of the question, any more than negative evidence ever does.

Voigt has also succeeded in producing vaccinia in the calf by inoculation with matter taken from human variola; and as was the case in Ceely's experiments, here also the positive result was achieved when, simultaneously, vaccinia was inoculated at a different part of the skin. True, the objection raised against these positive results as to an accidental introduction of vaccine matter, when only variolous matter was believed to have been used, is not an unfair one, and there can be, on scientific principles, no fault found with those who decline to accept it as conclusively proved, and free from any objection. But when some writers go so far as to say that because Chauveau and others (the writer amongst them) have failed to produce vaccinia in the cow by inoculating variolous matter, therefore it is proved that the two are essentially different eruptions, having nothing in common with each other; to these we are quite justified in saying, in opposition, that such a conclusion is not warranted, nor is it quite in harmony with the facts. Any number of failures incurred by one experimenter do not disprove the success of another, even if obtained in a single instance, any more than any number of negatives disprove one single positive; all that the failures prove is that the positive result is difficult of achievement and that the conditions under which it is or can be achieved are not known, but depend on chance. In the writer's report on inoculation of cows with variola (Medical Officer's Report to the Local Government Board, 1878) he has shown that by such inoculation of the cow some refractory condition against vaccinia is obtained, so that from this it must be concluded that a correlation between the two eruptions did exist.

Quite recently Fischer ('Münch. med. Woch.,' 1890, No. 43) has succeeded in producing in two calves an eruption by inoculation with human small-pox; the lymph of the eruption of these calves was transmitted through a series of other calves; from these lymph was obtained which, inoculated into infants, produced typical vaccinia. Two other experimenters (Dr. Simpson and Dr. King) have separately and independently, in India, produced vaccinia in the calf by inoculation with human variola.

Microbes.—Chauveau was the first to show by carefully arranged diffusion-experiments, that the active principle of sheep-pox and of vaccinia is particulate matter. Coze and Feltz, Hallier, Zürn, Cohn, Burdon Sanderson and others, have demonstrated in vaccine lymph the presence of bacteria, bacilli, and micrococci. The writer has shown the presence of micrococci in sheep-pock lymph, and in the lymphatics of the corium of sheep-pocks; F. Cohn, and then Weigert, showed the presence of micrococci in the lymph and lymphatics of human variola. Only comparatively recently, after the methods of cultivating micro-organisms had been perfected by Koch, has it been possible to isolate the different species of bacteria occasionally but not invariably present in variolous and vaccine lymph. Before giving the results of these cultivation

experiments it is necessary to state that various observers have found torula-forms in the variolous lymph (Hallier), and in the vaccine (L. Pfeiffer). These torula-forms are, however, admittedly only accidental contaminations derived from the air.

Of the micrococcus forms—accidental and occasional inhabitants of lymph—that have been cultivated by L. Pfeiffer are various sarcinæ: *sarcina lutea*, *sarcina tetragonus*, *sarcina aurant.* and *musculus*. Of bacilli there have been occasionally found in calf lymph, seldom in lymph of children, short motile rods, liquefying gelatine, and most probably comparable to Hauser's *proteus vulgaris*—the common putrefactive bacillus. Of micrococci there are two species which, both in clear and in turbid preserved lymph of vaccine and variola, are occasionally met with; viz. the *staphylococcus pyogenes aureus* *liquescent* and the *staphylococcus cereus albus* (non-*liquescent*). P. Guttman, L. Pfeiffer, and Garré have described these two species as often occurring in preserved lymph. The writer has devoted considerable attention to the culture from human vaccine lymph of micrococci, and he finds that the lymph of a considerable percentage of tubes examined by him, lymph which was and remained perfectly 'clear' when tested by cultivation on gelatine, agar, solid blood serum or broth, was found to be free from any of those microbes; no growth of any kind appearing in the tubes. There was also a percentage of tubes, the lymph of which was 'clear,' but on cultivation yielded a few colonies of *staphylococcus cereus albus*. In the case of lymph which was labelled 'clear lymph,' no *staphylococcus aureus* could be demonstrated by cultivation; but in a certain percentage of tubes discarded as 'opaque lymph' there was present, sometimes in large numbers, the *staphylococcus aureus* alone, or with *staphylococcus cereus albus*; in some cases gelatine tubes or agar tubes inoculated with a trace of the lymph yielded innumerable colonies of them, particularly of the *staphylococcus aureus*. Cover-glass specimens of some opaque lymph revealed large numbers of cocci, single, as dumb-bells, or in clumps. In a small percentage of tubes containing opaque lymph the number of micrococci present was small, and could not account for the opacity; in one case of opaque human lymph the writer cultivated a streptococcus in pure culture. Garré had already met with streptococcus *pyogenes* in the lymph of pemphigus complicating variola; the streptococcus which the writer has cultivated in the above case of opaque lymph was certainly not streptococcus *pyogenes*; it grew much more slowly, and its colonies in gelatine were conspicuously transparent when compared with those of streptococcus *pyogenes*.

The writer had the opportunity of examining the lymph of a child in which vaccinia was complicated by erysipelas, such lymph having produced erysipelas in two infants vaccinated with it. From this lymph the streptococcus of erysipelas was cultivated without difficulty. Gelatine-plate cultivations made with the lymph brought forth a pure culture of this streptococcus in innumerable colonies. So far as can be seen from the literature of the subject, this is the first time that the streptococcus of erysipelas has been actually demonstrated in lymph.

Though some experimenters assert that they have produced true vaccinia on inoculation of calves with cultures of one or the other of the microbes, it will on careful examination be agreed that these alleged positive results are not, and cannot be accepted. Competent investigators ('Berlin. Reichsgesundheitsamt,' 1881) have altogether failed to produce such results. The matter then stands thus: The microbes which have as yet been cultivated from lymph (variola or vaccinia) outside the animal body, freed of all accidentally

adhering animal matter, have not the power to reproduce the disease on inoculation into a suitable animal. After the numerous failures to cultivate on the usual media—gelatine, agar, solid blood serum, broth—any of the above bacterial forms from samples of 'clear' and very good and active vaccine, the writer is in full agreement with those negative assertions, and is prepared to say that the microbes hitherto demonstrated as being present occasionally in clear and particularly in turbid vaccine lymph have nothing directly to do with the contagium of vaccinia or variola. Moreover, the presence of some of them, particularly the staphylococcus aureus when abundant, as in some cases of turbid lymph, or that of the streptococcus of erysipelas, in vaccino-erysipelas, forms a serious, preventable, and objectionable admixture.

L. Pfeiffer ('Correspondenzblatt d. allg. ärztl. Intell.,' 840; Thüringen, 1887) describes in the epithelium of the pock of man and animals, a parasite belonging to the coccidia (sporozoa, Leuckart). This parasite is said to live and grow in the epithelial cells, and ultimately to form spores, in size not larger than micrococci. But nothing definite is as yet known about these alleged sporozoa, called by Pfeiffer 'monocystis epithelialis'; moreover, it seems that these alleged coccidia of Pfeiffer are due to pathological (dropsical) changes of the nuclei or the substance of the epithelial cells of the pock, which changes appear traceable through all intermediate stages from normal nuclei and the normal protoplasm, and that therefore Pfeiffer's interpretation of these changes as coccidia is not justified.

SCARLET FEVER

In this acute infectious disease the skin, the organs of the throat, and the kidney are the chief tissues implicated. (a) The eruption of the skin shows the following characters: hyperæmia and inflammatory cedema of the superficial layers of the corium; the epidermis (rete Malpighii) is thickened, due to an active multiplication of its cells, and the same holds good also for the hair follicles. Owing to the continued multiplication of the cells of the rete Malpighii also the stratum lucidum increases in thickness, and the subsequent extensive desquamation of the stratum corneum and stratum lucidum are due to the continued reproduction of the cells of the rete Malpighii.

(b) The kidney. In the early stages of the disease there occurs fibrous and hyaline degeneration of minute arteries and Malpighian tufts of the cortex; the nuclei of the epithelium covering the tuft and lining the Malpighian capsules are increased (glomerulo-nephritis); the epithelial cells lining some of the convoluted uriniferous tubules are swollen, granular, and opaque—parenchymatous nephritis. In some cases after only two days, in most, however, at later stages, there occurs interstitial nephritis, consisting in the aggregation of round cells (leucocytes) in the connective tissue around the arterioles and Malpighian corpuscles of the cortex; numerous such microscopic round-cell aggregations are to be found in the cortex, in some severe cases even involving large portions of the cortex. In such cases one or the other arterial branch is found blocked, and around it the tissue of the kidney, like a wedge-shaped infarct, is densely infiltrated with round cells, the uriniferous tubules with their epithelium in such parts being in a state of granular degeneration. In the organs of the throat in the typical cases, where there is ulceration of the fauces, the mucous membrane is found intensely inflamed, and showing the usual anatomical characters of hyperæmia, exudation of lymph and round cells, and finally loss of the epithelium and the papillary layer of the mucosa, i.e. the formation of an ulcer.

But in all cases that are subjects of a *post-mortem* examination, the spleen, liver, intestines, salivary glands, and pancreas, show more or less pronounced inflammatory changes.

Scarlet fever, as is well known, is a highly contagious disease, the contagion occurring in all stages of the disease. There exists, however, a diversity of opinion amongst sanitarians as to which is the most infective stage; while some maintain that contagion occurs in the later stages—during desquamation—others consider the desquamative stage of less importance in this respect than the earlier phases, i.e. the acute throat illness. That infection does take place in some cases during the desquamative stage, is well ascertained by the experience of many who have studied the subject, but it has yet to be proved that the contagion is as frequent in the later as it is in the earlier or throat stages.

Numerous epidemic outbreaks of scarlet fever are now known in which milk was the vehicle of the contagium. Up to 1881, a number of such epidemics have been described by Ballard, Buchanan, Jacob, Robertson, Darbishire, and many others; they have been carefully tabulated by Mr. Ernest Hart in the 'Transactions of the International Medical Congress for 1881,' and we need not further enter into them.¹ These milk epidemics are nowadays so easily recognised since the classical investigations of Ballard and Buchanan, that there is hardly a health officer in this country who has not had the opportunity, even during comparatively few years of his activity, to himself investigate, or become acquainted with such an epidemic. Whenever and wherever in any given locality a number of cases of scarlet fever occur simultaneously in different and distant households, having no intercommunication with one another—school, church, visitors—the first means of the spread of the contagium thought of is milk, and in all such epidemics on further inquiry it is found that milk (or cream) was actually the vehicle of the contagium. Secondary infection, i.e. cases which occur later and are due to direct contagion, are of course excluded.

Now, until 1882 all such milk epidemics were assumed to be due to infection of a particular milk supply from human scarlatina; it was assumed that either in the house of the milkman or the milkdealer, or at one or the other stations of the milk on its passage to the consumer, the contagium, derived from a human case of scarlet fever, was added to it. As a matter of fact, in the above-named tabulated account of milk scarlatina ('Transactions of the International Medical Congress for 1881,' vol. iv.) several epidemics are mentioned in which this mode of milk infection from a human source was clearly demonstrated. As an illustration may be mentioned the outbreak investigated by Dr. Robertson, of Keswick, in which the contagium had found access to the milk of a dairy closely adjoining a house where scarlet fever had existed for several weeks. The cows were milked, every night and morning, into an open tin can, which was carried across an open yard, past the affected house. The children who first caught scarlet fever in the locality played about the yard while in a state of desquamation. Subsequently, on a particular day a general epidemic of scarlet fever broke out in the town, between thirty and forty families being invaded. All those suffering from the disease received their milk supply from this particular dairy. Some member of every family supplied had on this day either a scarlatinal sore throat or scarlet fever. Other families supplied from a different source escaped the disease. A lodger had the milk raw for supper and was attacked.

¹ It is really curious to find how little such milk-scarlatina epidemics are known on the Continent and, as a matter of course, how sceptical Continental sanitarians are as to a question so well understood in this country.

His landlady boiled her milk the same night and escaped. We must here observe that a large number of cases of scarlatina occurring on the same day, the inference from this fact is that on a previous day, when the children who were peeling from recent scarlet fever were playing in the yard, they conveyed the infection to the milk which was in their neighbourhood.

But epidemics of milk scarlatina have become known in which such a mode of infection, viz. from a human source, can most probably be excluded. As an illustration of this category of milk scarlatina may be mentioned the outbreak of scarlet fever in Oxford in the spring of 1882, recorded by Dr. Darbishire, in the 'St. Bartholomew's Hospital Reports,' vol. xx. The substance of Dr. Darbishire's report is this:—Three cows were kept by those who sold the milk, and nine houses, containing eighty-five persons in all, were supplied morning and evening; the milk was never stored, as there was generally barely enough at each milking for all the customers. In the house to which the cows and paddock belonged there was a case of diphtheria in a young lady; she was removed to the infirmary on March 1. The cowman had a child ill with scarlet fever in his cottage from February 27 till March 8. On March 8 Dr. Darbishire had this child removed to the hospital and the cowman's cottage thoroughly disinfected; the cowman left his cottage to sleep in lodgings near, the care of the cows having been handed over to another man, engaged for that purpose. Now, if the milk had become infected from either of these two cases (one diphtheria and the other scarlet fever), this must have occurred for the first before March 1, for the other before March 8; and as the period of incubation of scarlet fever is known to be, as a rule, less than seven days, it follows that March 8 being the last day on which the milk could have received the contagium from a human being, March 10 would be the last day on which scarlet fever could have been produced by that milk, and the majority of cases of scarlet fever would have occurred before that day, as one cannot assume that in all these cases the period of incubation would be protracted to such length as seven days. But mark what really did happen. Dr. Darbishire states that no case occurred till March 10, on which day two cases of sore throat and one case of scarlet fever occurred; on March 11, one case of sore throat; on March 12, two cases of sore throat and one of scarlet fever; on March 13, four cases of sore throat and two of scarlet fever; on March 15, one case of sore throat and one of scarlet fever; on March 16, two cases of sore throat and one of diphtheria; on March 17, one case of sore throat; on March 18, one case of sore throat. Now, all these cases were proved by Dr. Darbishire to have been caused by that milk. There occurred subsequently other cases, but these were shown to have been due to secondary infection from person to person. This is a good illustration of a milk epidemic in which the milk most probably was not fouled by human agency; and there are other milk epidemics which on analysis of dates lead to the same conclusion.

Then a third group of milk scarlatinas is known, in which infection from a human source could be definitely excluded; as an illustration of this Mr. Power's report in 1882 on an epidemic outbreak of scarlet fever in St. Giles and St. Pancras may serve. 'The disease was distributed with a milk service derived from a Surrey farm. In this case two facts could be affirmed: the one that a cow recently come into milk at this farm had been suffering from some ailment, seemingly from the time of her calving, of which loss of hair in patches was the most conspicuous manifestation; the other that there existed no discoverable means by which the milk which had coincided with scarlatina in its distribution could have received infective quality from the human subject.'

The Medical Department of the Local Government Board has from these facts drawn the conclusion that 'distrust must be placed on the universally accepted explanation that milk receives infective properties directly by human agencies,' and further that 'the question of risk from specific fouling of milk by particular cows suffering, whether recognised or not, from specific disease was seen to be arising.' This view received striking confirmation and proof by a report of an outbreak of scarlet fever that occurred at the end of 1885 and the beginning of 1886 in the north of London, and was investigated by Mr. Power. His report is published *in extenso* in the Report of the Medical Officer of the Local Government Board for 1886; and we will here give the substance of it. Mr. Wynter Blyth, Medical Officer of Health for Marylebone, had in December 1885 observed a sudden outbreak of scarlatina in his district to be associated with the distribution of milk coming from a farm at Hendon, and had found reason for believing that the disease had prevailed exclusively among customers furnished with milk from that source. Mr. Power, on a more extended inquiry, found that a similar prevalence of scarlatina had occurred about the same time in other parishes in and near the metropolis which were furnished with milk from the same farm. After careful inquiry Mr. Power could with certainty exclude any contamination of the milk from a human source, and say that nothing of the kind known as 'sanitary' conditions could have had any concern with the infectivity of the milk. Mr. Power showed conclusively that only certain sections of the milk supplies of this farm, and finally only certain cows from which these sections of milk were derived, had any relation to the observed results. 'In the end,' says the Medical Officer, 'he has demonstrated, beyond reasonable doubt, the dependence of the milk scarlatina, occurring from day to day during December, on a diseased condition of certain milch cows at the farm, a condition first introduced there in the previous month by some animals newly arrived from Derbyshire; and he finds strong circumstantial evidence for believing that the latter phenomena of this dependence were brought about through the extension of the diseased condition of one set of animals to another set, after the fashion of an infection.' Now, this disease as it presented itself in some of these Hendon cows consisted in the presence of sores and scurfiness in different parts of the skin, with loss of hair in patches, an eruption leading to the formation of a sore covered with dark brown scab or crust on the udder and teats, and a visceral disease, notably of the lungs, liver, kidney, and spleen, which, although milder in character, very much resembled the visceral lesions occurring in cases of human scarlet fever. By experiment it was shown that the matter of the eruption of the udder is possessed of infective power, inasmuch as on inoculation into the skin of calves the same eruption is reproduced.

The microscopic examination of fine sections through the eruption of the cow shows the following conditions:—

The corium throughout the whole extent of the eruption is infiltrated with round cells. This infiltration, though densest in the central portions of the ulcer, is sufficiently pronounced even in the peripheral parts, but it gradually fades away on passing from the ulcer to the normal skin. The infiltration in the deeper parts of the corium is limited to the vascular branches, but in the superficial parts is more diffuse, the papillæ becoming at the same time thicker. This thickening of the papillæ fades off towards the periphery of the ulcer. The most noteworthy changes are, however, present in the epithelium. In the peripheral portions of the diseased part there are present in the superficial layers of the stratum Malpighii close to the stratum lucidum, as also in the stratum lucidum itself, numerous cavities of different sizes.

These cavities lie closely side by side; the most superficial ones are either covered by the stratum lucidum, or extend between the layers of this stratum. The former cavities descend into the depth of the epithelium; at the very margin of the diseased part they are smallest, and they do not in depth comprise more than the superficial third of the stratum Malpighii. They gradually enlarge in depth as we pass from the periphery of the ulcer towards its centre; at its very centre they involve the whole thickness of the stratum Malpighii. At the same time it is to be noticed that, at the marginal parts, the cavities, although closely placed side by side, are well separated from one another by thicker or thinner trabeculæ composed of epithelium; while at or near the centre of the ulcer these trabeculæ get destroyed, and the cavities become confluent, and the covering layers of the cuticle having here also given way, their contents extend on to the free surface of the ulcer. These contents, which go to form what has been above mentioned as the crust, thus gradually spread over the surface, not only of the centre, where the stratum lucidum has become lost, but also over the rest of the ulcer. In the marginal portions, i.e. where the superficial layers of the cuticle are still present as cover of the above cavities, this layer (i.e. the stratum lucidum) separates the contents of the cavities from the crust. The contents of these cavities consist (a) of an albuminous fluid, looking, in hardened sections, uniformly granular, or containing also fibrinous threads; (b) of a few red blood-corpuscles; and (c) chiefly of round cells or pus cells, the nuclei of which, near to, and on the surface, gradually break up into amorphous granular matter.

In the central parts of the ulcer the whole exudation undergoes degeneration into *débris*, not only in its superficial, but also in its deeper portions. While some cavities contain very few cells, and are filled chiefly with albuminous fluid (granular or fibrinous), others are almost entirely filled with pus cells closely packed together. In the papillæ near the cavities the blood-vessels are engorged and there is also escape of red blood discs.

On a careful examination it is evident that the origin of these cavities is in enlargement of and exudation into the tissue of the papillæ, but only of those portions nearest to the stratum lucidum, and hence arises the formation of cavities in the cuticle. The whole anatomical details of the distribution and arrangement of these cavities vividly recall the conditions observed in the vesicles of cow-pock and of sheep-pock, and on comparing under a low power of the microscope a section through a sheep-pock with a section through the eruption of the cow now under consideration, the similarity is very striking indeed.

There are, however, anatomical differences between the two diseases. The infiltration of the corium is slighter in the cow eruption than in the sheep-pock, and in the cow-ulcer the cavities are situated in a more superficial stratum of the epidermis.

There is in the disease we are now considering a good deal of infiltration of the epithelium by round cells derived from the cavities, not only into the stratum Malpighii, but also, and particularly in the marginal parts, into the cuticle; the round cells burrowing in great numbers between the scales of this stratum, and ultimately reaching the free surface to join those of the crust.

Fine sections made through the ulcer artificially induced by inoculation in the ear of a calf proved its complete identity in anatomical respects with the ulcer in the cow. The infiltration of the superficial corium; the formation of cavities, filled with exudation cells and fluid, in the superficial layers of the epithelium, and particularly between the layers of the cuticle; the

final destruction, in the centre of the eruption, of the covering cuticle ; and the extension of the exudation over the free surface to form the crust, are the same in both instances.

The microscopic examination of the internal organs reveals facts as follows :—

In the Lung.—Sections made through portions containing much congested lobules show not only great congestion of the blood-vessels, large and small, but a large amount of hæmorrhage, blood in substance being present in the air vesicles and infundibula, in the lymph spaces of the interlobular septa, and in the tissue and lymphatics of the pleura. In the latter membrane numerous diplococci are to be met with. Here and there the same diplococci occur in the alveolar wall and in the tissue of the interlobular septa.

Sections through the *liver* show a great deal of change. Under the capsule, as well as in the substance of the liver, there occur, in connection with the interlobular branches of the portal vein, larger and smaller foci of inflammation, consisting in the presence of numerous round cells. Some of these foci are several millimetres in diameter, others are much smaller. From the interlobular tissue the inflammation extends into the lobules between the liver cells. The liver cells of these lobules involved in the inflammatory process are swollen up, and many of them are undergoing disintegration. In some of these foci, particularly those situated in the vicinity of the capsule, the round cells are so much crowded that given foci look almost like miliary abscesses. The blood-vessels are much distended and filled with blood.

Numerous diplococci and short coccus-chains occur in the parts surrounding the inflammatory foci. These are particularly numerous near the capsule in the vicinity of inflamed parts.

Sections through the *kidney* show well-marked glomerulo-nephritis ; infiltration of the sheath of the cortical arterioles with numerous round cells ; the epithelium of the convoluted tubules swollen, opaque, and in many places disintegrating.

Since this Hendon outbreak of ulcerative disease on the teats and udder of milch cows, connecting the milk with the outbreak of human scarlatina, several such outbreaks have become known and investigated : one was described by Dr. Russell, Health Officer in Glasgow, who traced the infected milk to cows which were affected with an ulcerative disease of the skin of the teats and udder ; and the writer has become acquainted with one in the Camberwell district, London, where the scarlet fever was traced to milk coming from a particular dairy, in which at least one cow, at the time of the investigation (May 1888), was affected with sores on the teats. Calves were inoculated with matter (scrapings and scab) of such a sore, and it was conclusively proved that we had to deal with a contagious disease communicable to the skin of the calves, and leading on inoculation to the formation of ulcers of exactly the same kind as were produced in calves (in 1886) inoculated with matter from the sores of the Hendon cows.¹

Now, the Agricultural Department of the Privy Council had in 1887 and 1888 taken an immense amount of trouble in the matter and came to the conclusion that the contention of the Medical Department of the Local Government Board, as to a direct connection existing between eruptive

¹ An extensive outbreak of scarlatina occurred simultaneously in several districts of the south of London between March 28 and the first week of April, 1892 ; all the cases of this outbreak were conclusively traced to milk coming from one particular dairy farm, A, at New Cross. As a most instructive natural experiment performed during this outbreak the following may be mentioned :—A milkman about Chislehurst, in whose district no cases of scarlet fever had occurred previous to March 28, had on that day

disease of the cow and human scarlatina, was not correct; that there exist contagious eruptive diseases of the cow which are not connected with scarlet fever in man; and on these grounds they formulated a direct negative to the contention of the Medical Department of the Local Government Board. Various deputies of the Agricultural Department found outbreaks of eruptive disease of the teats and udders of cows in various parts of the country, which disease, through the milk of those cows, had not given rise to scarlet fever; this disease was maintained by them to be identical with the Hendon disease, which had been connected with human scarlatina, although they had no direct knowledge of the Hendon disease, and judged merely by the fact that in the Hendon disease there was ulceration on the teats and udder. Dr. Crookshank made very definite assertions in this matter. The Wiltshire disease, which Crookshank maintained to be the true Jennerian cow-pox, was an eruptive disease that served the Agricultural Department as model for almost every eruptive disease of the cow, but forgetting at the same time that, though all eruptive diseases on the teats and udder of the cow may be similar in some, they are nevertheless dissimilar in other respects, and forgetting that it is not in the points in which they are similar, but in those in which they are dissimilar, that a differentiation amongst them is possible.

All the eruptive diseases on the cow's udder have this in common amongst themselves, that the eruption commences as a papule on an injected skin; that this papule while enlarging changes into, now a distinct, now a less distinct vesicle; that this vesicle, when formed, contains lymph which is at first clear and then becomes purulent; further, that the eruption becomes covered with a dark crust underneath which is a sore of the cutis; the crust thickens and ultimately becomes loose and falls off, the sore becoming dry, contracting, and healing. As said just now, all eruptive diseases in the cow have, speaking roughly, those points in common, and in Jenner's time and afterwards were all grouped into two great classes, viz. true and false cow-pox; and even to the present time dairymen, farmers, and veterinary surgeons in this country know only of true and false cow-pox.

The Agricultural Department declared that all contagious eruptions of the teats and udder in recent years were cow-pox, pure and simple, and therefore that the Hendon disease described by us was cow-pox, the same as the Wiltshire disease. When, however, the different eruptions are more carefully studied, it is found that they differ: (a) in the progress and duration of the eruption; (b) in the nature of the infiltration of the corium; (c) in the rapidity with which they heal; (d) in the results of inoculation into calves; (e) in the question whether transferable to the hand of the milker or not; and (f) lastly, but not least, as to whether the disease is simply a local disease, or whether it is associated with constitutional and visceral malady. Now, the Wiltshire cow eruption was possibly, one might even say probably, cow-pox, but the Hendon disease was certainly not; here the eruption ran a much shorter course, the infiltration of the corium was more pro-

ceeded to his usual milk supply, owing to this being shorter than usual, a certain amount of milk from the dairy A of New Cross. A few days after (evenings of March 29 and 30) numerous cases of scarlet fever occurred almost simultaneously amongst the families in Chislehurst and Bexley supplied with this mixed milk. Now, in this dairy farm A at New Cross, fouling of the milk from a human source could with certainty be excluded; but a few days previous to the outbreak of scarlatina one cow had been ill; it was quiet, off food, coughed, its milk was ropy, and when seen about April 6 had two sores on the udder, covered with brown crusts; the sores were contracting and in a state of healing. Four other cows milked by the same hand had also sores covered with scabs on their udders.

nounced, the duration of the crust stage was considerably shorter, and the healing proceeded much more rapidly. The result of inoculation of calves was also different: the disease was not communicated to the milker's hands, and, as was shown above, there was distinct disease of the viscera.

The writer has shown that in the ulcers of the Hendon cows, and in the diseased portion of the viscera of these cows, there occurs a streptococcus that occurs also in human scarlatina (in the skin, in the blood), and which was called the streptococcus scarlatinæ. This streptococcus in culture and in morphological respects resembles other streptococci, amongst them the streptococcus pyogenes; but notwithstanding the numerous assertions to the contrary—that all or most streptococci are streptococcus pyogenes—our streptococcus scarlatinæ is by careful study capable of being distinguished from the streptococcus pyogenes. Experiments by feeding or inoculation made on calves and mice, made with the streptococcus scarlatinæ derived from the sores of the Hendon cows or of human scarlatina, produced definite general infection, and from such animals the microbe was recovered by cultivation: results which cannot be obtained with the streptococcus pyogenes.

More than that, by inoculation of six milch cows with the human streptococcus scarlatinæ, a definite eruptive disease was produced on the skin generally, and on the teats and udder particularly, associated with visceral disease identical in every respect with the cutaneous and visceral disease observed in the Hendon cows. It is, we consider, proved hereby that the streptococcus derived from human scarlet-fever cases, producing as it did the Hendon disease in the cow, i.e. the same disease which was the source of infection in the human subjects in the Marylebone epidemic, must be considered as the microbe of scarlet fever.

Professor Axe, Dr. Thin, and also Dr. Crookshank, have made certain assertions—accepted by the head of the Agricultural Department—as to the milk at the Hendon farm having been really infected not by the cows but by a boy who carried the contagium from a human case of scarlatina to the farm, the boy himself supposed to be free of the disease. But no such boy could have infected the Hendon milk; besides, the milk of that Hendon farm, as in most instances in which the cow disease is the cause of the infectivity of the milk, had infective power from day to day for weeks (compare an article in the 'Practitioner,' March 1889).

It was asserted by Professor Axe and Dr. Crookshank that the Hendon disease could not have been other than the Wiltshire disease, i.e. cow-pox, because at the time of the Hendon disease, and afterwards, there existed in different parts of the country an ulcerative eruptive disease of cows which was communicable to the milker's hands as vaccinia, and of course was not connected with scarlet fever; indeed, the head of the Agricultural Department classed all the eruptive diseases together as the same, being probably cow-pox. This was strikingly illustrated in Dr. Russell's case in Glasgow; here the scarlet fever epidemic was traced to milk coming from a dairy farm where cows were affected with an eruptive ulcerative disease on the teats and udder. Professor Brown submitted to Dr. Russell drawings of ulcers on the udder and teats of cows as the Hendon disease, which were not drawings of what was understood as the Hendon disease, but of the Wiltshire disease. It need hardly be said that drawings of ulcers on the teats and udder of cows all look very much alike.

But though perfectly familiar as the Agricultural Department and the veterinary profession in general seemed to have proved themselves to be with the fact that all eruptive diseases of the teats and udder of the cow then prevailing in England (1885–1888) are one and the same, there occurred an

outbreak of an eruptive disease amongst cows in Edinburgh (in 1887), which seemed to offer some differences and to make some rupture in that accepted uniformity of eruptive diseases.

During 1887 an eruptive disease appeared amongst certain cows in a dairy farm in Edinburgh, the milk of which had distributed amongst the consumers (in an educational establishment in that city) a form of febrile sore throat, which could not be recognised as either scarlatinal or diphtheritic. Dr. Sims Woodhead and Mr. T. M. Cotterill clearly proved the connection of this malady with the milk of that dairy farm; more than that, on the suspension of the consumption of the milk the epidemic quickly disappeared. When the use of the milk was recommenced sore throats of a similar character appeared again. Then all the milk was boiled before use, when the epidemic similarly yielded ('British Medical Journal,' June 9, 1888). Now the writer had the opportunity of receiving one of those cows, and he ascertained that the nature, progress, and duration of the ulceration on the teats and udder were quite unlike those of the Wiltshire malady, but similar to, though slightly different from, those observed in the Hendon disease. Inoculations of calves with the matter from the cow's sores yielded positive results. There was present in that cow a definite visceral disease (lung and liver). (Report of the Medical Officer of the Local Government Board for 1888.) From the ulcers of this cow a streptococcus was cultivated which also proved different from the streptococcus scarlatinae; it was in culture very similar to the streptococcus pyogenes; it did not produce any specific result on inoculation into rodents, but on cutaneous inoculation into calves it produced strikingly positive results—ulceration and crusts of the same character as those produced directly by cutaneous inoculation of the ulcer material of the cow. Here, then, we had a further eruptive disease in the cow, differing from the Wiltshire disease and also from the Hendon disease, and connected by means of the milk of the cow with febrile sore throat in the human subject; there occurred no direct infection of the hands of the milkers, as in the Wiltshire disease. The above positive experiments on calves prove, we think, that this streptococcus was the microbe of the Edinburgh disease, and was different from the streptococcus pyogenes on the one hand, and from the streptococcus scarlatinae on the other.

It was instructive to learn from a report by Professor Macfadyean, of Edinburgh, to the head of the Agricultural Department of the Privy Council that the Edinburgh cow disease was a different disease from the Wiltshire disease. But though the disease went on spreading amongst the cows, the milk of such cows was not consumed, or when consumed was first boiled and the throat epidemic ceased (*see* 'Practitioner,' March 1889).

In the next chapter a further eruptive contagious disease on the udder of cows will be described, but which in its progress and duration was no more, certainly much less, cow-pox than the Hendon disease, and which in its causation was no other than diphtheria, since it was artificially produced by subcutaneous injection of culture of the bacillus diphtheriae.

In typhus fever, whooping cough, measles, rubeola, varicella, and Oriental plague, no definite microbes have as yet been demonstrated; in whooping cough, micrococci, moulds, and bacilli have been asserted to be associated with the disease, but no real satisfactory evidence is forthcoming; in measles, in Oriental plague, in yellow fever, and other specific diseases, micrococci are often met with and have been mentioned by many observers.

So also in the case of Beriberi; while some observers (Lander, Ogston) maintain that bacilli occur in the blood, and regard them as the microbe of the disease, others (Pekelharing and Winkler) claim micrococci in the blood as the real cause. Rinderpest is maintained by Metschnikoff and Gamaleia to be caused by a bacillus in some morphological respects similar to anthrax bacillus; these observers maintain that they have found

the bacilli in the ulcers of the stomach and also in the blood of the affected animals. Further, they have cultivated the microbe and found it to grow on gelatine in a manner similar to the typhoid bacillus. Lastly, they assert that they have reproduced the disease in calves and guineapigs by inoculation of such cultures.

CHAPTER XIX

DIPHTHERIA

THIS acute infectious disease, to which children and young individuals are particularly prone, shows itself in most instances as a severe inflammation and fibrinous infiltration of the mucous membrane of the fauces and pharynx, or also the larynx and trachea, leading to, and early in the disease consisting in, a necrosis of the superficial part of the mucous membrane, and thereby changing this into a tenacious, whitish pseudo-membrane, the 'diphtheritic membrane.' In most cases only the mucosa of the fauces (tonsils, palatine arches, velum palati and uvula, upper part of pharynx) shows this change, i.e. into whitish grey 'diphtheritic membranes;' in other cases this necrotic change extends over the whole of the pharynx into the larynx, and even the trachea; in still other cases it starts in the larynx and invades this and the trachea—croup. In some cases a similar inflammation and the formation of diphtheritic membranes are observed in the stomach, in the intestine, in the urinary organs, and independently and primarily on wounds. In addition are to be mentioned myocarditis diphtheritica. The microscopic character of diphtheritic inflammation is generally this, that the mucous membrane is the seat of a severe inflammation and necrosis: engorgement of, and extravasation from, the superficial capillaries and veins, with stasis of blood in them, and swelling due to infiltration of the mucosa with fibrine and round cells; the epithelium as a whole is lost, the affected mucosa itself becomes necrosed and changed into a whitish-grey coagulated mass, in which fibrin, a close network of threads and septa, and in the superficial parts, lymph cells, may be recognised, this necrosed or coagulated portion forming the diphtheritic membrane; close to that part which comprises the necrosed mucosa, the outlines of blood-vessels filled with stagnated and coagulated blood, and extravasated blood, as also dense infiltration with lymph cells, may be recognised. When the process continues into the depth and breadth, this inflamed portion also becomes necrosed, and a part of the diphtheritic membrane. After the process passes the acme, the inflamed tissue, not necrosed by the exudation, gradually detaches the diphtheritic membrane above it, and an ulcer is left behind, which, like other healing ulcers, gradually contracts and becomes covered with healthy membrane and epithelium.

A section through a diphtheritic membrane shows a few nuclei in a dense, more or less fibrinous reticulated or hyaline matrix, more or less ill preserved; some of these take the staining, i.e. are not dead; in others already dead, the outlines can barely be recognised. In the superficial parts of the diphtheritic membranes a number of larger or smaller loculi are always seen, which are filled with clumps of bacteria (*see illustration*). These clumps of bacteria are of various kinds: staphylococci; streptococci, but not so common; thick and long septic bacilli, and groups of minute bacilli which we will call the diphtheria bacilli. These latter are found in larger and smaller masses on the surface, forming sometimes a continuous layer; in some cases sections show that in the middle, and occasionally, but rarely,

even in the deep parts, they are the only bacteria present; here they are in small clusters, or they form large masses (*see* fig. 78). In the mucous membrane next to, but not part of, the diphtheritic membrane, the writer has found them occasionally in small numbers; in the inflamed mucous membrane of the depth these diphtheria bacilli are, as a rule, rarely to be found. In the blood and in the viscera the bacilli are absent; nor are other micro-organisms to be found as constant inhabitants. In cases of diphtheria ending fatally, even if the disease only lasted a few days, the lungs are the seat of severe bronchial catarrh, lobular or broncho-pneumonia; the kidney is congested and shows distinct parenchymatous nephritis, the epithelium of many convoluted tubes of the cortex is granular, disintegrating and fatty; in the liver, fatty degeneration of the liver cells is generally present. As to bacteria of diphtheria there are many assertions.¹ The one species that is constant and can be easily isolated in many cases in almost pure cultivation from the superficial and even middle layers of the fresh diphtheritic membrane are minute bacilli (*see* figs. 74, 75): some are curved, most are straight, some slightly swollen at each end, or knob-shaped at one end, many of them pointed at one end; in fact, this may be regarded as the typical bacillus. These bacilli occur either singly or in dumb-bells; many show a segregation of their protoplasm into granules or rods of unequal size; amongst these 'granular' forms, one or both terminal granules are occasionally club-shaped. Some of the single bacilli in well-stained specimens show a deeply stained granule at each end. The bacilli of agar cultures show the same appearances as those in the diphtheritic membrane; in gelatine culture the bacilli are shorter, thicker, and many are conical (*see* fig. 76). These bacilli were first seen by Klebs, and by Löffler were regarded, owing to their constancy, as pathognomonic and pathogenic for diphtheria; Löffler had first isolated them by culture on blood serum, but he, and then Hoffmann, found a morphologically similar bacillus in the normal discharges of the fauces. Now, Löffler maintained that, while the former or the 'diphtheria bacillus' is pathogenic for animals, the latter or pseudo-diphtheria bacillus is not so; but this has not been accepted by all, though, as we shall see, it nevertheless corresponds to the facts.

Besides, in true diphtheria of the fauces, the diphtheria bacilli can be demonstrated in many cases of croup, and in diphtheria *following* scarlatina, but not in so-called scarlatinal diphtheria, that is, in necrotic change in the fauces occurring simultaneously with scarlatina. (Löffler, Kolisko and Palttauf, Tangl, Klein.)

The bacillus of diphtheria isolated by Löffler forms colonies of definite characters on serum and agar plates kept at 85–87° C.: round white colonies, thickest in the middle and gradually assuming here a yellowish-brown tint. According to Löffler it does not grow on gelatine at temperatures below 20° C., but the writer has obtained cultures even at 19° C. on gelatine; on potato it shows no visible growth. Löffler found this particular bacillus in a large percentage, but not in all of the diphtheritic membranes; Kolisko and Palttauf, Roux and Yersin, Zarniko and Escherich, found this microbe in all cases of diphtheria, and owing to its peculiar pathogenic action (*see* later) they definitely regarded it as the microbe of diphtheria. The writer has shown that there occur in diphtheritic membranes two species of bacilli, identical in morphological respects and in the mode of growth on and in agar plates, on serum, and on potato; but one species is not constant, and is probably the pseudo-diphtheria bacillus of Löffler, while the other is

¹ Oertel, Nassiloff, Wood and Formad, and others as to micrococci; Von Emmerich as to short bacilli; Prudden as to streptococci.

constant in large numbers in all diphtheritic membranes; this second species is the one which is pathogenic, and is the diphtheria bacillus of Löffler, while the first one is not pathogenic. There is an important cultural difference between the two species; it is this: the true diphtheria bacillus grows well in and on gelatine at 19–21° C., the other does not grow on this medium below 22° C. Besides, the true diphtheria bacillus grows very rapidly in broth, making the broth (at 37° C.) uniformly and distinctly turbid in twenty-four hours. We shall therefore consider this as the Klebs-Löffler or true diphtheria bacillus, and it only remains to be added that on gelatine the bacillus can be easily isolated from the fresh diphtheritic membrane in numerous colonies; a particle of the membrane is first well washed and shaken successively with two or three successive quantities of sterile salt solution; from the last plate cultivations are then established (*see illustrations*).

On gelatine the colonies are at first rounded, white, prominent dots, which broadening, thicken in the middle and here become slightly yellowish in reflected, dark brown in transmitted light, the peripheral part being thin, plate-like, and angular; in streak cultivation on gelatine the streak becomes marked as a white band, at first made up of droplets, but soon becoming confluent into a uniform band; at the margin the droplets and knob-like expansions can still be recognised; the middle is thick and prominent; in stab culture in gelatine the stab becomes indicated by a line of droplets, white in reflected, brownish in transmitted light; the upper point of the stab is occupied by a crenate convex white plate. Of course on gelatine, at 19–21° C., the growth is much slower than on agar at 35–37° C. In milk kept at 20° C. our bacillus grows luxuriantly and produces after three days, or even less, slight curdling of the milk, and minute flakes of coagulated casein; at 37° C. the growth is curiously less abundant in the same space of time, and the curdling far less. The diphtheria bacilli are killed by heating to 60° C. for five minutes.

Löffler maintains that with cultures of the diphtheria bacillus, definite pathological results—inflammation with something like diphtheritic necrotic membrane—can be obtained by rubbing them into an abraded surface of the mucous membrane (mouth, trachea) of rabbits, fowls, pigeons, and Roux and Yersin make the same assertion; but such results are not obtainable either with human diphtheritic membranes, or with the cultures of the diphtheria bacillus; neither in the rabbit nor fowl, pigeon nor guineapig, in the mucous membrane or the skin has the writer been able to produce any result that at all resembles diphtheria. If occasionally a sort of inflammation is obtained, this is only after severe application, but it certainly does not yield anything like a diphtheritic membrane. But on subcutaneous inoculation of guineapigs with diphtheritic membrane, and particularly with cultures of the bacillus diphtheriæ, definite results are obtained which coincide with those described by Löffler, Zarniko, Escherich, Roux and Yersin, and others.

After subcutaneous inoculation with cultures, a few days old, the result is very rapid, and more striking than with diphtheritic membrane; to obtain acute results only a small particle, not more than can be removed from a colony with the point of a platinum wire, often suffices. In the severe cases produced by injecting several minims of a recent broth culture, the animals are very quiet after from twelve or sixteen hours; and a soft painful swelling is found at the seat of inoculation. During the second day the hair is erect, the eyes small, and the temperature is raised; the animals are shaky and refuse food; the condition grows rapidly worse, movement ceases, the body temperature rapidly falls, and the animals are found dead in some

thirty to forty hours. In other cases the illness lasts two to three days; in still others as long as five days, or even more. The younger the culture, the more active it is, and the more bacilli are injected, the shorter the illness. On *post-mortem* examination we find hæmorrhage and œdema in and about the place of inoculation, in the subcutaneous and muscular tissues, extending sometimes over considerable areas; when inoculation is made into the groin the changes (hæmorrhage and œdema) extend over the thigh, abdomen, and even chest of the inoculated side; the inguinal glands of the inoculated side are deeply congested. The lungs are more or less congested, sometimes the greater part of one lobe or another is deep purple; pleuritis and pericarditis are often found; the liver is slightly or not at all congested, or is even pale; the spleen is not enlarged; the serous covering of the stomach and intestines is congested; the suprarenals are deep red; the kidney is congested in the medullary part. Neither from the heart's blood nor from the lung, liver, spleen, or kidney can any organisms be cultivated; from the subcutaneous tissue of the inoculated part, particularly from the congested inguinal glands, the writer has obtained the bacilli in pure cultivations, some tubes showing a limited, others an abundant number of colonies; but the culture test is not successful in all animals, though it is in most. Broth cultures (at 35–37° C.) twenty-four to forty-eight hours old, act very virulently, as has been already shown by Roux and Yersin,¹ who have separated certain chemical products from such cultures, and shown that those products themselves act poisonously in proportion to the amounts injected. Roux and Yersin have observed in experimental animals, after inoculation with small doses of broth culture or of the chemical products separated from broth cultures, the same kind of paralysis as occurs also in human diphtheria in the later stages, that is, after the acute symptoms have passed away. While this paper was passing through the press, Dr. Sidney Martin published an account of the chemical nature of the poisons occurring in the human diphtheritic membrane: these same poisonous principles (ferment, organic acid, albumoses) were also obtained from albumen cultures of the diphtheria bacilli. Dr. Martin shows that with some of the chemical products diphtheritic paralysis can be produced, and he further shows that this paralysis is due to degeneration of the peripheral nerves. (Reports of the Medical Officer of the Local Government Board, 1891–1892). Löffler, Roux and Yersin, and others, have concluded that in diphtheria we have to deal with a chemical poisoning, the chemical poison being produced by the living bacilli in the diphtheritic membrane of the human mucous membrane, and in the case of the experimental guineapigs at the seat of inoculation, and absorbed by the system, producing the whole set of general disease symptoms in the lung, liver, kidney, and nervous system, associated with, and characterising diphtheria. The absence of the bacilli from the circulation and all affected organs and their localised presence in the diphtheritic membrane, suggest this. It follows that if the growth and multiplication of the bacilli in the diphtheritic membrane could be prevented or checked sufficiently early—by cautery or otherwise—the amount of the poison would be small, and the disease would cease.

It has been asserted by various authors that a necrotic, chronic, infective process, observed in the mucous membrane of the mouth and pharynx in calves and pigeons, is intimately connected with human diphtheria; but Löffler² has shown that this is not the case, since these necrotic processes are altogether different diseases, both as to pathology and as to the microbe.

¹ *Annales de l'Institut Pasteur*, I.

² *Mittheil. aus dem kais. Gesundheitsamte*, vol. ii., p. 482.

Cats, however, have unquestionably been observed¹ to show disease in connection with human diphtheria. In houses where human diphtheria obtained, cats have been known to become ill, either antecedently, or coincidentally, or subsequently; they appear to have some kind of throat illness, and cannot swallow; they sneeze and their eyes water; as a rule, bronchial mischief is noticed early, and if the disease is protracted through several weeks, as it sometimes is, they become much emaciated and die. On *post-mortem* examination the lungs are full of grey, consolidated, lobular patches, and the kidneys are always *enlarged and white*; on a section the whole cortex is found to be in a state of fatty degeneration, while the medulla shows congestion. Further, the writer has ascertained that an infectious disease with the same symptoms, and leading to the same result, exists naturally amongst cats; the animals have severe lung trouble, become emaciated, and die with the same pathological appearances, notably on the part of the kidney. In one case the writer has seen such a cat after several weeks' illness showing paresis of the hind extremities.

When cats are inoculated subcutaneously in the groin with a particle of human diphtheritic membrane, they become distinctly ill, show after twenty-four hours a painful swelling in the groin, have high temperature, and refuse food. In the severe cases these symptoms increase in intensity during the next few days, and the animals die before the end of the week. On *post-mortem* examination the subcutaneous and muscular tissues at and near the seat of inoculation are found to contain hæmorrhage and œdema, and the tissues are separated into layers, which are more or less necrotic. The viscera show much congestion, particularly the lungs, also the serous covering of the stomach and intestine as well as the peritoneum; the kidneys are *large and white*, the medulla congested, while the cortex is more or less uniformly fatty (*see fig. 187*). This condition is more marked the longer the illness; when the animals die in three to four weeks, or later, the condition of the kidney is very striking, and then also the lungs show lobular patches of grey consolidation. Still more striking is the result when a particle of a virulent culture of the bacillus diphtheriæ is subcutaneously inoculated. If a fresh culture—twenty-four to forty-eight hours old—is used, the animals are very ill in twenty-four hours: they are quiet, refuse food, the temperature is raised, and at the seat of inoculation is a painful swelling; some animals die after two, three, or four days, others live to the end of the week. On *post-mortem* examination the same appearances of the viscera, notably of the lungs and kidney, are found; and here also the fatty white kidney and the pneumonia are the more marked the longer the duration of the disease; in animals that die forty-eight to seventy-two hours after inoculation with culture, the subcutaneous and muscular tissues about the seat of inoculation show much hæmorrhage, in many parts the tissues are almost gangrenous. When using recent broth cultures derived from the congested inguinal lymph gland of an experimental guineapig (*see above*) the effect of the subcutaneous inoculation of such culture in the cat is most striking; all animals die within forty-eight to fifty hours. In many instances after subcutaneous inoculation, on the death of the animal, the bacillus diphtheriæ has been recovered by cultivation in numerous colonies, but no bacilli can be demonstrated in the lungs, liver, or kidney.

The only animal in which, either with diphtheritic membrane or with

¹ Dr. George Turner, Dr. Bruce Low, Dr. C. T. Benshaw, Dr. A. Downes, Dr. Thursfield; *see the writer's Report in the Volume of the Medical Officer of the Local Government Board, 1889, p. 162.*

cultures of the bacillus, it was possible to produce a definite and striking result on the cornea and conjunctiva, is the cat. If from the cornea and conjunctiva the superficial epithelium is scraped off, and then on such a surface a piece of diphtheritic membrane or a particle of an artificial culture of the bacillus is rubbed in, the result is in most instances positive; after twenty-four hours the conjunctiva looks injected, and the eye watery; after forty-eight hours the animal is quiet, and has the eye closed with much muco-purulent discharge from the inner angle of the eye; the conjunctiva is much congested, swollen, and œdematous; the cornea is opaque, and covered with a muco-purulent film; the condition gradually increases in intensity from day to day, so that by the end of the week the cornea shows a distinct ulcer, crater-like, its margin swollen and opaque. In severe cases the process gradually increases till the end of the third week, when the ulceration of the central part of the cornea reaches the lamina Descemeti; after this time the disease gradually abates, and recovery takes place. In two cases perforation of the cornea took place and purulent panophthalmitis set in. In other cases the duration of the disease is shorter and the process is not so intense. That we have here to deal with a specific infectious disease, is proved by the fact that matter scraped from the ulcerated cornea and transferred to the cornea and conjunctiva of a fresh cat produces again the same specific disease. Ordinary purulent matter applied to a scraped cornea or conjunctiva does not produce any result; after twenty-four hours the conjunctiva is found injected, and the eye watery, but after forty-eight hours nothing abnormal is to be noticed. From the opaque tissue bordering the ulcer in the cornea of the above successful cases the writer has succeeded in obtaining cultures of the bacillus, on gelatine, in numerous colonies; the cultural characters of the colonies were identical with those of the human diphtheritic membranes, but the bacilli themselves were conspicuously smaller in size, shorter and thinner (*see* fig. 78). Also by scraping the mucous membrane of the palate and fauces of the cat, and then rubbing in a particle of diphtheritic membrane, or of a culture of the bacillus, an ulcerative process is produced which reaches its height by the end of the first week, or earlier; the ulcer is surrounded by swollen congested mucous membrane, and is covered with a grey pyogenic membrane.

On account of the above very definite results obtained in the cat with diphtheritic membrane, or with cultures of the bacillus (subcutaneously or on the cornea), and of the peculiar disease of the lung and particularly the kidney, observed in the cat (experimentally or naturally) in connection with human diphtheria, the writer holds that the disease occurring naturally in this animal is equivalent to human diphtheria, and that therefore the cat must be considered as susceptible to human diphtheria, and capable of communicating the disease to other cats and also to human beings.

The important question that must be asked in connection with this assumption is this: Since in the natural disease in the cat there is no diphtheritic membrane to be found in the fauces, how is it possible for a cat to communicate diphtheria to human beings? Such has unquestionably taken place in several instances (*see* Reports of the Medical Officer of the Local Government Board, 1889-1890). A cat in the subcutaneous tissue of which, by inoculation of a particle of diphtheritic membrane, or of an artificial culture, local œdema, hæmorrhage, and necrosis are established at and about the seat of inoculation, contains in the local tumour the bacillus diphtheriæ; and it is here, just as in the case of the guineapig after subcutaneous inoculation, that in consequence of the growth and multiplication of the bacilli the chemical poison is produced which, absorbed by the

system, sets up the disease of the viscera mentioned above. Neither in these experimental cats, nor guineapigs, could the diphtheria bacilli be demonstrated in the diseased viscera or the blood of the circulation; neither in the case of the ulcer of the cornea and the conjunctivitis produced by inoculation into these tissues with human diphtheritic membrane or artificial culture could the diphtheria bacillus be demonstrated in the viscera. And the same we have seen to hold good for human diphtheritic process, i.e. the disease is in the first place a local one, and owing to the elaboration at that locality by the growing and multiplying diphtheria bacilli of certain chemical poisons is the general visceral disease established. But the important question is, Which is that primary locality in the cat affected with the natural disease in which the diphtheria bacilli grow and multiply and produce the chemical poison? From the fact that the animals early show lung disease and bronchitis, leading to broncho-pneumonia, and from the fact that the disease is communicated by them to other cats and to children (generally the children have been fondling and nursing the animals) we may, I think, justly conclude that the lungs are the locality which in the natural disease harbour the bacilli. In examining sections through the diseased portion of the lung of a cat affected with the natural disease, a condition is found to obtain in the large and small bronchi and in the infundibula, vividly recalling the diphtheritic membrane of the human subject; the bronchial mucous membrane is swollen and completely changed into a necrotic, reticulated mass in which the outlines of blood-vessels, distended by and filled with stagnated blood, and a few nuclei are recognisable, the cavity of the bronchi and infundibula being distended and filled with that diphtheritic mass. The writer has in two instances had an opportunity of examining the diseased lung of early cases, the animals dying a few days after they were seized with the illness. In these cases the presence of the diphtheria bacilli in considerable numbers could be demonstrated in the exudation filling the bronchi and infundibula in some of the lobules. But also indirectly he has been able to show that the lung in the cat is most probably the primary locality in which the diphtheria bacillus produces diphtheria, and where it elaborates the chemical poison. Of broth culture of the diphtheria bacillus forty-eight hours old a small quantity ($\frac{1}{2}$ -1 c.c.) was directly introduced into the cavity of the trachea without injuring the mucous membrane. The effect of this injection was most striking. Before twenty-four hours had elapsed the animals were found very ill; in one instance death took place in thirty hours. On *post-mortem* examination the lungs were found greatly congested, and there was also hæmorrhage; so also in the liver, the peritoneum, and the kidney. In the bronchial tubes there was much sanguineous exudation; and in this the diphtheria bacilli could be demonstrated in large numbers. In another case the animal lived till the sixth day; it did not feed, got thin, coughed a good deal, and appeared paralysed in one hind extremity. On *post-mortem* examination the whole middle lobe of one lung was found consolidated, was full of blood, and sank in water. On section through this consolidated part, the bronchi and infundibula and also air cells were distended by, and filled completely with an exact counterpart of diphtheritic membrane; also the exudation in the bronchi contained the diphtheria bacilli in smaller or larger clumps. Both kidneys in this case were greatly enlarged and white; in section the medulla was congested, but the entire cortex was white and in a complete state of fatty degeneration. From these experiments the conclusion is justified that, in cats, diphtheria in the lung is possible, and that most probably also in the natural disease affecting the cat, the primary diphtheritic locality is in the lung.

As is well established, diphtheria is a highly contagious disease, transmissible from person to person, its contagium belonging to the group called fixed contagia. But it is likewise well established that milk infected from a human source has, in several epidemics, been the means of producing diphtheria in the consumers. It is also known that a room in which a diphtheria case has once existed may for years harbour the contagium of diphtheria, so that any new-comer or inhabitant may contract the disease; moreover, it is known that in a locality in which diphtheria has once been rife, the disease may at any time reappear, and in these instances the transmission of the contagium from sewers is maintained by some sanitarians. Lastly, it has been shown by Mr. Power, Dr. Mason, and Dr. Philpott, that in certain epidemics of diphtheria (Yorktown and Camberley, Barking, Croydon), while the milk was the vehicle of infection, the milk did not receive its infective power from a human source.

Several epidemics of milk diphtheria, in which fouling of the milk with human diphtheritic material could not be demonstrated, but, on the other hand, could be excluded, have of late years become known, and in these cases the suspicion attached itself to the cows, for it could be shown that there existed on the farms concerned no other condition which in any way could account for the infectivity of the milk; besides, this infectivity was inherent to the milk over a certain period. In the case of the Yorktown and Camberley epidemic (*see* Mr. Power's Report in the volume of the Medical Officer of the Local Government Board for 1886), the cows were certified by a veterinary surgeon to have been in good health, though even several days after the human diphtheria cases had ceased to occur, two of the cows showed some slight signs of 'chaps' on their teats. Mr. Power saw at the farm one cow which had suffered from chapped teats in October 1886 (the month in which the epidemic occurred), and which still had at the beginning of November a scab or crust at the site of a 'chap.' At Barking the cows whose milk produced the diphtheria (in 1888) suffered from a distinctly contagious eruptive disease on the teats and udder, showing itself in sores covered with brown black crusts. The same was noticed in connection with an outbreak of diphtheria (through milk) at Croydon in November 1890. The question which was therefore considered important to decide was this: Can cows be infected with the bacillus diphtheriæ? During the early part of 1890 and 1891 the writer made experiments on milch cows (which had calved some weeks previously), which strikingly showed that this is really the case. The results of these experiments are so definite and so important in connection with milk derived from such cows being charged with the diphtheria contagium, that the writer may be excused for giving two of these experiments somewhat in detail.¹

A broth culture was made of the bacillus diphtheriæ derived from a human diphtheritic membrane, but passed through several gelatine subcultures; the broth culture had been growing for a few days at 37° C.

One cubic centimetre of the culture was injected under the skin into the subcutaneous tissue of the left shoulder in each of two cows. These animals were, at the time of the experiment, in very fine condition (teats and udder quite clean, copious milk secretion), and had been so during eight to ten days, during which they had been under observation. During the second and third days after inoculation, the body temperature showed a slight rise (to 40°·6), and they did not feed well on those two days; but afterwards the temperature went down to the normal state, and the animals recovered. But at the seat of the inoculation there was a painful, large soft tumour

¹ Report of the Medical Officer of the Local Government Board for 1889, p. 168.

to be felt and seen. On the fifth to the sixth day, for the first time, there was noticed on the udder and on one teat in one cow an eruption of about half a dozen firm papules: red and injected, projecting above the surface of the skin, the subcutaneous tissue indurated with a nodule. In addition to the papules about half a dozen vesicles and two round patches covered with brown crusts could be seen on the udder. Some of the vesicles contained clear lymph, others were pustular, i.e. purulent. On the seventh day new papules and vesicles were found; those of the previous day had already become changed into dark brown crusts. On the eighth day a new crop of vesicles could be noticed on this cow's udder, and on that day for the first time about half a dozen were also seen on the udder of the second cow. In fig. 136 the condition is accurately represented: some are vesicular, others pustular, and still others covered with brown black crusts; the vesicles and pustules were round and prominent, with a narrow margin of injected skin, the crusted places irregular. The whole thickness of the skin and subcutaneous tissue felt hard and nodular. For two or three days (ninth to twelfth day) this went on in the first cow; that is, new vesicles appeared: those that were vesicles with clear lymph one day were pustular the next, and crusted the following day. The crusts did not remain long; after two or three days they became loose, and left a dry healing sore behind, which when recent, on removal, showed a bleeding sore of the corium underneath.

We have, then, here a new eruptive disease on the teats and udder of the cow: a disease marked by papule, vesicle, pustule, sore, and crust, but of a very rapid progress, since the crusts fell off and the sore healed in less than seven to nine days since its first appearance, the skin being at the same time much indurated. This eruptive disease on the udder, be it well observed, was produced by inoculating the animals subcutaneously in the region of the left shoulder with a culture of the bacillus diphtheriæ.

As stated above, in both animals on the second and third days there was a painful, soft tumour to be felt at the seat of inoculation. From day to day the tumour became larger; about the end of the week it was as large as a man's fist; after this time it gradually became firm; but about the fifth and sixth days it was still soft, felt like œdema, and on pressure a quantity of clear serum could be squeezed out from it. After the death of the animals (one died after a fortnight, the other was killed on the twenty-fifth day) the tumour was examined, and it was found to be located in the subcutaneous tissue, but was firmly connected both to the skin above and the muscular tissue below, and was surrounded by œdematous tissue. It presented white streaks, was firm, but on section clear serum could be pressed out from it. Under the microscope the tissue of the tumour was of the same nature as diphtheritic material; a general matrix of reticulated necrotic tissue in which remnants of nuclei, outlines of blood-vessels, and remnants of extravasated blood could be recognised: this tissue shaded gradually both into the cutis and into the surrounding muscles.

Both animals showed normal temperature to the end, but they both coughed and gradually ceased feeding, and did not take any water: one of them by the end of the fortnight suddenly became worse: it took no food or water, its milk failed, its evacuations became scanty and dry, its breathing became very rapid, and after a sudden collapse it died. The other animal twenty-four days after inoculation grew much worse, and was therefore killed.

In both animals the lymph glands nearest the left shoulder, i.e. close to the tumour, were much enlarged, very œdematous, and contained hæmorrhage;

no change in the organs of the throat; both lungs showed extensive congestion, in fact, almost amounting to red hepatisation of the upper lobes and the upper portion of the middle lobe, petechiæ, and hæmorrhagic patches under the pleura; the pleural lymphatics everywhere in the congested portions were conspicuous and distended, either with clear lymph or, as was the case in the second cow, tinged with blood. On cutting into the congested portions the lung was seen to be highly cedematous, a large quantity of blood-tinged serum flowing from, and accumulating at the cut end; the lobules were well mapped out, and there was also sharp demarcation by cedematous connective tissue between the normal lung tissue and the deeply congested lobules, as also between groups of lobules and individual lobules in the congested areas; there were hæmorrhagic spots and patches on the parietal and visceral pericardium. The liver showed yellow-grey necrotic patches, the spleen grey necrotic streaks in the capsule; both kidneys showed congestion of the medulla, and fatty patches in the cortex. We have, then, in both these animals a striking result, completely coinciding with the disease in the cat.

The next important point ascertained in these cows had reference to the distribution of the diphtheria bacilli inoculated. In the tissue of the tumour of both animals after death, i.e. after fourteen and twenty-four days respectively, the diphtheria bacilli could be demonstrated without any difficulty under the microscope in the sections, and by culture. On sections the necrotic tissue of the tumour contained large numbers of the bacilli in clumps; culture experiments on gelatine and on agar with a particle of the tissue of the firm tumour produced innumerable colonies of the diphtheria bacillus; when examined under the microscope they resembled the human diphtheria bacillus in all respects (fig. 77). They were also tested on guineapigs and found to act extremely virulently, causing death of the animals with typical appearances in thirty to fifty hours. But neither in the heart's blood, nor in the lung or liver of these cows, could any microbes be demonstrated in microscopic specimens or by culture. So far, then, there is complete analogy between the cows, guineapigs, and cats, that is to say, the diphtheria bacilli introduced into the subcutaneous tissue produce here by growth and multiplication the chemical poison, setting up the general disease in the viscera. But it will be asked, Is the remarkable eruption on the udder of the cows also a result of the absorption of the chemical poison produced at the seat of inoculation (subcutaneous tissue of the left shoulder)? The nature and progress of the eruption, it must be admitted, had all the characters of what one would be inclined *a priori* to consider under other conditions as an infectious process; and if so, it most probably is not merely a symptom of the work of the chemical poison, though it might be, since, for instance, in certain chemical poisonings, e.g. in bromide, a 'bromide rash' in the skin, even resembling vaccinia to a certain extent, is well known. That in these cows the eruption was not of this nature was directly proved by inoculating, into the subcutaneous incisions in the groin and abdomen of calves, matter taken from the eruption of the cows in the vesicular and pustular stages. The result of this inoculation was that the typical eruption of the cow was reproduced, though its appearance in the calves was somewhat delayed.

But the presence of the diphtheria bacillus in the eruption of the cow could be demonstrated both microscopically and by culture during the vesicular and pustular stages; in the latter also numerous pus cocci.

That in the cow the diphtheria bacillus as such passed into the system of the animal and appeared, not in the viscera, but on the udder, was demonstrated conclusively by the fact that before the end of five days after inocu-

lation, the presence of the diphtheria bacillus could with certainty by microscopic and culture observations be demonstrated in the milk of the cow collected under all precautions; the number of bacilli present on that day in the milk amounted to thirty-two per cubic centimetre.¹ It need hardly be added that these results lead in great measure to a right understanding of certain epidemics of milk diphtheria, such as those of Camberley and Yorktown, Enfield, Barking, and Croydon.

Von Emmerich isolated short thick rods from diphtheritic membranes, with which he produced a fatal disease in pigeons, rabbits, and mice. He found that, inoculated into the mucous membrane of the trachea of rabbits, the microbe caused death in sixty hours, with grey fibrinous membranes on the mucous membrane; the bacilli were present in the mucous membrane, blood, and viscera.

Löffler² showed that the so-called diphtheritic deposits in the mucous membrane of the fauces, larynx, and conjunctiva of fowls and pigeons are not the same as those of human diphtheria; in the pigeon it is different from that of fowls, while in the former it is caused by minute bacilli, thinner and a little longer than those of rabbit's septicæmia (Davaine, Koch); he also showed that the so-called diphtheria of calves is not the same as human diphtheria, since it is caused by long bacillary threads. Lingard and Batt had previously found the same bacilli in the necrotic masses in the mouth in calves; they have described the disease as a chronic ulcerative necrotic stomatitis. Dr. Lingard has shown that it is transmissible to the rabbit's ear, wherein the characteristic bacilli produce the same chronic necrotic ulcerative process.

As to the necrotic deposits in the fauces and mouth of fowls, not at all rare amongst poultry, and regarded by some as identical with human diphtheria, Löffler has pointed out that it is different from the similar disease in the pigeon; it certainly is not due to the same bacteria as those shown by Löffler to be the cause of the pigeon's disease. The writer has cultivated from the caseous yellow white deposits in the pharynx and mouth of such a fowl an organism which was present in almost pure culture. The yellow-white deposits are dry and brittle, and are made up of epithelial cells and *débris*. There are present various species of microbes in the superficial layers; but in the deeper parts one species of minute more or less constricted rods predominates, of the same size as those of fowl cholera, but differing from these latter by the fact that on potato they form rapidly a characteristic deep yellow growth; on gelatine, even after twenty-four to forty-eight hours they form white, round, prominent dots, which become more yellowish and project over the surface like little buttons, and are easily lifted off bodily; they are very tenacious, and do not break up when shaken in fluid.

¹ During 1890-91 the writer repeated these experiments on milch cows, and in two further instances out of six cows the eruption was produced on the udder and teats; in one of these positive cases the milk contained the diphtheria bacillus at about the end of the first week after inoculation (subcutaneously into the shoulder) with culture of the bacillus diphtheria.

² *Mittheil. aus dem kais. Gesundheitsamte*, vol. ii.

SECTION C

CHAPTER XX

FEVERS

TYPHOID OR ENTERIC FEVER

THE most prominent pathological changes characteristic of this and of no other disease are those affecting the Peyer's glands of the ileum, the mesenteric glands, and the spleen.

(a) *The Peyer's Glands*.—These show in the earliest stages—i.e. during the first week of the disease—a conspicuous increase in size, due to multiplication and enlargement of the cells of the lymph follicles, and to exudation of plasma into the latter; hence they are also more juicy. The mucosa and the sub-mucous tissue around and between the follicles of the Peyer's glands are much infiltrated, swollen, and the blood-vessels congested; and so, in a conspicuous degree, are the vessels of the lymph follicles constituting a Peyer's gland and of the parts immediately surrounding it—*stage of infiltration*. When the enlargement of the lymph follicles reaches its maximum the centre part undergoes a necrotic change—coagulation necrosis—the blood-vessels here being found in a state of hyaline degeneration; under the microscope one finds a network of coagulated fibrin threads, many lymph cells, which are enlarged, opaque and not taking dyes; in some the nucleus is faded and indistinct and not easily distinguishable, others breaking down into granular *débris*. This second stage, or *stage of coagulation necrosis*, extends in all directions till all parts of the Peyer's patch, particularly towards the internal surface, have become necrosed; the deeper parts, i.e. those situated in the sub-mucous tissue, as well as the latter, remain actively inflamed, their lymphatics being filled with coagulated fibrin and the connective tissue more or less infiltrated with lymph cells. During the second week the necrosed part being in a state of granular *débris* gradually breaks away, and thus an ulcer is established—*stage of ulceration*. The ulcerated Peyer's patch is crater-like, and is surrounded by a thickened, swollen, densely infiltrated mucosa, whose lymph cells are greatly enlarged; the base of the ulcer is the deep, sub-mucous portion of the Peyer's gland, while its surface is covered with granular *débris*, the remains of the necrosed part of the Peyer's patch. The ulcer, that is, the necrosis, enlarges (third week) in breadth at the expense of the mucosa immediately surrounding the original Peyer's patch, and in depth at the expense of the sub-mucous tissue and the circular muscular coat; but the reactive inflammation in the depth is, as a rule, a barrier and causes a gradual removal of the necrosed *débris*, and then the healing process commences (fourth week). In this process the place of the original Peyer's gland is transformed into a depressed pigmented scar, limited towards the depth by the outer layer of the muscular coat. During the increase of the inflammation and ulceration into the depth, i.e. into the outer muscular coat, the process may lead to perforation; the ulceration into the depth may also involve one of the larger vascular trunks of the sub-mucous tissue, and thus produce hæmorrhage.

The cicatrix which after healing occupies the place of the original Peyer's gland is easily detectable as a pigmented depression.

(b) *The Mesenteric Glands*.—The mesenteric glands show changes of the same kind as those of the Peyer's glands already mentioned; but no ulceration

takes place here. During the first week the glands are considerably swollen, very juicy, and much congested. In a section it is noticed that the gland contains a good deal of fluid; under the microscope it is seen that the lymph follicles of the cortex, particularly their central parts, are filled with fibrinous exudation, that the lymph cells are enlarged, and some contain many nuclei; in the sinuses, both cortical and medullary, the cells of the reticulum filling the sinuses are also much swollen and very conspicuous. The lymph glands continue to increase in size and juiciness till the third week. During the second week, there are found, as in the lymph follicles of the Peyer's glands, numerous spots and patches, in which coagulation necrosis has occurred. These necrosed patches gradually increase in size till the end of the third week; the lymph cells and fibrin networks constituting these necrotic patches undergo granular disintegration and fatty metamorphosis, and in this condition gradually become absorbed and disappear. There are present in the lymphatic tissue, in all parts of the gland and at all times, lymph cells which are conspicuous by their size, spherical in shape, and several times as large as the largest normal cells: they contain either one large nucleus or several smaller nuclei, their substance being filled either with coarse granules, or vacuoles, and at times also with blood-discs.

(c) The spleen is always enlarged, and this is chiefly due to an enlargement of all blood spaces and of the lymphatic tissue, i.e. the Malpighian corpuscles. As to the former the blood spaces of the pulp are considerably distended and filled with blood, and the cells constituting the pulp are enlarged; in some places all the pulp cells are changed into large spherical corpuscles containing blood-discs, being occasionally crammed full with them. In the enlarged Malpighian corpuscles the central parts also undergo the necrotic change described above, and these patches gradually increase in size, involving the surrounding pulp tissue to a greater or lesser extent. At the same time necrosed patches are found independently in the pulp, and are due directly to infarct in the pulp; such infarcts, and consequent necrosis, may produce extensive necrosis of the pulp tissue. The large cells filled with blood-discs above named are always present in the pulp from the earliest stages, and are occasionally washed into the circulation, and can then be met with also in the circulating blood.

Other changes in typhoid fever occurring at later stages must be considered as sequelæ, and due to secondary causes: thus the pneumonia is due to an infection with microbes capable of producing pneumonia. The diplococcus pneumoniae, streptococcus pyogenes, and Friedländer's bacillus have been isolated from such pneumonias. The granular and fatty degeneration observed in the liver cells must also be considered as due to secondary causes, perhaps chiefly the high temperature; the waxy degeneration of striped muscular tissue of Zenker is probably due to the same causes. Necrotic patches in the liver and spleen pulp—the infarct above mentioned—the necrosis and suppurations occurring as sequelæ in various tissues are probably due to a secondary invasion with pyogenic cocci or similar microbes. In the necrotic patches in the spleen, clumps of micrococci are almost constantly met with.

On the whole, then, we may say that the swelling and coagulation necrosis observed in the lymph follicles of the Peyer's gland, mesenteric glands, and the tissue of the spleen are the primary lesions, and in the intestine the necrosis leads to the formation of open ulcers.

Etiology.—Since the clear and precise statements by Budd in his well-known memoir on typhoid fever, this disease has been considered to be disseminated by the stools of a previous case of typhoid fever. That in this dissemination water plays an important part has long been accepted, and one

might almost say has been all but proved. Various epidemics and groups of cases have been investigated, both in this country and abroad, where a contamination of drinking water by sewage (old cesspools and drains), and by inference with typhoid excretion, had been proved to be connected with the outbreak and the spread of the disease. Von Pettenkofer, no doubt, showed for Munich that in the dissemination of typhoid the state of the ground water plays an important part, inasmuch as after a fall of this, leaving behind in the superficial layers of the soil the germs of the disease, the outbreak and spread of typhoid are caused and favoured, while on the rise of the ground water these poisonous particles again become submerged and prevented from escaping, and hence a subsidence of typhoid fever cases. But this ground-water theory, though applicable to Munich, has not been found to explain the rise and fall of typhoid fever in other localities during other epidemics, and therefore other conditions, notably, those of contamination of water or food stuffs with matter directly derived from sewage, and indirectly from bowel evacuations of typhoid fever cases, had to be considered. Further, Ballard has shown that in the dissemination of the virus of typhoid fever, as in the Islington epidemic, milk plays an important rôle; such milk typhoid epidemics, where the milk had directly, or by the vessels containing the milk, been brought in contact with sewage-polluted water, have since been abundantly recorded, as proved by an inspection of the tables collected for the 'Transactions of the International Medical Congress,' London, 1881.

The most complete and instructive evidence as to contamination of drinking-water by typhoid stools, and wholesale infection by such drinking-water, was brought forward by Dr. Thorne Thorne in elucidating the now famous epidemic of typhoid fever in and about Caterham. This report for 1878, published in the volume of the Medical Officer of the Local Government Board, contains the full account on this most instructive and now classical piece of epidemiological work. It was proved that one of the *employés* at the Caterham works of the Kent Water Company, while actually affected with typhoid fever, managed to contaminate with his stools the water at the original wells of the company, and this contaminated drinking-water, in its distribution about Caterham, Reigate, and other places, produced in the consumers typhoid fever.

The next important question that requires to be answered is, What is there in the stools which must be regarded as the virus of typhoid fever? This virus must be a microbe which evidently can live and thrive in sewage and in the soil; further, that it can retain vitality even when placed in such a poor medium as water; it must be possessed of considerable resistance towards putrefaction, towards cold, and towards the gastric juice; when brought, in the stools of a typhoid fever case, into drains, cesspools, and the like, it must be capable of preserving its life for an indefinite time—all these are requirements postulated by what is known of the epidemiological relations of typhoid fever. Many sanitarians have formulated these general conditions into a single sentence by saying that the typhoid-fever microbe must be capable of forming spores, and by these overcome all the above difficulties, since it is known that a microbe having formed spores can under favourable conditions preserve vitality till the suitable moment arrives, e.g. when finding entrance into a new individual; the spores are capable of withstanding all kinds of unfavourable conditions of environment, e.g. cold, putrefaction, paucity of nutriment, and struggle with other less specialised microbes, &c. Numerous have been the assertions of the occurrence of micrococci in the intestine, the mesenteric glands, and the spleen

in typhoid fever, but only during late years, since exact methods of staining and cultivation have been employed, has a definite species been shown to exist in the typhoid stools in the diseased intestinal mucous membrane, in the mesenteric glands, and in the spleen, occasionally also in metastatic foci of other organs: liver, lungs, brain, and kidney. Eberth first showed that in many cases of typhoid fever there occur in the swollen mesenteric glands, peculiar bacilli rounded at their ends, and occasionally including within a pale sheath one or more spore-like granules. Gaffky¹ may justly be said to have first successfully isolated and cultivated these bacilli of Eberth. Gaffky easily demonstrated them by cultivation from the typhoid stools, from the contents of the ulcerated Peyer's glands, from the swollen mesenteric glands, and particularly from the swollen spleen. In sections he demonstrated them in large and small clusters, in the swollen or already ulcerated Peyer's glands, in the swollen mesenteric glands, and in many places in the spleen. Though Gaffky did not succeed in cultivating them from every case, yet from the majority of cases he did succeed. Gaffky showed that the bacilli are always absent from the circulating blood, while from the spleen and the intestinal contents they can be readily isolated by cultivation and by staining cover-glass specimens. Numerous observers have confirmed these statements, and these bacilli are now, by many, if not most pathologists, considered as the microbes or bacilli of typhoid fever. They are also known as Gaffky's bacilli; the individual bacilli are cylindrical rods or filaments of various lengths, possessed in the fresh state of active locomotion: the short ones move briskly, the longer cylindrical rods or filaments showing a peculiar, slow, serpent-like movement. The bacilli grow well at 16–20° C. on and in gelatine, better of course at 25–37° C., e.g. in alkaline broth, on agar, in gelatine, on blood serum. On boiled potato at 85–87° C., they grow in a fairly characteristic manner, forming on it a transparent thin pellicle, whose thickness increases as the growth proceeds.

On the surface of gelatine they form colonies visible after thirty-six hours as translucent round dots, which during the third and fourth days become flat, translucent plaques with crenate outline; under the microscope they are brownish in transmitted light and granular; in streak cultivation the line of inoculation becomes marked after two to three days as a greyish translucent band, with uneven, knobbed or crenate outline; in stab culture the stab after a few days is seen as a grey line under the microscope, and in transmitted light it is made up of light brownish droplets; on the surface of the stab is a crenate, thin grey plaque. On agar they form a rapidly growing paint-like, cohesive, greyish-white to light brown pellicle; broth is made uniformly turbid after forty-eight hours; a greyish, powdery, or floccular precipitate, no distinct pellicle being formed. The bacilli are facultatively aerobic, but the character of the bacilli in and on gelatine, agar, or broth is not sufficiently marked to distinguish these bacilli from many others, but taking their appearance in the fresh state and in cultures on potato, together with the appearances on other media, the diagnosis can be made without much difficulty.

Typhoid fever has not been communicated by any experimenter to the lower animals; although septic, toxic, and septicæmic results have been produced in rodents by injection of typhoid fever stools and typhoid matter, there is no record of any result that can be at all interpreted as typhoid fever. Not even in monkeys has the writer been able to produce positive results by feeding them copiously on typhoid stools. Fränkel and Simmons, injecting rabbits with considerable quantities of cultures of the Gaffky bacillus,

¹ *Mittheil. aus dem kais. Gesundheitsamte*, vol. ii., p. 372.

produced death in a day or two under symptoms and pathological appearances which were considered by those observers to be indicative of typhoid fever, but which all pathologists now consider as due to septic infection. Sirotinin, Beumer, and Peiper¹ have shown that the broth in which the bacillus had been growing, injected after the removal of the bacilli, produces, according to the quantity used, severe septic intoxication and even death; further: animals (mice) injected with such a quantity of the broth culture as will produce illness, but not death, are thereby protected against a (second) fatal dose. But in these respects the toxic character of the broth cultures and the protective influence of small quantities of such cultures against a (second) fatal dose is equally true of a number of other microbes, as has been shown by numerous observers (Löwenthal, Roux and Chamberland, Roux and Yersin, Beumer and Peiper, Salmon, and many others).

White mice are in a large percentage of cases very susceptible to the pathogenic action of the cultures of Gaffky's bacillus; the majority of the mice inoculated with a small particle of a culture under the skin of the back die in one to three days with symptoms of septicæmia; the blood of the general circulation and the enlarged spleen containing the bacilli in great crowds, and pure cultures on gelatine can easily be obtained from these tissues.

The writer has met with bacilli in the swollen mesenteric glands of a child dead with the symptoms of acute summer diarrhoea, which microbes were isolated and cultivated, and whose cultures, and cover-glass specimens of such cultures, showed a remarkable similarity to Gaffky's bacilli. In plate cultivation, in streak and stab cultures, in broth and on potato, no distinction was possible between the two; in fresh specimens, and in stained cover-glass specimens, the bacilli appeared to be the same; when cultures were injected under the skin of mice exactly the same septicæmia, with crowds of bacilli in the blood of the general circulation and in the swollen spleen, was the result. After a long investigation, a difference was found between the two species; it is this: the bacillus of typhoid fever possesses a markedly lower resisting power to perchloride of mercury than the bacillus obtained from that case of summer diarrhoea; the latter grows fairly well, though retarded, on gelatine to which perchloride of mercury is added in the proportion of 1 in 40,000, while such addition has a much more marked restraining power on the typhoid bacillus.

Eberth, Gaffky, and others maintained that the typhoid bacillus is capable of forming spores; in potato cultures the bacilli constantly show bright terminal globules which Gaffky interpreted as spores; but Buchner has conclusively shown that these globules are, morphologically, not to be regarded as spores, that they are to be seen only in potato cultures, and are not identical with vacuoles also present, but that they represent involution products. Both Wiltshoud and Schiller ('*Arb. aus d. k. Gesundheitsamte*, V.) concur in this view. The drying of a thin layer of the culture of this bacillus—no matter of what age the culture, and no matter what the medium and whether grown on the surface or in the depth—kills the microbe, and in this respect there is a striking difference between real spore-bearing bacilli and the typhoid bacillus. The writer has convinced himself also of the fact that the typhoid bacillus never survives a temperature of 60° C. for five minutes, and in this respect too it behaves in a manner totally different from spore-bearing bacilli.

As must appear from the foregoing, the difficulties in accepting as satisfactory the evidence concerning the etiological importance of the Eberth-

¹ *Zeitschrift f. Hygiene*, Band I., p. 489.

Gaffky bacillus are neither few nor small: (a) the bacilli are not constant in every case of typhoid fever; (b) they are not present in the blood; (c) they occur in tissues (intestinal wall, mesenteric glands, spleen) in which owing to the nature of the disease serious disorganisation occurs, and therefore a secondary immigration of microbes from the cavity of the intestine is hereby much favoured and facilitated, particularly if it be remembered that the occurrence of the bacilli in these tissues has, for obvious reasons, not been demonstrated before the second or third week; (d) the bacilli do not form spores; and lastly, but not least, (e) domestic animals are not susceptible to typhoid fever, and therefore the crucial argument cannot be furnished.

That in typhoid fever this particular species should abundantly occur—they are never numerous in the stools, and if they appear they are not to be demonstrated before the eighth day of illness (Wiltshoud, St. Petersburg, 1887)—but not in other states, may only mean that the pathological conditions of the intestine in typhoid fever, being peculiar and *sui generis*, favour the growth and multiplication of this particular species of bacilli more than others. It is, however, by no means certain that, as was once believed, these bacilli occur only in the stools of typhoid fever; the results of recent observations seem to point to the conclusion that the bacterium coli commune of Escherich, which is a microbe present in the normal contents of the large intestine, is so similar in morphological, cultural, and physiological respects to the typhoid bacillus, that many observers cannot distinguish the one from the other (Arloing, Roux and Rodet, Congress of Hygiene, 1891). But we must be clearly understood that we do not deny that the Eberth-Gaffky bacillus may be the microbe of typhoid fever; we only wish to point out that anything like good and satisfactory evidence in this respect is not forthcoming, though many take it for granted that it has been proved that this bacillus is the cause of typhoid fever, and even pretend to be able to diagnose from the presence of this bacillus in water and other materials that such water and other materials have been and are capable of being the cause of typhoid fever. The utmost that in such cases can be said is that possibly, perhaps probably, such water and other material has been contaminated with excremental matter. In connection with this the conclusions arrived at by Lehmann as to the uncertainty of identifying the typhoid bacillus outside the body ought to be remembered; by Babes,¹ as to nineteen different varieties of typhoid bacilli found in the bodies of patients dead of typhoid fever; and lastly, of Cassedebat,² who found in Marseilles drinking water three different kinds of bacilli resembling the typhoid bacillus.

The bacilli of Gaffky when kept in common drinking water, or better still in sterilised water, gradually diminish in numbers; in fact, many die of inanition, as has been proved by numerous observers. In this respect these bacilli do not differ from other non-spore-bearing bacilli.

MALTA FEVER

Under this term a continuous fever has been described by Dr. Bruce, of Netley (the 'Practitioner'), which occurs in Malta, and until recently was confused with typhoid fever, the symptoms of the disease being similar in both cases, though from Dr. Bruce's careful account the temperature curve in Malta fever differs materially from that in typhoid fever. No typhoid bacilli were found in the spleen in cases of Malta fever. The pathology of the two diseases is also in this respect different, that in Malta fever there is no ulceration of Peyer's glands, so characteristic of typhoid fever. The

¹ *Zeitschr. f. Hygiene*, Band IX., p. 2.

² *Annales de l'Institut Pasteur*, 1890, No. 10.

spleen in Malta fever is usually enlarged. Dr. Bruce invariably found in the swollen spleen of nine cases the same species of micrococcus, which shows the following characters in microscopic specimens and in cultures. Under the microscope the micrococci appear as small oval cocci, measuring about 0.0006-0.001 millimetre, not forming chains. They grow best in agar jelly above 22° C.; at or below this temperature they show no growth, or only very scanty and retarded growth. At 25° C. it takes about seven days before the growth becomes visible to the naked eye; at 35° C., about half the time. In stab cultures in agar jelly, there appears along the track of the needle a row of minute white dots, but on the upper free end of the track a characteristic rosette-shaped group of whitish knobs or patches makes its appearance. There is no tendency to liquefaction of the jelly, and even after the lapse of twenty-one days the growth on the surface is restricted to the original point of starting.

Experiments of inoculation of cultures into guineapigs, rabbits, and mice, had no result. But in one monkey subcutaneous inoculation with a culture produced a definite and well-marked result, inasmuch as it set up a severe fever (temperature rising to 105, 106, and even 107° F.), lasting for twenty-one days, when the animal succumbed. The spleen was found enormously enlarged, and from it the characteristic micrococci were easily obtained by culture.

RELAPSING FEVER

The most noteworthy pathological changes in this disease affect the spleen, the marrow of the bones, and the liver (Pomfick). The spleen is enlarged, due to increase in size of the blood spaces in the pulp tissue as well as to an enlargement—inflammatory hypertrophy—of the Malpighian corpuscles; in cases that are the subject of a *post-mortem* examination, the spleen shows infarct, or necrotic patches in a state of breaking down and suppuration. The same applies to the red marrow of bones, which shows the same appearances as the spleen, viz. as a rule, inflammatory hypertrophy, occasionally in severe cases infarct and necrotic patches. In the liver the whole organ seems enlarged, the liver cells almost uniformly swollen, in many lobules opaque and granular; in severe forms there are larger and smaller accumulations of round cells in the interlobular tissue, extending also into the interior of the lobules; at the same time there are places where in and around the central vein of the lobules the vessels are much distended, with blood in stasis, thus forming an infarct; such a part is whitish on naked-eye view, and under the microscope the liver cells are opaque, granular, and show coagulation necrosis.

Obermeyer ('Centralbl. f. d. med. Wiss.,' 1878, No. 10) discovered during the febrile stage, in the circulating blood of patients affected with this fever, innumerable spirilla actively motile: they disappear from the blood immediately before the end of the febrile stage.

The spirilla are very thin and about 20-80-40 μ long; their movement is that of rapidly progressing spirals. Koch has demonstrated by photography of dried and stained specimens the presence of the flagella in the spirilla. Weigert has shown that, unlike other bacteria, they are barren of a cellulose sheath, since dilute liquor potassæ dissolves the whole substance of the spirilla. By drying and staining cover-glass specimens it has been shown that the spirilla are uniform spirals, and do not show anything that might be interpreted as being made up of shorter elements, comma bacilli or vibrios. The spirals when long are often plicated, but their turns are always close, and more or less in the manner of a corkscrew. Immediately

preceding the febrile stage they appear in the blood, grow more and more numerous during the fever, and disappear again completely from the circulating blood before the fever quite ceases. During the non-febrile stage they are most probably present in the spleen and marrow of bone—Birch-Hirschfeld found many of them in the necrotic foci of the spleen—where perhaps they undergo germination and reproduction. It is feasible to assume that when during the febrile stage they reach the height of their development they gradually break down, leaving spores in the shape of granules behind: these are carried into the spleen and bone marrow, where they accumulate. During the non-febrile stage these spores germinate here again, and gradually grow into the spirilla, which when ripe and motile gradually find their way again into the blood. Such a view would well harmonise with the facts of the case and also with what has been shown to occur in regard to the plasmodium malarie. As a matter of fact the spirilla in the blood often show bright granules in their interior which might well be spores.

Koch has shown that in artificial culture the spirilla are capable of growing into long spiral filaments matted together, but no real artificial cultures have been as yet produced. That the spirilla are the real microbes of relapsing fever is proved by the experiments of Vandyke Carter ('British Medical Journal,' October 1881), who was the first to produce typical relapsing fever in the ape after injection of blood of a patient taken during the febrile stage and containing the spirilla. The disease produced in the ape was true relapsing fever, and the animal's blood contained during the febrile stage the identical spirilla in large numbers. Koch, Heydenreich, and Metschnikoff have confirmed this. Motschutkowsky ('Deutsches Archiv f. klin. Med.,' Band XXIV.) has produced relapsing fever in the human subject by inoculation of blood containing the spirilla.

Metschnikoff ('Virchow's Archiv,' Band CIX., 1887) maintains that the final disappearance of the spirilla from the system, i.e. recovery, is due to phagocytes, that is to say, that the white cells of the spleen tissue eat up the spirilla, and thus purge the system of them; but though spirilla are found enclosed within white cells in the disease in the monkey, it does not follow that his view is correct, for Baumgarten has justly pointed out that as in other localities in other diseases so also here in relapsing fever the enclosure of the spirilla by leucocytes may and probably is only a result of the microbes having previously been killed by other agencies; for after this they are easily taken up by the white cells just like other dead formed matter.

CHAPTER XXI

ASIATIC CHOLERA—DYSENTERY

THE name 'cholera' is given to a group of acute diseases, which manifest themselves by profuse diarrhoea of more or less fluid evacuations and vomiting. In severe forms there are cramps of the muscles of the lower extremities, pain in the abdomen, coma, and suppression of urine. But the causes and pathology are widely different in the different kinds. There is first of all a form of cholera which almost in all large cities occurs in the summer months as a few sporadic cases: such are called sporadic or English cholera, or cholera nostras. But also at other times of the year cases occur similar to

those, and not having any tendency to spread; such cases occurred in the north of London in numbers during the winter months of 1887; further, isolated cases occur which are ascribed to the eating of over-ripe fruit, of tinned meat, of ice cream, of sausages, of pork pies and various other articles. In these cases there is no tendency of the disease to spread; it only attacks those individuals who have been directly subjected to the influence (eating) of the poisonous matter. But there is one form of cholera which has this great and fundamental character, that it rapidly spreads, and thus assumes the proportions of an epidemic. This form is called Asiatic or true cholera. It is endemic in the Delta of the Ganges; whence at particular periods it spreads over other parts of Asia, where its ravages in some years assume terrible proportions. In Europe, Asiatic cholera has repeatedly broken out epidemically, and then the fact that it has been introduced from a previously infected locality (Africa, Asia) has been fully established. Similarly its appearance in America has thus been shown to be due to introduction from an infected locality. It is not our object to consider in detail the epidemiological aspect of this dire malady, but a few points bearing on the subject of the etiology will be mentioned here. First of all the fact that an epidemic of cholera in a given locality outside Asia may, and often does, recur the next year and perhaps the third year, though the intensity of the epidemic and the number of its victims gradually becomes lessened, is proved in almost every one of the several visitations by cholera of European countries (for instance, the cholera that appeared in France in 1884 and 1885, in Italy and Spain in 1885 and 1886). Next as to the way cholera is introduced into a new locality. This has been proved to be either by articles derived from an infected person or an infected district, linen soiled by cholera dejecta, or a person or persons suffering from cholera, moving from one locality to another. In this move persons may—and have been known to have actually done so—give rise to new foci wherever they alight and wherever their discharges (evacuations and vomit) get access to drinking-water or food. Once a person affected with cholera enters a new locality and his discharges find access to a suitable soil, where the virus is capable of multiplying, a number of new foci can be hereby established, each of which in its turn may supply the poison for water and articles of food for considerable areas. Thus it happens that after the discharges of a person affected with cholera have gained access to the soil, where the virus multiplies and spreads and finally finds opportunity to gain access to drinking water, this latter becomes at once the means of producing infection in a large number of persons partaking of such water (*see* Dr. Snow's Report on the famous Broad Street Pump epidemic). Wherever in one way or another the cholera virus is deposited in a suitable soil it multiplies and spreads, and is capable of contaminating articles of food, fruit, milk, or water, and thereby causing infection. A further important etiological point of view is the fact that while the choleraic discharges have been known to be the vehicle of the cholera virus, there are cases on record where the cholera discharges, though copiously present and introduced into certain localities in which all conditions of sanitation were of the worst description—both as to filth, overcrowding, and contamination of water—did not produce infection. That this is the case in India and also in Europe has been repeatedly observed. Thus in India cholera in anything like an epidemic form is observed only during certain seasons of the year, December to July; later than July, or earlier than November, though cases may and do occur, or new cases may be and are introduced into a locality, as a rule no such marked tendency to spread and to assume the epidemic form is noticed. In Europe, April till October are

the months in which cholera, when it does occur, assumes an epidemic form ; during the winter months cases are scarce, or with the winter months cases cease altogether, to break out again in the next spring (see the last cholera epidemics in France, Italy, and Spain).

Further, besides season, locality plays an important part in the spread of cholera. Cholera, as has been so often shown, and as has been so much insisted upon by Sir John Simon in his writings on the spread of infectious disorders, is essentially a filth disease, that is to say, it depends in its introduction and in its spread on filth, this being the ground on which it grows and multiplies luxuriantly, and it is such filth that gives infective power to water and food stuffs. But although what health officers term 'insanitary conditions' play the very first rôle in the introduction and spread of cholera, yet there are localities which, notwithstanding their highly insanitary conditions, seem nevertheless unsuitable for the disease taking any strong footing (Lyons, Rome). Such localities possess a certain immunity against cholera, an immunity which has always seemed to offer a serious obstacle to the assumption—almost generally made—that the cholera dejecta *per se* in the fresh condition contain the actual cholera virus. Von Pettenkofer, Dr. Cunningham, the late Sanitary Commissioner to the Government of India, Drs. Lewis and D. D. Cunningham, and many others, who have studied the various outbreaks of cholera in India and Europe, have always insisted on this fact, viz. that unless season and particularly locality are favourable, cholera dejecta, though deposited and introduced in large quantities, do not seem to exert infective properties. It is therefore assumed—to explain this peculiar behaviour of the cholera virus—that the cholera dejecta contain the virus only *in posse*, that this for its 'ripening' requires to be deposited in a suitable soil ; that is to say, a soil in which it can live and thrive, and in which it can reach and attain that form in which it becomes infective to those whose alimentary canal it enters. That most cases of cholera are brought about by contaminated water and food, or, to speak generally, by the alimentary canal, is almost a universally admitted axiom, and it would appear that the infective stage of the cholera virus which it attains after sojourning in a suitable soil is something comparable to a spore stage. We shall see later on from other etiological reasons that this is most probably the case, and we shall give several instances in which cholera dejecta, though abundantly introduced in certain localities, did not produce infection.

In Asiatic cholera one of the most conspicuous symptoms is the diarrhoea of thin watery evacuations, associated with vomiting. When a cholera patient succumbs during the first twenty-four to fifty hours of the disease the *post-mortem* appearances are these : In a number of cases the serous covering of the stomach, jejunum, and ileum shows redness, is pale rose-coloured, either patchy or diffuse, with a sticky thin layer of exudation ; the same sticky exudation is found on the mesentery ; the mucous membrane of the jejunum and ileum, particularly the latter, is tumid and injected ; this is especially noticeable in the lower ileum, where here and there deeply congested patches occur in the mucous membrane. The epithelium of the mucous membrane throughout the small intestine is detached in flakes or is only loosely adherent ; by placing the opened intestine under water the epithelium is seen to be in the process of detaching itself from the villi in large areas ; the small intestine is distended and its cavity contains a more or less colourless, watery fluid in which numerous small or large mucus flakes are suspended. Under the microscope these are made up of a homogeneous mucus basis in which are enclosed epithelial cells of the sur-

face either isolated or more generally in small or large groups; some of these flakes are entirely made up of groups of epithelial cells. These are the cases which, during life, voided large quantities of fluid, watery evacuations—per mouth and rectum—and in which numerous mucus flakes are suspended. Owing to this condition, the evacuations are called rice-water evacuations; these cases are most typical also in other respects, viz. violent symptoms of vomiting and purging of rice-water-like fluid, great fall of temperature in the first stage—*stadium algidum*, cold extremities, violent and painful cramps in the calves, sunken eyes, ghastly look of face, small hollow voice, suppression of urine.

Not only the small intestine but also the large intestine contains that watery rice-water-like fluid, though in the earlier stages lumps of faecal matter may be still discerned in the lower parts of the colon. If during life copious rice-water stools have passed off, the large intestine is found *post mortem* to be void of solid faeces. In another set of cases, also ending fatally, during the first day or two, though the contents of the small intestine are fluid, they yet contain coloured, thin, almost fluid faeces, often mixed with blood; in these cases the mucous membrane of the ileum shows petechiae and hæmorrhagic patches. If death takes place after the first stage or the *stadium algidum*, i.e. during the typhoid stage, the small intestine contains little of the fluid matter, though the general injection of the mucous membrane is still distinct; in these cases the large intestine contains faecal matter. The kidneys, spleen, and liver show venous congestion, so do the cord and brain; the pia mater particularly is much injected.

Examining microscopically the intestinal discharges of acute cases of cholera one notices besides detached epithelial cells and mucous corpuscles, numerous bacteria belonging to different species of micrococci and bacilli. Some there are amongst them which are comma-shaped, i.e. curved, cylindrical rods, single or double, or S-shaped; they are motile, spinning round or moving in a spiral; they are of different lengths and of different amount of curvature, but, as cultivation experiments show, all belong to the same species; namely, the comma bacilli, or vibrios or spirilla of Koch, discovered by him as constantly present in the acute stages of Asiatic cholera, and as showing definite cultural characters.¹ There exist, however, considerable differences with regard to the number of these comma bacilli present. In some acute cases with typical rice-water stools the mucus flakes or the intestinal fluid contain these comma bacilli in enormous numbers, almost to the exclusion of other bacteria; such is the case in some typical cases in the mucus flakes taken directly from the watery contents of the ileum, though the mucus flakes taken in the same body from the jejunum, in all other respects identical, contain but few of these comma bacilli. In other equally typical acute cases they are scanty, other bacteria being very numerous. The writer cannot confirm Koch when he says that the more acute, severe, pure and typical a case, i.e. the more the intestine contains only the rice-water fluid with mucus flakes, the more numerous are the comma bacilli in the contents of the lower ileum, since the writer did not find this definite relation to exist either with regard to the severity, acuteness, or purity of the case. Some cases there no doubt are in which the mucus flakes of the rice-water stools directly taken from the contents of the lower ileum are crowded with the comma bacilli, but in a considerable percentage of typical cases this condition does not obtain; there are comma bacilli present, but on the whole they appear scarce. The epithelial flakes detached and suspended in the contents of the ileum, as well as the epithelial flakes loosened but not quite

¹ Conferenz zur Erörterung der Cholerafrage, *Berliner kl. Woch.*, 31, 1884.

detached from the mucous membrane, both of the villi and of the mouth of the Lieberkühn's follicles, contain comma bacilli as well as other bacteria. In sections through the hardened mucous membrane of the ileum one can find sometimes comma bacilli as well as other bacteria within the tissue of the superficial mucosa denuded of epithelium, and in the cavity of the Lieberkühn's follicles, and in spaces artificially produced by the loosening and detachment of the epithelium of the Lieberkühn's follicles, but their presence in these localities is due to immigration from the free surface into a disorganised mucous membrane, and neither bears any relation to the onset nor to the severity of the illness. Where the comma bacilli are scarce in the intestinal contents, they, or other bacteria, are altogether missed from the mucosa; where they are abundant in the contents and on the surface, and where the moribund stage lasted for hours, they penetrate from the surface into the disorganised mucous membrane.

In the illustrations accompanying this part, comma bacilli are shown as occurring in fresh mucus flakes of typical acute cases of Asiatic cholera, and it will be seen from these that, as regards size, curvature, and numbers, there exist considerable differences. The comma bacilli are met with in almost all cases of Asiatic cholera, beginning with those that show as yet only diarrhoea, more or less profuse, up to those that have shown all the typical characters, with vomiting and purging of copious rice-water evacuations. After the acute stage has passed, and the typhoid stage has set in, the comma bacilli become less numerous, and gradually disappear, so that when after three, four, or five days the evacuations again assume the character of faeces, the comma bacilli are either only found with difficulty or are altogether missed; in fact, in cases in which they are scarce at the earlier stages, they are not to be seen later than the third day.

If cholera stools, particularly rice-water stools, are kept for a day or so, one meets with comma bacilli which have formed spirilla; some are wavy threads, others are distinctly corkscrew-shaped, some short, others long; in dried and stained preparations many of these spirilla are seen to be chains of comma bacilli; spirilla are occasionally found even in the fresh stools or fresh mucus flakes, but as a rule the comma bacilli are present as single vibrios or as dumb-bell vibrios, i.e. S-shaped forms. As regards the amount of curvature and length of the individuals there exist great variations (*see* figs. 87 and 88).

The comma bacilli of Asiatic cholera show on cultivation in nutrient gelatine well-defined appearances, which together with their morphological characters enable us to recognise them, so much so that in suspicious cases of cholera their demonstration by cultivation in the evacuations is of diagnostic value. But in connection with this it must be borne in mind that in some early cases or in non-typical cases their demonstration by the culture test, owing to the vast predominance of other bacteria, is a matter of some difficulty. Where they are present in large numbers their demonstration by the culture test is a matter of comparative ease. All that is necessary is to place a small particle of the evacuation in a few (8-10) cubic centimetres of sterile (well boiled) salt solution, shake it up well, and then with a droplet of this inoculate nutrient gelatine, contained in a test-tube, liquefy the gelatine in warm water, shake up and then pour it on sterile glass dishes for the object of plate cultivations. A particle of a mucus flake of a rice-water stool, rich in the comma bacilli, diluted with several cubic centimetres of sterile salt solution, and a trace of this mixture used for plate cultivation, yields large numbers of colonies of the comma bacilli. These show themselves (at 20° C.) after thirty-six to forty-eight hours as greyish white minute specks

barely visible to the unaided eye ; after three days they are distinctly visible as small, clear, circular depressions, due to liquefaction of the gelatine within this depression. In the centre of the depression is a round, greyish speck surrounded by clear, liquefied gelatine ; looked at under a lens this speck appears granular, with a more or less uneven margin ; in the centre of the speck is a more opaque, round granule. Each of these colonies gradually enlarges ; the zone of clear, liquefied gelatine becomes broader, and the whitish central speck or patch enlarges ; where the colonies lie close together at the outset, the progressing liquefaction soon produces a coalescence of the adjoining colonies, and then we get a number of circular zones of clear, liquefied gelatine, each with a central grey patch, the zones being fused together at the points of contact. When during further growth the gelatine becomes liquefied over extensive areas, the outlines of the original colonies are lost, and on the surface of the clear, liquefied gelatine are thin, filmy flakes, and at the bottom minute whitish granules. In all stages, before and after the liquefaction of the gelatine has become well pronounced, there are found under the microscope rapidly motile vibrios, single commas, S-shaped dumb-bells, and numerous longer or shorter spirilla, chains of commas, some wavy, others uniformly spiral. The above-named 'granules' and 'flakes' are masses of commas and spirals intimately matted together, and when examined in the fresh state look like so many clumps of different shapes, rapidly revolving.

In stab culture in gelatine the characters of these comma bacilli are also well marked ; they are accurately represented in Plate XXX., and need not further be described.

After several days to a fortnight, there is noticed a distinct pellicle on the surface of the liquefied gelatine : this latter is clear, but contains a few whitish granules marking the outline of the funnel-shaped channel of liquefied gelatine.

Alkaline broth (at 86–88° C.) is slightly turbid even after twenty-four hours' growth : the turbidity increases during the succeeding days. After a week or so the superficial layers become gradually clearer, and this clearing extends steadily and insensibly towards the deeper layers ; hand in hand with this goes the deposit of a greyish white powdery precipitate ; a more or less distinct pellicle is noticed after a few days, and gradually increases in thickness. Under the microscope the comma bacilli in the fluid and in the pellicle are seen to be connected with beautiful spirilla, some of these of great lengths, some as many as twenty to thirty turns, the long spirilla more or less plicated and bent.

The growth on agar mixture is not characteristic, being in the form of thin, translucent patches and films, with rounded or knobbed outline, assuming as growth goes on, i.e. after some days, a slight brownish tint.

On boiled potato the comma bacilli grow only at temperatures above 25° C. ; at 86° they form after a few days a thick, smeary, brown film. Comma bacilli grow well and rapidly, if mucus flakes of a cholera intestine containing numerous comma bacilli are placed on linen kept damp. After twenty-four hours the comma bacilli have increased to an enormous extent, almost to the exclusion of other bacteria originally present, provided these were at the outset less numerous than the comma bacilli. Löffler has photographed the flagella, after staining by means of a new method ; there are always present several flagella at one end of each comma bacillus.

Comma bacilli grow well and luxuriantly between 17° and 40° C., on almost anything—paste, boiled egg, turnip, cucumber, cabbage, bread, meat, various fruits. &c. They grow best at 85–87° C., if the medium is faintly

alkaline; but they grow also on neutral medium, and even on some media which are slightly acid, like potato and fruit. The writer has seen comma bacilli which, having started on nutrient gelatine, kept for a few days at 20° C., continued to grow slowly but steadily, the gelatine being then kept at 15-16° C. Comma bacilli gradually die off if nutriment is insufficient, e.g. in distilled water; they are gradually killed in faecal matter; and they do not grow well in the absence of oxygen.

In all cultivations of the comma bacilli one meets with forms which differ from the typical curved, cylindrical vibrios, in so far as they are much thicker, plano-convex, bi-convex, or even approaching the spherical shape with a clear vacuole in the middle. In well-stained and well-washed specimens also the most typical comma bacilli show, within the sheath, the protoplasm collected at the ends—as a granule at each end—whereas the middle part remains clear. The above atypical forms are merely further developments of their normal form, caused by the enlargement of the central clear space or vacuole. Such atypical forms are to be met with in all cultures; their number, however, varies greatly with the character of the culture. If comma bacilli, originally derived from the cholera intestine, are carried through many successive sub-cultures in gelatine, say one or two dozen, the number of such atypical bi-convex or spherical forms is found greatly increased after a few days' growth. The writer has in his possession cultures of choleraic comma bacilli obtained from Calcutta direct from the choleraic intestine. For six months, sub-cultures were carried on through fifteen or twenty generations; all cultures made in broth, in gelatine, or in agar mixture by this time showed during the first few days all and every comma bacillus of a spherical shape; these were possessed of active motility and were of different sizes. After several more days, typical curved vibrios gradually made their appearance in the culture until only these were present. Plate cultivations prove most decidedly that at all times the culture is pure, but in every sub-culture the above spherical actively moving forms are at first present in large numbers (fig. 91).

The writer has in another place shown that such actively motile spherical forms by division give origin to two or even three comma-shaped, or more or less semicircular comma bacilli. The spherical forms are met with in numbers also in agar culture, if the agar mixture is at starting of neutral reaction, but they are abundant in all media (broth, gelatine, agar) after the cholera culture has been carried through many generations of sub-cultures. Such modified comma bacilli grow in gelatine somewhat more slowly than the recent ones, i.e. those recently derived from the cholera intestine.

Comma bacilli under culture rapidly undergo degeneration into granular *débris*; in fact, a good deal of the white deposit in gelatine and broth cultures is due to their *débris*. Degeneration goes on comparatively more rapidly in agar culture than in gelatine cultures. It is a notorious fact that on the surface of agar cultures the whole of the growth is found dead after from a few to several months, so that no new culture can be started from such an old culture. This degeneration and death occur sooner or later in all cultures; this alone proves sufficiently that the comma bacilli do not form permanent seeds or spores. Koch has proved by many experiments of drying that the comma bacilli are invariably killed by drying, unlike spore-bearing bacilli, and that at no time do the comma bacilli form spores. Heating cultures (old or recent) of comma bacilli to 60° C. for five minutes invariably kills the cultures—a proof that no spores are formed. The assertion of Hueppe that the terminal granules observed in comma bacilli are spores, viz. arthrospores, is definitely negatived by the above direct experiments.

Comma bacilli of cholera mucus flakes or of cultures, recent or old, are killed by acids, e.g. a fluid containing 0·2 per cent. hydrochloric acid,¹ so that the normal acid fluid of the stomach kills the comma bacilli; also this is opposed to there being spores present in the comma bacilli.

The comma bacilli occur in cholera as a rule only in the cavity of the small and large intestines, chiefly the lower part of the ileum and large intestine; no bacteria occur in the blood or other tissues. Comma bacilli and also other bacteria may and sometimes do immigrate into the disorganised tissue of the wall of the ileum, and in a few cases have been traced even as far as the liver and gall bladder; but in the large majority of cases the comma bacilli are limited to dead tissues, i.e. the contents of the ileum and large intestine and the superficial dead and disorganised parts of the internal surface of the mucous membrane of the ileum. For this reason Koch maintained that the disease is caused by a chemical poison—a kind of ptomaine—which, being elaborated by the comma bacilli within the ileum, is absorbed into the blood, and hereby sets up the disease cholera. We have mentioned above that the facts of the case prove that the comma bacilli are not found in the mucous membrane of the ileum unless as the result of secondary immigration, and therefore the explanation of the chemical poison elaborated by the comma bacilli in the cavity of the intestine being absorbed is not well supported; for it would require us to assume that at the time that the comma bacilli are notoriously few, i.e. at the onset of the disease, sufficient of the chemical poison is already present to produce the symptoms of cholera, whereas, as a matter of fact, the comma bacilli are only present in large numbers—and this only in a minority of cases—when the typical disease with typical rice-water stools has well set in. But it must be evident from the nature of the disease, that at this stage the intestinal wall can hardly be in a state capable of absorbing anything; on the contrary, large quantities of fluid are poured out from its mucous membrane. If the comma bacilli were present in considerable numbers in the mucous membrane at the onset of the disease, one could understand that producing the chemical poison in sufficient quantities the absorption of this would set up the symptoms of the disease; but the actual facts follow in exactly the reverse order, viz. the comma bacilli are scarce at first, and *cateris paribus* become more numerous as the symptoms of the disease become more pronounced, and when they are found abundant the typical disease has well set in. The comma bacilli in the cholera intestine are then present only in practically dead tissues, the fluid and detached mucus flakes of the contents of the ileum. And further, though the whole small intestine offers in typical acute cases the same appearances, the whole of the jejunum and ileum being filled with fluid and containing mucus flakes, yet we find the comma bacilli chiefly, generally solely, in the lower ileum, thus indicating that, like putrefactive bacteria, they increase in numbers as the large intestine is approached. The comma bacilli of Asiatic cholera produce in artificial cultivations toxic chemical products, cadaverine (Hueppe), such as are produced by various putrefactive bacteria; these products injected in sufficient quantities into rodents are capable of rapidly setting up intoxications leading to death. The comma bacilli of Finkler, and the comma bacilli of Deneke, although differing in cultural characters from the choleraic comma bacilli, produce the same toxic substance, cadaverine. Finkler's comma bacilli are not peculiar to the intestinal contents in sporadic cholera, as was first believed by Finkler and Prior, for Frank and Kartulis have not found them in cases of sporadic cholera, but they have been isolated by Brieger from the faecal matter of the normal

¹ Koch, Watson Cheyne (*Brit. Med. Journal*, 1885).

intestine; yet, as we have pointed out, these comma bacilli of Finkler are capable of producing the very same toxic substance, cadaverine; so also the comma bacilli of Deneke, found by him in stale cheese. There are other notorious saprophytes which have been proved to be capable of producing similar toxic principles.

The comma bacilli, then, do not differ from some putrefactive bacteria in this power of producing toxic substances in certain albuminous culture fluids. Van Ermengem¹ has first shown that those chemical poisons are not produced by the choleraic comma bacilli in all culture media to the same extent; while in broth and gelatine little of them is produced, in aqueous humour and in serum they produce them rapidly in large quantities. Further, Hueppe has shown that in white of egg (growing anaerobically in the egg) the choleraic comma bacilli rapidly produce toxic cadaverine. The choleraic comma bacilli produce in artificial cultures indol and nitrites (Salkowski), like many putrefactive bacteria.

Bujwid² was the first to show that broth cultures of comma bacilli, incubated at 37° C., give with ordinary hydrochloric acid, or nitric or sulphuric acid, a definite colour reaction of red-purple—the cholera red. Dunham³ has found that for this reaction the presence of peptone in the broth is essential, and that other comma bacilli (Finkler's and Deneke's) also show this reaction, though in a lesser degree. Salkowski showed that this reaction is closely associated with the formation of indol and nitrites in the culture fluid by the comma bacilli. Thus far we see, then, that in chemical respects the choleraic comma bacilli do not differ from other putrefactive bacteria.

Since they are capable of producing toxins, it follows that when they have an opportunity of multiplying sufficiently they produce a sufficient amount of the toxic substance cadaverine, and thereby are capable of producing acute poisoning. The experiments of Van Ermengem prove that where this chemical substance is produced in sufficient quantities (e.g. in aqueous humour or serum) even small quantities injected into rodents (guineapigs) produce acute intoxication. Van Ermengem further showed, and in this his views have been confirmed and extended by the observations of McLeod and Mills ('Reports from the Laboratory of the Royal College of Physicians,' Edinburgh, vol. i.), that if in the intestine of a guineapig the comma bacilli can be brought into rapid multiplication (*see* below), the intestinal fluid teeming with the comma bacilli contains those same toxic principles in a concentrated form, so that a small quantity of it injected into a fresh guineapig produces intoxication and death. In order to produce a similar intoxication in guineapigs with broth cultures or gelatine cultures it is necessary to inject considerable quantities of these cultures. In some cases it is, however, possible to produce a septicæmic infection in mice or guineapigs by subcutaneous injection of cultures of comma bacilli. Koch has succeeded in mice, Ferran and Dr. D. D. Cunningham have succeeded in guineapigs, the writer has in several instances by using large doses of culture fluid (0.5–2 c.c.) succeeded in producing such septicæmic infection in guineapigs, equally with choleraic comma bacilli or with Finkler's comma bacilli, the blood and the tissues containing crowds of the comma bacilli. Dr. D. D. Cunningham has in several instances succeeded in producing such septicæmic infection and death, the blood, the exudation on the serous covering of the intestine, and the intestinal contents containing an abundance of comma bacilli.

All these experiments, however interesting they may be, do not throw any

¹ *Recherches sur le microbe du Choléra Asiatique*, Brussels, 1885.

² *Zeitschrift f. Hygiene*, II., p. 52.

³ *Ibid.*, p. 837.

real light on the question whether or not the comma bacilli are the real cause of cholera; they only show that towards rodents the choleraic comma bacilli behave like so many other notoriously putrefactive microbes, e.g. the Naples bacillus of Von Emmerich, the bacterium of Brieger isolated from normal human fecal matter, the bacillus of Bienstock, the comma bacillus of Finkler, &c.

But it is asserted that the comma bacilli when introduced into the small intestine in a living state are capable of setting up an acute illness resembling Asiatic cholera, and death.

Koch in his first pamphlet on cholera (*l.c.*, p. 27) told us that he had made every imaginable effort to produce cholera in animals experimentally. The experiments of feeding white mice with cholera dejecta, first made by Tiersch and then by Burdon Sanderson, were repeated by Koch over and over again on fifty white mice fed with this material (dejecta of cholera patients, and the contents of the intestine of cholera corpses) and with choleraic material after it had begun to decompose, but no result whatever followed; the mice remained healthy. 'We then made experiments on monkeys, cats, poultry, dogs and various other animals that we were able to get hold of, but we were never able to arrive at anything in animals similar to the cholera process. In precisely the same manner we made experiments with the cultivations of comma bacilli; these were given as food in all stages of development. When experiments were made by feeding animals with large quantities of comma bacilli, on killing them and examining the contents of their stomachs and intestines with a view to find comma bacilli, it was seen that the comma bacilli had already perished in the stomach, and had usually not reached the intestinal canal. . . . The comma bacilli had been destroyed in the stomachs of these animals. . . . The experiment was therefore modified by introducing the substances direct into the intestines of the animals. The abdomen was opened, and the liquid was injected immediately into the small intestine with a Pravaz syringe. The animals bore this very well, but it did not make them ill. We also tried to bring the cholera dejecta as high as possible into the intestines of monkeys by means of a long catheter. This succeeded very well, but the animals did not suffer from it.' 'I must also mention,' says Koch, 'that purgatives were previously administered to the animals in order to put the intestine into a state of irritation, and then the infecting substance was given, without producing any different result. The only experiment in which the comma bacilli exhibited a pathogenic effect, which therefore gave me hope at first that we should arrive at some result, was that in which pure cultivations were injected directly into the blood-vessels of rabbits or into the abdominal cavity of mice. Rabbits seemed very ill after the injection, but recovered after a few days. Mice, on the contrary, died from twenty-four to forty-eight hours after the injection, and comma bacilli were found in their blood. Of course they must be administered to the animals in large quantities; and it is not the same as in other experiments connected with infection, where the smallest quantities of infectious matter are used, and yet an effect is produced. In order to arrive at certainty as to whether animals can be affected with cholera, I made inquiries everywhere in India as to whether similar diseases had ever been remarked amongst animals. In Bengal I was assured such a phenomenon had never occurred. This province is extremely thickly populated, and there are many kinds of animals there which live together with human beings. One would suppose, then, that in that country, where cholera exists in all parts continually, animals must often receive into their digestive canal the infectious matter of cholera, and in just as effective a form as human beings;



but no case of an animal having an attack of cholera has ever been observed there. Hence I think that all the animals on which we can make experiments, and all those, too, which come into contact with human beings, are not liable to cholera, and that a real cholera process cannot be artificially produced in them.'

Starting from the idea that the comma bacilli are killed by the gastric juice, and that in order to develop their pathogenic powers they have to get unscathed and living into the small intestine—their natural breeding-ground—it occurred to Koch¹ that this difficulty might be obviated by first neutralising or making alkaline the contents of the stomach, and introducing *per os* the comma bacilli. He therefore kept guineapigs for twenty-four hours without food, and then injected into their stomach *per os* 5 cubic centimetres of a 5-per-cent. watery solution of carbonate of sodium. This does not noticeably injure the stomach, and, as direct observation proved, kept the contents of the stomach in an alkaline condition for three hours. Some minutes (twenty) afterwards he introduced by catheter 10 cubic centimetres of a cultivation of the comma bacilli in meat infusion.

The result is noteworthy. Seven guineapigs thus experimented upon remained perfectly well: 'They were killed after twenty hours,' says Koch, 'and the contents of their stomach, intestine, and cæcum were examined by gelatine plate cultivations. In six of the seven animals the cholera bacteria could be demonstrated in the small intestine. The experiment had thus in so far succeeded that the cholera bacilli had passed uninjured through the stomach, but they had not set up any disease in the animals.' Similar experiments were then made on eight other guineapigs. These animals also remained quite healthy. Finally four guineapigs were similarly experimented upon (5 c.c. of solution of sodium carbonate, then 10 c.c. of cultivation of the comma bacilli in meat infusion); three remained well, the fourth appeared ill next day, looked shaggy and did not eat; on the following day it was very ill; paralytic weakness of the posterior extremities came on, the respiration was weak and slow, the head and extremities were cold, and the animal died in this condition. On *post-mortem* examination the small intestine was markedly reddened and full of a flakey, watery, colourless fluid. The stomach and cæcum contained a large quantity of fluid. 'The examination with the microscope and with gelatine plates,' says Koch, 'showed that the contents of the small intestine contained a pure cultivation of the choleraic comma bacilli.' 'That this one animal only should have died, out of a series of nineteen, uniformly experimented upon, suggested some peculiar condition that had obtained in this one animal, and as a matter of fact on examination it was ascertained that this animal had aborted immediately before the injection, and on *post-mortem* examination it was found that the abdominal walls were very flaccid and the uterus still greatly enlarged. This led me to the idea that either the abortion *per se*, or perhaps its unknown cause, had acted on the other abdominal organs, more especially on the small intestine, in such a way as to produce a temporary relaxation with arrest of peristaltic movement; and thus had rendered it possible for the comma bacilli to remain longer and gain a footing in the intestine.' This conclusion appeared to Koch justifiable, inasmuch as by direct experiment he thought he had proved that the contents of the stomach pass too rapidly into, and through the small intestine, and that the comma bacilli could only unfold their poisonous action, i.e. could produce the chemical poison, if they had time to remain there and to multiply. Consequently if they were not delayed in their passage through the small intestine they would not multiply there,

¹ Conferenz zur Erörterung d. Cholerafrage, Berlin, May 1885.

and once in the cæcum, where the reaction is acid, they would become harmless. To this method of reasoning, one may be allowed to take exception, Koch shows by direct experiment that even twenty hours after injection the comma bacilli can be recovered from the small intestine in a living state. Now the most important character of all pathogenic bacteria is this, that when introduced into the particular tissues suitable for their propagation they set up their pathogenic power. How is it, then, one might reasonably ask, that the comma bacilli, if even only for a few hours in the small intestine, do not in swarms invade the epithelium and superficial layers of the mucous membrane? Koch does not, and of course cannot, deny that all absorption of the chyle must take place in the small intestine, and since the comma bacilli are much smaller than the large chyle globules, and are possessed of spontaneous mobility, it follows of necessity that the comma bacilli can and must readily pass into the epithelium and the superficial layers of the mucous membrane; and since the epithelium and the superficial mucous membrane, according to Koch's own statement and belief, are the suitable nidus for the multiplication and action of the comma bacilli, all conditions would therefore here exist which are required for their settling down and acting. Add to this that the 10 c.c. of a broth culture injected, containing millions and millions of comma bacilli, are subject to absorption by the small intestine for twenty hours (see the above-mentioned observations of Koch), and that such vast crowds of comma bacilli in a few hours kept at the body temperature ought to yield a most formidable host of descendants, and grave doubts must arise as to the tenability of Koch's explanation.

But to continue. In order to produce a condition similar to the one in the above single successful experiment on the guineapig, Koch injected tincture of opium into the peritoneal cavity after the introduction of the sodium carbonate and the cultivation of the comma bacilli: this answered well for achieving positive results. Immediately after the administration of the 10 c.c. of the culture of the comma bacilli, 1 c.c. of German tincture of opium for every 200 grms. of the animal's bodyweight was injected into the peritoneal cavity: the animals became narcotised for half an hour, and died after one and a half to three days, with the same symptoms as the above guineapig. 'Eighty-five guineapigs have been infected in this way with cholera.'

Now the following criticisms can, we think, be justly applied to these experiments: (1) According to Koch's own showing, it cannot be the narcosis which is essential, even allowing for the present that relaxation of the intestine may have been produced by the intraperitoneal injection of opium tincture, since alcohol alone, injected by Koch into the peritoneal cavity, produces the same result; he says that thereby 'we were most successful in making the animals susceptible to the cholera infection.' (2) Can narcosis of the animal be produced by opium without furthering in the least the process of the experiment? This has been tried over and over again; watery extract instead of tincture of opium is injected into the peritoneal cavity, and narcosis lasting for one hour is produced, but the animals remain well; tincture of opium is subcutaneously injected, the animals fall into narcosis, lasting for from forty to eighty minutes, but no result is obtained from the previous introduction of the comma bacilli; in fact, the experiment as designed by Koch was repeated by the writer on a large number of guineapigs, thirty in all, but instead of producing narcosis by injection of tincture of opium into the peritoneum, he produced it by intraperitoneal injection of watery extract of opium, or subcutaneous injection of tincture of opium and watery extract of opium, but all in vain. The comma bacilli used were of recent broth culture, or of gelatine culture, and were beyond

question or doubt the choleraic comma bacilli. (8) It is not proved that injection of tincture of opium into the peritoneal cavity produces relaxation of the intestine and arrest of the peristaltic movement; there is no proof given for this by Koch as regards the guineapig; on the contrary, there are experiments on record made on the dog, when the result of such injection was quickening of the peristaltic movement.

From all these considerations it appears unwarranted to conclude, as Koch does, that the multiplication of the comma bacilli in the small intestine, and their fatal action by the chemical products they elaborate, take place on account of a relaxation and arrest of the peristaltic movement by the opium. Another explanation appears much more probably correct. It is this: provided the intestine is first made diseased, either in consequence of slight peritonitis, as was probably the case in the guineapig that had aborted, or in the experiments in which tincture of opium or alcohol alone was injected into the peritoneal cavity, or from other reasons, the comma bacilli that are present in the intestinal cavity undergo rapid multiplication, and by their chemical products, not only increase the disorder of the mucous membrane, but eventually poison the animal. And from this we conclude, further, that a multiplication of the comma bacilli can and does take place only when the intestine is previously brought into a diseased state. Under this view all Koch's and Van Ermengem's results become at once intelligible.

We maintain, then, that the living choleraic comma bacilli, in however large a number they be introduced into the small intestine, are quite innocuous; but they are rendered capable of great multiplication if the intestine is previously, from some cause or another, diseased. The chemical products of such multiplication act as poisons analogous to the ptomaines obtained from other putrefactive bacteria.

That this is the true explanation we find proof in some of Koch's experiments with other bacteria, notably with Finkler's and Deneke's comma bacilli. With both these organisms, on experimenting in the above manner, he obtained positive results; not so constantly, it is true, but still he did obtain positive results, not identical, but similar. Of course it is not to be expected that, seeing these are three different species, they would act in the same manner. Finkler¹ published a large series of experiments, in which, with his comma bacilli, and after the method of experimentation employed by Koch, he produced results identical with those gained by Koch with the choleraic comma bacillus. There can be no doubt, from what has been shown above, that Finkler's comma bacillus has nothing to do with cholera nostras, or with any other infectious disease, but that it is simply a putrefactive organism. And on the same grounds Koch's comma bacillus cannot be said by these experiments to have been proved to have a causal relation to Asiatic cholera, any more than has Finkler's comma bacillus, or any of the other species of septic bacteria that are capable of producing chemical poisons analogous to ptomaines. All that can be said is that, provided that conditions are established by which the choleraic comma bacilli are enabled to grow and multiply in the intestinal canal, these chemical poisons may be produced. A very instructive and parallel case is found in the so-called typhoid bacillus. As is now generally held, the experiments published by Fraenkel and Simmonds, in which they maintain that they have produced typhoid fever and death in rabbits after injection of large quantities of cultivations of the typhoid bacilli, do not prove any real infective action of the typhoid bacillus for the rabbit; it has been conclusively proved that this result is entirely due to certain chemical substances generated by the typhoid bacillus in the cultivations

¹ *Ergänzungsheft z. Centralbl. f. allg. Gesundheitsh.*, I., 5 and 6.

(Sirotinin, Beumer and Peiper). This pathological condition can be produced, entirely apart from the bacilli by chemical substances (typhotoxin) produced by them in cultivations, and as is the case in other similar toxic substances the severity of the abnormal state depends on the quantity injected. Moreover, it has been shown by Beumer and Peiper that by the injection of a small quantity of the chemical substance, a refractory state against an otherwise fatal dose of the same substance can be produced (Beumer and Peiper, 'Zeitschr. f. Hygiene,' II., p. 110).

Dr. D. D. Cunningham ('Scientific Memoirs, by Medical Officers of the Army of India,' Part VI., Calcutta, 1891) has shown that there are typical cases of acute cholera in which no comma bacilli occur, and further he has made the important discovery that the comma bacilli occurring in Asiatic cholera belong to a number of well-defined species, differing from one another in size, in the rapidity with which they liquefy the gelatine, and in the manner in which they grow on potato, so that these facts alone enable Cunningham to deny the causal relation to cholera claimed for Koch's comma bacilli.¹

From these experiments, and similar ones, we conclude that the disease thus produced in guineapigs has no claim to be considered as Asiatic cholera, since Finkler's comma bacillus and Deneke's bacillus induce, under the same method of experimentation, a similar fatal disease. Besides, the disease thus induced in guineapigs differs in this important respect from cholera, that the temperature of the animals is at first raised, and only shows a fall when the animal is really moribund.

Various species of comma bacilli are known: Finkler's, Deneke's (cheese spirillum), the spirillum of noma, spirilla in various mucous substances isolated and described by Weibel,² and others.

Gamaleia³ has shown that a vibrio in many respects similar to, if not identical with, Koch's comma bacillus of Asiatic cholera causes a septicæmia in fowls and pigeons similar to, but not identical with, fowl cholera; it is the one he named vibrio Metschnikoff. He further stated that the two vibrios are in so far inimical to each other as that an animal that has survived the disease produced by one is possessed of immunity against the other. Pfeiffer, however, shows ('Zeitschr. f. Hygiene,' VII., 8), that these last assertions of Gamaleia are incorrect. While he confirmed Gamaleia as to the septicæmic effect of the vibrio Metschnikoff on pigeons (with copious occurrence of the vibrios in their blood), he proved that morphologically, culturally, and experimentally (as tested on pigeons and guineapigs), they are two different species, and that there exists no inimical action of one against the other.

Pfeiffer has also shown that Gamaleia's assertion as to the susceptibility of pigeons to infection with the choleraic commas (it is in pigeons that Gamaleia maintained that he had succeeded with his protective inoculations) is incorrect, pigeons being insusceptible to such infection. The writer can fully confirm Pfeiffer in this, since, even with large doses of cultures of the choleraic comma bacilli injected into pigeons, he has not been able to produce any result whatever; moreover, on injecting into the pectoral muscle of pigeons as much as 2-3 c.c. of broth culture, and searching

¹ It ought to be stated that recently Dr. Friedrich directly traversed these statements of Cunningham (*Arbeiten aus d. k. Gesundheitsamte*, Berlin, 1892). The writer, however, has been able fully to confirm Cunningham's statements as to the permanently different cultural characters of his various species of choleraic comma bacilli.

² *Centralbl. f. Bact. und Parasit.*, II., No. 16; IV., Nos. 8, 9, 10.

³ *Annales de l'Institut Pasteur*, No. 9, 1888, p. 482.

by culture test and cover-glass specimens for comma bacilli twenty-four hours later, no trace of them can be discovered; the pigeons remain perfectly well.

DYSENTERY

This is an affection of the large intestine, occurring in the human subject, chiefly in hot climates. It consists in a hæmorrhagic effusion into, and necrosis of, the mucous membrane; in the affected parts the mucous membrane (the mucosa) is the seat of extensive engorgement of the blood-vessels, stasis of, and hæmorrhage from, the veins and capillary blood-vessels; at the same time there is exudation of cellular and fluid matter, inclusive of blood *en masse*, into the cavity of the intestine, and of course in the evacuations; the hæmorrhage is followed by necrosis of the mucosa in the area of the hæmorrhage, with a reactive inflammation surrounding the necrotic parts, both in the mucosa and sub-mucosa; in consequence of this inflammation, the necrotic parts become gradually loosened and detached, and the deficiency is partly filled with the products of the inflammation. In the affected parts, the size of which varies from that of punctiform petechiæ, to that of areas several inches in length, the tissue elements undergo the change known as coagulation necrosis; though they retain their outline for a little time, their substance appears opaque, granular, and they do not take the ordinary stains; the epithelium of the surface is loosened, or altogether lost, from extensive parts of the surface; the lymphatic tissue of the mucosa, and the epithelium lining the Lieberkühn's crypts, which becomes loosened and detached from the membrana propria, fail to take the ordinary stains, become granular, opaque and dry looking, and are gradually transformed into a granular detritus. Around the part thus affected numerous leucocytes accumulate in the mucosa and sub-mucosa, and these leucocytes gradually invade the necrotic portion.

The serous covering of the intestine, as well as the mucous membrane of almost the whole of the large intestine, is much congested. The mesenteric lymph glands are swollen and highly congested. As a frequent sequela there occurs formation of abscess in the liver.

Lösch (Virchow's 'Archiv f. pathol. Anatomie,' 1875, Band LXV., p. 196) was the first to discover the *amœba coli* in great numbers in a case of ulcerated large intestine in the human subject. This case, in all its clinical and pathological symptoms, resembled true dysentery.

Kartulis ('Centralbl. für Bact. und Parasit.,' VII., 2) has shown that in the cases of tropical dysentery which he examined, numerous *amœba* (*amœba coli* of Lösch) were present in the characteristic sanguineous stools showing active amœboid movement, and he gives good reasons for considering these the cause of the dysentery; though others who had met with similar amœbæ in intestinal diseases in Russia (Massiatin, 'Centralbl. für Bact. und Parasit.,' VI., Nos. 16 and 17) did not think so.

Further, Kartulis has shown that in twenty cases of abscess of the liver complicating dysentery he found in every one of them the same dysentery amœbæ; they could be seen in sections through the wall of the abscess, but in the pus of the abscess cavity he did not find them.

A considerable amount of literature exists at present on the occurrence of amœbæ in certain forms of dysentery, chiefly those that run a chronic course, and on their absence and the presence of various species of bacilli in other forms of acute dysenteric inflammation of the large intestine. While some have confirmed Kartulis (Osler, Councilman, Maggiora, and others), others have missed the amœba, but describe various species of bac-

teria as connected with the disease. From the careful bibliography collected by Maggiora ('Centralblatt für Bact. und Parasit.,' XI, Nos. 6 and 7) there can be no doubt that what is clinically spoken of as dysentery, is not in etiological respects one single disease, since some dysenteric affections are, others are not, caused by the *amœba coli*.

CHAPTER XXII

INFANTILE DIARRHŒA OR SUMMER DIARRHŒA

THIS disease, causing considerable mortality in children, occurs in an epidemic form in the summer months in many English towns; in some, as Leicester and Nottingham, to a larger extent than in others. Dr. Ballard, who has for several years been investigating this disease, formulates in his report ('Supplement to the Report of the Medical Officer of the Local Government Board,' 1887, p. 7) as the causes of the diarrhœa the following conclusions:—

'That the essential cause of diarrhœa resides ordinarily in the superficial layers of the earth, where it is intimately associated with the life processes of some micro-organism not yet detected or isolated.

'That the vital manifestations of such organism are dependent among other things, perhaps principally, upon conditions of season and on the presence of dead organic matter, which is its pabulum.

'That, on occasion, such micro-organism is capable of getting abroad from its primary habitat, the earth, and having become air-borne, obtains opportunity for fastening on non-living organic material, and of using such organic material, both as nidus and as pabulum, in undergoing various phases of its life history.

'That in food, inside as well as outside the human body, such micro-organism finds, especially at certain seasons, nidus and pabulum convenient for its development, multiplication, or evolution.

'That from food, as also from the contained organic matter of particular soils, such micro-organism can manufacture by the chemical changes wrought therein through certain of its life processes a substance which is a *virulent chemical poison*; and

'That this chemical substance is, in the human body, the material cause of epidemic diarrhœa.'

It will be observed that this provisional hypothesis is sufficiently elastic to include, as a common cause of diarrhœa, chemical products of bacterial life manufactured indifferently within or outside the human body. Elasticity, to this extent, of a provisional hypothesis has been necessary for the reason that, in the present state of our knowledge, certain cases and groups of cases of diarrhœa, not distinguishable from epidemic summer diarrhœa, have now and again been found to possess the faculty of being directly communicable from person to person. The account of Dr. Bruce Low's experience at Helmsley, in Yorkshire, appended to the above report (Appendix H) is illustrative of this class of cases.

It will be obvious that in the stools of such infective cases of diarrhœa the hypothetical organism causative of the malady may be looked for with good hope of success.

A point worthy of further inquiry, and hitherto rendered suspicious by many health officers, is this, that infants fed from 'the bottle' are the principal victims of fatal diarrhœa in summer.

Pathology (Dr. Ballard's Report, pp. 13 and 14):—

'In the bodies examined there were marked pathological changes, not only in the intestines, but in all the viscera, and not alone in the viscera of protracted cases, but in those of infants the total duration of whose illness had not exceeded twelve or fourteen hours.

For the most part the intestines were empty, or only contained a little yellow faecal matter, or a little opalescent mucoid fluid, or the surface was coated with some thick, creamy, catarrhal exudation. The amount of obvious hyperæmia of the mucous membrane of the stomach and intestines varied; sometimes there was observed some follicular ulceration both in the small and large intestines. Generally there was more or less inflammatory thickening of the mucosa, and even in the cases of only a few hours' duration denudation of the epithelium, both of the stomach and intestines. Now and then ecchymoses were seen, or even a little blood effused into the alimentary canal. The solitary and agminated glands of the small intestine were most prominent; the mesenteric glands were enlarged. The condition of spleen varied: it was sometimes congested, or exhibited hyaline degeneration of the arteries, or swelling of the Malpighian corpuscles with degeneration of the central portion of them. The condition of the liver varied. It was either congested, or pale and bloodless, the former condition prevailing in the cases of short, and the latter in those of longer duration; but in every case examined there was one invariable condition, viz. fatty degeneration of the liver cells, slight in cases of short (only a few hours') duration, but pronounced and extensive, or complete in all parts of the organ, when the illness had been protracted. The kidneys, even when normal to the naked eye, were invariably found to be diseased when examined microscopically, showing inflammatory and degenerative changes, intense glomerulo- and parenchymatous nephritis being demonstrated even in cases of very short duration. The lungs, although they might be in parts collapsed, presented marked evidence of acute catarrhal and interstitial pneumonia. The blood may be inspissated and coagulate imperfectly. There was nothing in the microscopical investigations of the tissues, blood, or excreta to indicate that in the cases which furnished the organs, blood, or excreta, the malady was due to any micro-organism developing within the alimentary canal or permeating any of the tissues.'

As regards microbes, various bacteria have been mentioned as occurring in the intestinal contents and in the stools in this disease, but there does not seem to be any relation proved to exist between them and the disease. From the writer's own observation he is prepared to say that among those microbes hitherto described as the cause of infantile summer diarrhoea, none can claim that distinction.¹ The following may be quoted here from Dr. Ballard's Report as an instance of an organism very similar to that of Gaffky that was found in an acute case of summer diarrhoea in the intestinal contents, penetrating also into the mesenteric glands, viz. :—

'The case was one of typical acute summer diarrhoea admitted into the hospital in Great Ormond Street, and which ended fatally with fits of eclampsia. In the cavity of the intestine there were found numerous mobile bacilli which in size and shape bore a certain resemblance to the bacilli known as occurring in typhoid fever. These bacilli were also found on microscopical examination of the sections of the swollen mesenteric glands, but were not numerously present there. From these glands cultivation experiments were made on nutritive gelatine; the bacilli were thus isolated and pure cultivations obtained. Comparing these cultures with those of the typhoid bacilli of Gaffky, a striking resemblance is noticed: (a) They look alike in microscopical specimens. (b) They grow alike in plate cultivation. (c) They grow alike in broth. (d) They are very much alike in stab and streak cultures on gelatine and on agar agar mixture.' Comparing the two in respect of their action on animals, it is found that when inoculated into white mice they produce in a large percentage of cases death under septicæmic appearances; the bacilli could be recovered from the heart's blood by cultivation. The differences between them, so far as can at present be seen, are: (1) That this bacillus from the case of infantile diarrhoea grows on gelatine more rapidly than Gaffky's typhoid bacillus; and (2) that the former has a somewhat greater resisting power to the action of perchloride of mercury than the latter.'

A severe form of diarrhoea occurring during the summer months amongst adults, not referable to consumption of poisoned food, and of a highly in-

¹ Lesage describes in the green stools of infants affected with diarrhoea a species of minute rods slightly motile, which he considers the actual cause of the disease.

² There was a brief notice of this microbe in the *Annual Report of the Medical Officer of the Local Government Board* for 1886, p. 447.

fectious nature, is recorded by Dr. Bruce Low. These forms of the disease evidently occupy an altogether different position from the infantile summer diarrhoea, as well as the choleraic attacks due to toxic effect of certain food stuffs. They are fully described in Dr. Ballard's Report, pp. 127-131.

CHOLERAIC DIARRHOEA DUE TO TOXIC EFFECT OF CERTAIN FOOD STUFFS.

Various forms of acute disease are known which manifest themselves in severe gastro-enteric disturbance, vomiting, diarrhoea, pains in the abdomen and gastric regions, and more or less muscular prostration; in some severe cases there is suppression of urine, cold skin, collapse, and even death. While these cases have a certain clinical resemblance one to another, they are etiologically different. In some the symptoms set in very soon, sometimes a few hours or an hour only after the introduction of the poisonous matter, while in others there is a distinct incubation period, varying from twelve, sixteen, to twenty-four hours, and even longer. In the latter cases the illness is always more protracted, and when death takes place the lungs, liver, and kidney are found congested, the spleen flaccid, and slightly enlarged; in the stomach and intestines the mucous membrane is much congested, and there are even hæmorrhagic petechiæ, the mucosa is swollen, the epithelium of the surface partially loosened or altogether detached. In the cavity of the stomach and intestines there is a mucoid fluid occasionally containing extravasated blood; but these appearances on the part of the alimentary canal are found also in the acute fatal cases of the first category.

In the first category of cases the disease is due to a chemical poison present in certain articles of food stuffs (ice cream, cheese, sausage, mackerel, tinned food materials); this poisonous substance has, in some instances, been isolated (Dr. Vaughan, tyrotoxicon), as a crystalline substance probably identical with certain crystalline bodies isolated by Brieger and others, and belonging to the group of toxins, e.g. cadaverine (pentamethylene diamine), in contradistinction to the ptomaines of Selmi, e.g. putrescine (tetramethylene diamine).

Now in some cases it has been shown that such toxin is formed in food stuffs by definite bacteria, having no action on proteid materials comparable to putrefaction in the ordinary sense, i.e. that process which starts with the change of proteid into peptone, decomposing this latter into leucin and tyrosin, forming at the same time ptomaines with the evolution of sulphuretted hydrogen and ammonia. The writer has had the opportunity of investigating several such forms of acute chemical poisonings brought about by the consumption of veal pie in one case (Dr. Ballard), pork pie in another, the latter outbreak reported by Dr. Spear in the Report of the Medical Officer of the Local Government Board ('Report of the Medical Officer,' p. 106, 1887-88). In both instances a bacterium was found in large continuous masses in the pies, and it was isolated and cultivated in artificial media. Mice fed with the pies succumbed rapidly under the appearances of severe gastro-enteric symptoms; mice fed with the artificial cultures succumbed in the same way; but no microbes were found in the body, and the conclusion arrived at was that these bacteria are capable by their growth (in gelatine as well as in broth) of elaborating the toxic substance. The symptoms of disease in these mice set in after a few hours, as was the case in the human subject, and this clearly pointed to a chemical poison. The bacterium isolated from veal pie, as also that from the pork pie, produced when growing in gelatine a greenish blue colouring matter, which gradually increased in amount and became diffused through the gelatine. In the case of the veal pie

bacterium, liquefaction of the gelatine began only after several weeks had passed, and it proceeded very slowly; it took several months to liquefy the gelatine (about 10 c.c.) in a test tube; the liquefied gelatine was thick and syrupy. In the case of the pork pie the bacterium did not liquefy the gelatine at all. Besides, the veal pie bacterium did not show any growth when kept above 25° C.; it grew best at 20–22° C., while the pork pie organism grew also at temperatures above this—at temperatures up to 36° C. So that we have here two distinct sets of acute chemical poisonings produced in food stuffs not showing any sign of what is ordinarily called putrefaction, poisons produced therein by two different species of bacteria, and which when they were introduced into the stomach set up gastro-enteritic disturbance.

A third similar instance was the Portsmouth beef-pie poisoning. Here a motile cylindrical bacillus was found, which did not liquefy the gelatine; mice fed with the beef pie or with the cultures of the bacillus succumbed with symptoms of acute gastro-enteritis. ('Report of Medical Officer of the Local Government Board for 1890–91').

The second category of gastro-enteritic disturbances produced by food stuffs, those with distinct incubation, are true infections.

Dr. Ballard's report of Welbeck and Nottingham ham-and-beef poisoning ('Report of the Medical Officer of the Local Government Board, 1880') gives an illustration of such infection. In this epidemic, ham was shown to have contained a peculiar short motile bacillus, which was found in large numbers in the vessels of the glomeruli and in the spleen of fatal human cases (fig. 152). Another similar epidemic occurred in Carlisle in 1889, and was due to certain pork pies. The pork and gravy stock from which the pies were made was proved to act poisonously by feeding mice on it. From such mice (congested lung) a bacillus was recovered by cultivation, which was proved to be the same as in the gravy stock. Cultures of this bacillus by feeding and subcutaneous inoculation produced in mice the same disease as the pork and the gravy stock—enteritis with sanguineous mucus, sanguineous diarrhoea, congestion of the lungs.

Gärtner ('Correspondenzblätter des allgem. ärztl. Vereins von Thüringen, 1888,' p. 578) found in the flesh of a cow dead with symptoms of acute diarrhoea, and also in the spleen of a man who partook of that meat and died in twelve hours, a peculiar species of bacillus—*bacillus enteritidis* Gärtner—which is motile, does not liquefy gelatine, and is very pathogenic for mice, guineapigs, and rabbits, both after feeding and after subcutaneous inoculation.

Gaffky and Paak described an epidemic of gastro-enteritis at Egelsdorf and Böhrsdorf amongst factory men who had eaten of meat and sausages made from horse flesh. The cause was found in a bacillus, 'sausage bacillus,' similar to, but not identical with, Gärtner's bacillus ('Arbeiten aus dem k. Gesundheitsamte,' Band VI., 2 and 8, 1890).

CHAPTER XXIII

INTERMITTENT OR MALARIAL FEVER—AGUE

THE pathological changes in intermittent fever, as observed in those malignant cases which terminate fatally, are chiefly those of the blood, the spleen, the liver, the marrow of bones, less constantly of the brain and kidneys. The blood in all cases shows a large number of black pigment granules singly

and in clumps, free and enclosed in peculiar corpuscles (*see below*), which granules are proved to be derived from the colouring matter of the red corpuscles. These granules are either spherical, irregularly shaped, or oval, and they correspond to what is known as melanin. The spleen is always enlarged: this enlargement is due to an enlargement of the spleen pulp as well as of the Malpighian corpuscles; the latter are hypertrophied, in the central parts the lymph cells are swollen, many of these showing necrotic changes; in the spleen pulp the cells are greatly swollen, many contain several nuclei and masses of (black) blood pigment, the same pigment being present in the connective tissue of the trabeculae, around, and in the wall of, the blood-vessels; also in the capillary vessels of the Malpighian corpuscles. This abundance of black pigment distinguishes the enlarged spleen in malaria from that occurring through other causes. In the red marrow of bones many of the marrow cells are much enlarged, and contain masses of black pigment; in the liver the blood-vessels of the lobules contain masses of black pigment granules, the liver cells themselves in many lobules showing fatty degeneration; in the interlobular connective tissue numerous round cells infiltrate the tissue, and in chronic cases these infiltrations lead to the new formation of connective tissue. In severe cases (*febris perniciosa*) necrotic changes of the liver cells around the central vein are noticeable (Garnieri), the blood in the latter being stagnated, and the capillaries leading to it full of leucocytes and pigment masses. In the kidneys severe changes occur, consisting of granular and fatty degeneration of the epithelium lining the convoluted tubes; around the arterial vessels of the cortex are found miliary aggregations of round cells. Pigment granules in larger and smaller masses are also found in the capillaries of the kidney, whereby many of them become plugged and impermeable. Also in the brain, pigment granules are found in capillary vessels, and in the severe form (*febris perniciosa comatosa*) plugging of them with pigment granules has been observed.

As to the etiology of the disease, most important discoveries have been made within the last few years. Laveran ('Comptes Rendus, 1882,' No. 17) and Richard (*ibid.*, No. 8) were the first who discovered in the blood of malaria cases, in the febrile stages, spherical or crescentic bodies consisting of a pale, homogeneous substance enclosing clumps of pigment granules; these bodies are possessed of cilia by which they are enabled to perform rapid movement. Laveran considered these bodies as the true cause of malaria, and identified them as protozoa. This discovery was a few years later (1885) confirmed and considerably amplified by Marchiafava and Celli. The credit of the important discovery of the malaria parasite belongs therefore unquestionably to Laveran, though the observations of Marchiafava and Celli have amplified by a good deal our knowledge of them; their merits, though considerable, are not commensurate to the credit they generally receive as compared with that accorded to Laveran; they have shown us, however, a good deal of the life history of the parasite, and have given it the name *hemoplasmodium* or *plasmodium malariae*. The researches of Golgi have made important additions as regards the life history and distribution of these parasites, and particularly as to their relation to the various forms of malarial fevers. We will follow in our description chiefly the very exhaustive observations made by Golgi and described by him in the 'Fortschritte der Medicin, 1889,' No. 3; but it ought to be mentioned at the outset that, according to Metschnikoff, Celli and Garnieri, the malaria parasite belongs really to the class of sporozoa amongst the protozoa, and, therefore, the name *plasmodium malariae* introduced by Marchiafava and Celli is, according to Metschnikoff, incorrect.

Marchiafava and Celli¹ were the first to show that during the beginning of the febrile stage the parasite invades the red blood-corpuscles as small, globular, pale, homogeneous corpuscles measuring not more than a fifth to a seventh of the diameter of a red blood-corpuscle; in this host the parasite performs active amoeboid movements, hereby changing continually its shape; but it gradually increases in size and consumes the substance of the red blood-corpuscle, leaving black pigment granules—iron-free melanin—in the disc. These pigment granules, as the parasite grows to the size of the original red blood disc, are contained within the body of the parasite, in which they appear uniformly distributed. When the disc of the red blood-corpuscle is entirely consumed by the growth of the parasite, this latter appears free in the blood plasma, its substance filled with the melanin granules; some of these free parasites have cilia by which they move actively—these are the corpuscles seen by Laveran. Next, the pigment granules aggregate in the central part of the parasite and the peripheral, pale, homogeneous portion gradually undergoes a more or less regular mode of segmentation, in the course of which small globular particles or sporules become constricted off from the main body; when this segmentation has been completed, the young gemmæ or sporules all disappear from the blood, so also the pigmented central parts, and are stored up in the spleen, liver, and bone marrow; this terminates one febrile attack. The next febrile attack is caused by the sporules again invading the blood-corpuscles of the general circulation, and therein undergoing the same series of changes as just described. So that each febrile stage comprises the invasion of the blood-corpuscles by the sporules, the germination, amoeboid movement, and growth of these latter within and at the expense of the former, then the gemmation and segmentation of a new crop of sporules, and finally the disappearance of these from the general circulation. Golgi by his numerous researches was able to show that the various forms of malarial fever are due to various species of the parasite, at any rate that in the different forms of intermittent fever the time in which the parasite passes through all the above-mentioned phases of its development is different, and stands in a definite relation to the form of the fever. Thus, Golgi found that in the febris quartana, the parasite from its first appearance in the red blood-corpuscle, that is, from the onset of a febrile attack, through the complete segmentation of the full-grown parasite into the sporules, and to the disappearance of these from the general circulation, i.e. till the end of the febrile stage, requires three days, whereas in the febris tertiana it requires only two days. Besides, there are certain slight morphological differences between the parasite in the febris quartana and in that of the tertiana, as also differences in the mode of segmentation (*see* Plate XLII.). As to the parasite in the fever of irregular type, Golgi shows that also in this the time occupied for passing through its phases is irregular, either too rapid or too slow. The crescentic form of the parasites mentioned by Laveran and Marchiafava and Celli are present only in fever of irregular type, and are really an atypical form in the development of the parasite. So also the flagellate forms seen by Laveran are atypical forms.

Whether in these different forms we have really to deal with different species of the same group of parasites as Golgi inclines to think, or rather with differences in the life history of the same species caused by unknown conditions, e.g. individual person, different tissue, season, locality, &c., is not decided.

Canalis ('*Studi della Infezione malariana*,' Torino, 1889) studied the atypical forms of malarial fever, characterised by longer or shorter febrile

¹ '*Untersuchungen über die Malaria-Infektion*,' *Fortschritte d. Med.*, 1885, pp. 389 and 787.

intervals. He found in these cases an endoglobular form of the plasmodium malarie, which has been signalised already by Golgi, viz. a crescentic form; but also here the commencement of the attack is characterised by the amœboid endoglobular forms, and the life cycle of the parasite becomes completed by its division into sporules.

Thomas Smith described in 1889 in the red blood discs of cattle affected with Texas fever the presence of parasites very similar to those occurring in human malarial fever, and the number of the infected blood discs is so enormous that it seems impossible to doubt that those parasites are the cause of the Texas fever; hence the bacilli described by Billings (*see* a former chapter) assume the character of saprophytic additions.

Danilewsky, Grassi and Feletti, Kruse, Pfeiffer, Celli and Sanfelice and others describe the occurrence of similar parasites in the red blood-corpuscles of a number of different animals, frogs and birds (*see* 'Fortschritte d. Med.,' Band IX., Nos. 12 and 18, 1891).

CHAPTER XXIV

TETANUS—RABIES AND HYDROPHOBIA

CARLE and Rattone ('Giorn. dell. r. Accad. d. Med.' Torino, 1884) were the first to show that tetanus is a communicable disease. They succeeded in producing in rabbits typical tetanus, terminating fatally, by inoculating them with pus taken from the ulceration of a human being in whom tetanus had set in. Purulent exudation was taken in these rabbits from the place of inoculation and transferred to fresh rabbits, and here typical tetanus was again produced. In human tetanus the place of infection (produced in the skin of the hand or foot by a tainted splinter, earth, or other material) becomes marked as a purulent inflammation leading to ulceration; the tissue surrounding the ulceration is much infiltrated, and there is always hæmorrhage in it. After death the membranes of the brain and cord are found much injected, and so also the grey matter of the medulla and cord; occasionally there is a slight accumulation of red and white blood-corpuscles around the vessels.

Nicolaier ('Inaugural Diss.,' Göttingen, 1885) made the important discovery that earth taken from superficial layers of the soil is often capable of producing, when inoculated into the subcutaneous tissue of the mouse, rabbit, or guineapig, a local suppuration and hæmorrhagic effusion about the seat of inoculation, rapidly followed by typical tetanus and death. In that earth and in the pus and exudation of the seat of inoculation he demonstrated the constant presence of fine, straight bacilli, which he considered as the *tetanus bacilli*. The purulent matter containing these bacilli, inoculated into fresh mice, rabbits, or guineapigs, again produces tetanus. Rosenbach ('Archiv f. klin. Chirurgie,' Band XXXIV., 1886) showed that the same bacilli exist in the exudation at the place of infection in human tetanus. Hochsinger, Beumer and Peiper, Bonone, Shakespeare, Raun, and many others have confirmed the existence of these bacilli in tetanus, but no one of these succeeded in cultivating them in pure cultivations. Though numerous cultivations have been established, and tetanus has been produced in animals with them by the aid of foreign bodies—cotton wool, splinters, &c.—yet these cultivations were always in an impure state, until recently Kitisato ('Zeitschrift f. Hygiene,' Band VII., p. 225) succeeded in cultivating the tetanus bacillus of Nicolaier in pure cultivations (*see* Plate XXIX., fig. 37) and in producing

tetanus with such pure cultures. Minimal doses inoculated into mice produced tetanus in twenty-four hours, death in two to three days. In the case of rats, rabbits, and guineapigs the dose had to be somewhat larger, 0·3–0·5 c.c. of broth culture. Rats and guineapigs are ill with tetanus even after twenty-four to thirty hours, rabbits not before two to three days. On *post-mortem* examination of such animals there is no suppuration at the seat of the inoculation, but only hyperæmia; hence the suppuration observed in other cases is not an essential feature, and in former experiments and in the case of human beings is probably only due to the presence of the foreign bodies themselves (earth, splinters, &c.) which were the vehicles of the tetanus bacilli; in the internal organs there is no definite change. In the organs there are no bacilli present, nor was it possible to produce tetanus in other animals by inoculating them with the cord, nerves, blood, or spleen of the animals dead of tetanus. In rabbits, Kitissato produced typical tetanus by injection of 0·5 cubic centimetre of broth culture of the tetanus bacillus into the vein of the ear. Also by injection of the culture into the *dura mater* after trephining, Kitissato produced typical tetanus; but neither in the brain, nor in the cord, nor in the blood or other viscera of these animals, could the tetanus bacilli be found.

The success of obtaining pure cultures of the tetanus bacilli was achieved by Kitissato by cultivating tetanus pus anaërobically; preliminarily to this he watched for the time when in ordinary cultures of the tetanus pus on serum or agar he found organisms amongst the different species of bacteria present, which, by their peculiar shape and by their containing a terminal thick spore, he recognised as the tetanus bacilli. By exposing such impure cultures for three-quarters of an hour to an hour in water at 80° C. all bacteria were killed except the spores. With material thus treated he made gelatine plate and tube cultures, but in such a way that the air was excluded by substituting for it an atmosphere of hydrogen gas, or by planting the bacilli in the depth of the gelatine. Under these anaërobic conditions he obtained pure cultures (Plate XXIX. fig. 87). In this figure the character of the cultures is well seen; the microscopic appearance of the bacilli is shown in fig. 102.

It appears, then, from these exact researches that the introduction of the tetanus bacilli under the skin is followed by the production by them of a chemical virus, which, as it is being produced at the seat of inoculation, is absorbed into the system and sets up the disease; but the bacilli themselves appear to remain limited to the seat of inoculation, and do not enter the blood or any other tissue, and therefore only the seat of the inoculation contains the infective principle, i.e. the bacilli; for this reason, the brain, cord, nerves, blood, and viscera have no power of producing infection.

Brieger has, as a matter of fact, isolated from the exudation at the seat of infection in human tetanus a toxic principle, *tetatin*, the injection of which produces tetanus symptoms in animals; and Kitissato showed this held good also for the tetatin obtained from the cultures of the tetanus bacilli.

Behring and Kitissato ('Zeitschrift für Hygiene,' X.) showed that the blood of rabbit (previously made insusceptible to tetanus) injected into a mouse (otherwise susceptible to tetanus) neutralises in this latter the action of the tetanus bacillus.

Vaillard and Vincent ('La Semaine médicale,' No. 51, 1890) have published strong chemical evidence to show that *tetatin* is neither an albumose nor an alkaloid, but is related in its chemical characters to snake poison.

Buchner and also Kitt ('Centralb. f. Bact. und Parasit.,' VII., No. 10) have also succeeded in obtaining pure cultures of the tetanus bacilli. Their

characters agree with those stated by Kitisato, except that Kitt found that the tetanus bacilli obtained from the horse liquefy solid blood serum. Kitt points out that in the tetanus of horses—a not uncommon disease—the pus taken from the local abscess, generally about the hoof, when inoculated into mice and horses, produces without fail fatal tetanus. Kitt has found that such pus dried even for four months, when inoculated in minimal doses, has still virulent action; this fact is easily explained by remembering that the tetanus bacilli present in the pus and in the local exudation contain spores.

In his 'Experimental Researches on the Poison of Tetanus' ('*Zeitschr. f. Hygiene*,' X., 2) Kitisato gives a full account of the influence of light, heat, drying, and of various chemical substances on the tetanus poison.

Tizzoni and Cattani, in a series of memoirs, demonstrated the means by which animals, naturally possessed of slight or great susceptibility, can be made altogether insusceptible to tetanus; further, the blood serum of animals, made previously insusceptible, when injected into animals possesses a decided antitoxic action. They have isolated from such blood serum this substance—the tetanus antitoxin—by precipitating with alcohol, drying in vacuo, and dissolving in water. In four cases of human tetanus, the disease was arrested by the injection of the antitoxin of Tizzoni, and the patients recovered ('*Centralbl. f. Bact. und Parasit.*,' Band X., No. 24, p. 785).

RABIES OR HYDROPHOBIA

This disease, which always terminates fatally, is common to man and carnivorous animals: to man it is invariably transmitted from a rabid animal—dog, wolf, cat. Dogs are the common breeders of this malady, next come wolves. In Russia especially this species yields a considerable number of cases. In India, rabies amongst dogs is common, amongst jackals not uncommon. Cats when bitten or infected by rabid dogs become affected with rabies; cows, horses, asses, sheep, and deer are susceptible to rabies, and occasionally become affected by it owing to the bite from a rabid dog; rabbits and monkeys have been experimentally proved by Pasteur to be easily susceptible to the disease.

The pathology of the disease in man does not offer striking changes; and they are chiefly limited to the place of infection, i.e. the bite of a rabid animal. The wound with the onset of the disease is tumid, injected, and painful—in fact, it is this change in the place of infection which indicates the onset of the disease, previous to the actual outbreak of the disease. There is no symptom observed at the place of inoculation during incubation. After death the most noteworthy pathological changes refer to the salivary glands, the stomach, and the central nervous system. In all these organs we find general congestion: in the salivary glands the gland alveoli are in a state of exhaustive secretion; in the parotid the gland cells of the alveoli are swollen and opaque, and the lumen of the alveoli almost obliterated; in the submaxillary gland the number of mucous alveoli is greatly in excess of what is the case in the normal gland; the cells of all mucous alveoli are in a state of extreme mucous change; and at the same time there are present numerous round cells in the interalveolar and interlobular connective tissue. The most noteworthy changes are found in the central nervous system. Besides the general congestion of the blood-vessels, many vessels of the grey matter are surrounded by leucocytes and red blood-corpuscles, almost forming in some places special sheaths of white and red corpuscles in the perivascular lymph spaces surrounding the blood-vessels; also in the lymph spaces surrounding the ganglion cells, i.e. the pericellular lymph spaces, a few

leucocytes are contained. In the central nervous system the accumulation of cells in the perivascular and pericellular lymph spaces is very marked. In animals (dogs, rabbits) the mucous and serous coats of the stomach, particularly the former, show petechiæ and extensive patches of hæmorrhages; in the dog affected naturally with rabies—street rabies—the sub-maxillary glands are swollen, congested, and the mucous cells lining the alveoli enlarged and full of mucus.

That rabies is a communicable disease, transmitted from animal to animal, and from animal to man by the bite of a rabid animal, has been well recognised by physicians and veterinarians, though the spontaneous origin of the disease has been occasionally asserted; but not until Pasteur's observations was it proved that the disease is experimentally transmissible from animal to animal. The experimental proof furnished by Pasteur has been the starting point for a more precise understanding of the malady, till then shrouded in a good deal of mystery; it has further been the beginning of an entirely new era in the prophylaxis against this terrible disorder, a prophylaxis, the value of which can now no longer be doubted. The discoveries by Pasteur of the nature of, and protective inoculation against, this malady may justly be regarded as some of those few discoveries which mark a new epoch in scientific medicine. Before Pasteur's researches the experimental production of rabies was practically unknown; Pasteur showed that the central nervous system of an animal or human being dead of rabies contains the virus of the disease, and that if a portion of such central nervous system—the cord in the fresh state—be injected subdurally into a susceptible animal, this latter becomes, with absolute certainty, the subject of the malady, the period intervening between the injection and the actual outbreak of the disease differing with the material used and with the animal into which it is injected. This discovery at once proved of immense diagnostic value, since it was now possible to determine whether in a given case the human being or the animal, as the case may be, had really died from hydrophobia. This proof was of especial importance in those cases in which the pathology and symptoms of the antecedent disease had not been typical, and where the diagnosis of rabies could not be formed with anything like certainty from the clinical and pathological point of view, and such cases, as is well known, are not by any means rare. With Pasteur's discovery it became easy to decide the question: the fresh spinal cord (a portion emulsified in water or broth) injected subdurally into a rabbit produces rabies with absolute certainty, if the human being or animal from whom that cord was taken had really died from hydrophobia. This is a considerable step in advance of all previous knowledge, and in itself represents a definite landmark.

We cannot do better than quote *in extenso* from the Report of the English Commission on Pasteur's Researches on Hydrophobia (consisting of Sir James Paget, chairman; Dr. T. Lauder Brunton, Dr. G. Fleming, Sir Joseph Lister, Sir R. Quain, Sir Henry Roscoe, Professor Burdon-Sanderson, and Professor V. Horsley, secretary). A *résumé* of experiments made in this direction by Professor Horsley is here reproduced from that report, pp. 1 and 2:

'Through the kindness of M. Pasteur two rabbits inoculated by him were placed at the disposal of the committee on May 5, 1886, and were conveyed within twenty-four hours safely to the Brown Institution, where the experiments were carried out by Mr. Horsley.

'In these two rabbits the first symptoms of rabies appeared on May 11 and 12, and the disease followed exactly the course described by M. Pasteur.

'At first the animals appeared dull, but continued to take food readily until symptoms of paralysis appeared. The first of these symptoms was commencing paralysis of motion

of the hind legs, not accompanied by any loss of sensibility. The paralysis soon extended to the muscles of the forelegs and later to those of the head, and the animals died comatose.

'After *post-mortem* examination, portions of the spinal cord of each of these rabbits were crushed according to M. Pasteur's method in sterilised broth, and the liquid so obtained was injected beneath the dura mater into four rabbits and the same number of dogs, all being first rendered insensible with chloroform or ether.

'Of the four rabbits so inoculated, the first two showed the first symptoms seven days after the inoculation, the third and fourth on the sixth day. The symptoms as well as the incubation period exhibited by these rabbits were exactly the same as were observed in those brought from M. Pasteur's laboratory. Careful notes and photographs were taken in the case of all the animals, in order that the constant and specific nature of the disease might be demonstrated by observations during life and after death. It was also observed that during the incubation period the temperature of the body remained normal, that is, about 39.4° C. With the first definite symptom the temperature rose to about 40.4° C., which is the temperature usually observed during the first day of the obvious illness. By the next day it began to fall, and on the third day, after the appearance of the first symptom, it averaged 37.5° C. On the last day it was always below normal, and on one occasion fell before death to 24° C. The animals did not appear to suffer any pain whatever in the course of the disease. They were free from the spasms which, in the earlier stages of the malady in man, form so painful a feature of the disease, and, indeed, the disease in them resembled throughout that rapidly fatal but painless disease of man known as acute ascending paralysis.¹

'The *post-mortem* appearances in the rabbits were remarkably uniform. As a rule, nothing abnormal, save congestion, presented itself either in the brain, spinal cord, heart, blood-vessels, or serous membranes. The larynx, pharynx and, more especially, the epiglottis and the root of the tongue were frequently intensely congested. The lung showed almost invariably capillary congestion, and sometimes small patches resembling bronchopneumonia were observed. The mucous membrane of the stomach was very markedly congested, and there were at its cardiac extremity numerous hæmorrhages.¹ The constancy of these appearances was most remarkable, and corresponded in every particular with those subsequently observed in rabbits which had died of rabies from the bite of rabid dogs.

'Of the four dogs inoculated, the first showed on the eighth day after inoculation an alteration in the voice and commencing excitement; on the following day the excitement became excessive, and the bark was quite characteristic; on the eleventh day the dog was aggressive, notwithstanding slight paralysis of the legs; on the twelfth day the paralysis had increased; and on the next day there was complete paralysis and coma, and death occurred on the fifth day after the onset of the symptoms.

'The second dog showed the first symptom on the ninth day after inoculation, when it was very dull and partially paralysed; its bark was characteristic. Next day the paralysis was almost complete, and on the twelfth day the animal died. This was therefore a case of the rapid paralytic form; whilst in the first dog the disease was of the ordinary furious form of rabies terminating in paralysis.

'The third dog showed the first symptom on the ninth day after inoculation, and from that time became gradually paralysed, and died on the sixteenth day.

'The fourth dog showed the first symptom in from eight to nine days after inoculation, and during the first day was extremely aggressive; on the two following days the characteristic bark was observed; and on the twelfth day there was paralysis of the hind legs; it died on the thirteenth day. Thus the furious form and the paralytic or dumb form of rabies were represented in equal numbers, whereas, in the usual mode of infection by biting, the former is more prevalent.

'The *post-mortem* appearances were as follows: The brain and central nervous system were in some of the dogs the seat of considerable congestion, in others these organs appeared normal. The serous membranes were perfectly normal; the larynx especially and, sometimes, the pharynx were congested; the lungs always congested, especially in the lower lobes; the heart normal; the blood usually fluid, occasionally with *post-mortem* clots; the stomach was always found to contain foreign bodies, such as straw; and its mucous membrane was congested, frequently showing numerous hæmorrhages; the small intestine was always empty, and the large glandular organs showed venous congestion.

'For the purpose of exact comparison of the disease just described with that produced when rabies is communicated to the rabbit in the ordinary way, some rabbits previously

¹ 'In some, signs of *post-mortem* digestion were found.'

narcotised with ether were caused to be bitten by rabid dogs of the streets, or were inoculated by trephining with material obtained from the spinal cord of dogs or other animals which had died of rabies, and in one instance from that of a man who had died with hydrophobia.

Four series of experiments were made in which rabbits were bitten by rabid dogs from the streets. In one of them the dog by which the rabbit was bitten exhibited the dumb form, in others the furious form of the disease. In each series excepting the first a large proportion of the rabbits died; the symptoms presenting themselves in these cases were identical with those observed in the rabbits inoculated from M. Pasteur's virus, but the duration of the symptoms was usually longer. As has been stated, rabbits inoculated by M. Pasteur's virus rarely show symptoms during more than three days before death, whereas the rabbits bitten by rabid dogs from the streets often live for a week after the appearance of the first symptoms.

The *post-mortem* appearances in the rabbits dying after having been bitten by rabid dogs of the streets were the same as those already described in rabbits inoculated with the virus from M. Pasteur's rabbits.

In the case of rabbits inoculated by trephining with the virus from animals dying of rabies of the streets, the incubation period was from fourteen to twenty-one days. In all cases the symptoms were similar to those produced by M. Pasteur's virus, and those of rabbits bitten by rabid dogs from the streets, but in the prolongation of the disease approached more closely in character to the latter.

The results of these experiments confirm several of the chief observations made by M. Pasteur, especially—

1. That the virus of rabies may certainly be obtained from the spinal cords of rabbits and other animals that have died of that disease.

2. That, thus obtained, the virus may be transmitted by inoculation through a succession of animals without any essential alteration in the nature, though there may be some modifications of the form, of the disease produced by it.

3. That, in transmission through rabbits, the disease is rendered more intense, both the period of incubation and the duration of life after the appearance of symptoms of infection being shortened.

4. That, in different cases, the disease may be manifested either in the form called dumb or paralytic rabies, which is usual in rabbits; or in the furious form usual in dogs; or in forms intermediate between or combining both of these, but that in all it is true rabies.

5. The period of incubation and the intensity of the symptoms may vary according to the method in which the virus is introduced, the age and strength of the animal, and some other circumstances; but, however variable in its intensity, the essential characters of the disease are still maintained.

The certainty that the virus of rabies can thus be transmitted without essential change made it desirable, in the next place, to ascertain whether, as M. Pasteur states, it can be so attenuated that it may be inoculated without risk to life, and whether animals thus inoculated are thus made safe from rabies.

The next point ascertained by M. Pasteur was the possibility of preventive or protective inoculation. The spinal cord of an animal dead of natural or experimental rabies, and subjected to drying, was found to lose virulence in the proportion of its drying, so that according to the period of drying, strong, medium, or weak virus may be obtained, the injection of the first producing rabies, and the latter producing slight or no appreciable result.

Animals bitten by a rabid dog, and soon after, i.e. in the early part of the incubation period, subjected to inoculation with the above-mentioned virus, can be prevented from developing the disease, the inoculation being carried out on repeated occasions, starting with virus of lesser virulence, i.e. from a cord dried longer, and following with injection of a cord of increasing virulence, and finishing with inoculation of a cord of intensive virulence, such as if at once used would unfailingly produce fatal rabies. Under this mode of treatment the animal does not contract the disease, and the effect of the original infection, i.e. by the bite of a rabid dog, is prevented. In a like manner intact dogs treated in this way and then subjected to infection with rabic virus (either by the bite of a rabid dog or by artificial injection with virulent

material), prove themselves completely *protected*. To test this important proposition the English Commission made the following experiments (*ibid.* pp. 2 and 3):—

‘Six dogs were “protected” by injecting subcutaneously the emulsions of spinal cords of rabbits which had died of rabies, beginning with that of a cord which had been dried for fourteen days and, on each following day, using that of a cord which had been dried for one day less, till at last that from a fresh cord was used.

‘None of these dogs suffered from the injections; and when they were completed, the six dogs thus “protected,” and two others unprotected, and some rabbits unprotected, were made insensible with ether, and were then bitten by rabid dogs, or by a rabid cat, on an exposed part.

‘A “protected” dog, No. 1, was bitten on July 8, 1886, by a dog which was paralytically rabid. It remains perfectly well.

‘An “unprotected” dog, No. 1, was bitten a few minutes afterwards by the same rabid dog, and died paralytically rabid.

‘A “protected” dog, No. 2, was bitten on November 6, 1886, by a dog which was furiously rabid; it remains well. At the same time, four “unprotected” rabbits were bitten by the same rabid dog, and of these two died of rabies in the usual form (i.e. 50 per cent. of animals bitten).

‘The same result followed with the “protected” dog, No. 3, and the “unprotected” rabbits, bitten at the same time. The dog still lives; the rabbits died of rabies.

‘The “protected” dogs, Nos. 4 and 5, were bitten on January 20, 1887, by a furiously rabid dog; and on the same day the “unprotected” dog, No. 2, and three “unprotected” rabbits were bitten by the same dog. The protected dogs remain well; the unprotected dog and two rabbits died with rabies (i.e. 75 per cent. of the animals bitten).

‘The “protected” dog, No. 6, was bitten on three different occasions by a furiously rabid cat on September 7, 1886; by a furiously rabid dog on October 7, 1886; and by another furiously rabid dog on November 6, 1886. It died ten weeks after being bitten for the third time, but not of rabies. It had been suffering with diffuse eczema during the whole of the time that it was under observation, and it died of this. At the *post-mortem* examination, no indication of rabies was found; and two rabbits inoculated by trephining with the crushed spinal cord showed no sign of rabies, either during life or when they were killed several months afterwards, in any appearance after death. It was thus made certain that the dog was not rabid.’

In these experiments it was found that the cord of a rabbit dead of the disease, dried at 20° C. over caustic potash for fourteen days, is the weakest degree of virulence to be employed; dried for one day the strongest; longer drying than fourteen days almost destroys the virus. Pasteur then proceeded to use this method for preventive inoculation of human beings bitten by rabid dogs (‘Report,’ p. 3, and pp. 4, 5, and 6).

‘The evidence that an animal may thus, by progressive inoculations, be protected from rabies suggested to M. Pasteur that if any animal or any person, though unprotected, were bitten by a rabid dog, the fatal influence of the virus might be prevented¹ by a timely series of similar progressive inoculations. He has accordingly, in the institution established by him in Paris, thus inoculated a very large number of persons believed to have been bitten by rabid animals; and we have endeavoured to ascertain with what amount of success he has done so.

‘The question might be answered with numerical accuracy if it were possible to ascertain the relative numbers of cases of hydrophobia occurring among persons of whom, after being similarly bitten by really rabid animals, some were and some were not inoculated. But an accurate numerical estimate of this kind is not possible.

‘1. It is often difficult, and sometimes impossible, to ascertain whether the animals by which people were bitten, and which were believed to be rabid, were really so. They may

¹ ‘The terms referring to “preventive” treatment will be used for that designed to prevent the occurrence of the disease in one already infected; those referring to “protective” treatment for that designed to protect a man or an animal from the risk of becoming infected. And it may be well to state that, though the usual custom is followed of employing the name of “hydrophobia” for the disease in men, and of “rabies” for that in animals, they are really the same disease.’

have escaped, or may have been killed at once, or may have been observed by none but persons quite incompetent to judge of their condition.

'2. The probability of hydrophobia occurring in persons bitten by dogs that were certainly rabid depends very much on the number and character of the bites; whether they are on the face or hands or other naked parts, or if they have been inflicted on parts covered with clothes, their effects may depend on the texture of the clothes and the extent to which they are torn; and in all cases the amount of bleeding from the wounds may affect the probability of absorption of virus.

'3. In all cases, the probability of infection from bites may be affected by speedy cauterising or excision of the wounded parts, or by various washings or other methods of treatment.

'4. The bites of different species of animals, and even of different dogs, are probably, for various reasons, unequally dangerous. Last year, at Deptford, five children were bitten by one dog and all died; in other cases a dog is said to have bitten twenty persons, of whom only one died. And it is certain that the bites of rabid wolves, and probably that those of rabid cats, are far more dangerous than those of rabid dogs.

'The amount of uncertainty due to these and other causes may be expressed by the fact that the percentage of deaths among persons who have been bitten by dogs believed to have been rabid, and who have not been inoculated, or otherwise treated, has been, in some groups of cases, estimated at the rate of only 5 per cent., in others at 60 per cent., and in others at various intermediate rates. The mortality from the bites of rabid wolves, also, has been, in different instances, estimated at from 80 to 95 per cent.

'To ascertain, as far as possible, the influence of these sources of fallacy in cases inoculated by M. Pasteur, the members of the committee who went to Paris requested him to enable them to investigate, by personal inquiry, the cases of some of those who had been treated by him. He at once, and very courteously, assented, and the names of ninety persons were taken from his note-books. No selection was made, except that the names were taken from his earliest cases, in which the periods since inoculation were longest, and from those of persons living within reach in Paris, Lyons, and St. Etienne.

'The notes made on the spot concerning all these cases are given in the Appendix (p. 8), and they include, as far as was possible, the evidence whether the dogs deemed rabid were really so, the situation and kind of bites, the immediate treatment of them, the statements of medical practitioners and veterinary surgeons to whom any useful facts were known.

'Among the ninety cases there were twenty-four in which the patients were bitten on naked parts by undoubtedly rabid dogs, and the wounds were not cauterised or treated in any way likely to have prevented the action of the virus; there were thirty-one in which there was no clear evidence that the dog was rabid; others in which the bite, though inflicted by undoubtedly rabid animals, having been through clothes, may thus have been rendered harmless. Among these, therefore, it is probable that, even if they had not been inoculated, few would have died. Still, the results observed in the total of the ninety cases may justly be compared with those observed in large numbers of cases similar to these as regards the uncertainties of infection, but not inoculated. The estimates published as to the mortalities in such unassorted cases are, as we have said, widely various. We believe that among the ninety persons, including the twenty-four bitten on naked parts, not less than eight would have died if they had not been inoculated. At the time of the inquiry, in April and May 1886, which was at least eighteen weeks since the treatment of the bites, not one had shown any signs of hydrophobia, nor has any one of them since died of that disease.

'Thus, the personal investigation of M. Pasteur's cases by members of the committee was, so far as it went, entirely satisfactory, and convinced them of the perfect accuracy of his records.

'After the first few months in which M. Pasteur practised his treatment, he was occasionally obliged, in order to quiet fears, to inoculate persons who believed that they had been bitten by rabid animals, but could give no satisfactory evidence of it. It might, therefore, be deemed unjust to estimate the total value of his treatment in the whole of his cases as being more than is represented by the difference between the rate of mortality observed in them and the lowest rate observed in any large number of cases not inoculated. This lowest rate may be taken at 5 per cent. Between October 1885 and the end of December 1886 M. Pasteur inoculated 2,682 persons, including 127 who went from this country (*see* Appendix, p. 19). Of the whole number, at the rate of 5 per cent., at least 130 should have died. At the end of 1886 the number of deaths stated by M. Vulpian, speaking for M. Pasteur, was thirty-one, including seven bitten by wolves, in three of

whom the symptoms of hydrophobia appeared while they were under treatment, and before the series of inoculations were complete. Since 1886 two more of those inoculated in that year have died of hydrophobia.

The number of deaths assigned by those who have sought to prove the inutility of M. Pasteur's treatment is, as nearly as we can ascertain, forty out of the 2,682; and in this number are included the seven deaths from bites by wolves, and probably not less than four in which it is doubtful whether the deaths were due to hydrophobia or to some other disease. Making fair allowance for uncertainties and for questions which cannot now be settled, we believe it sure that, excluding the deaths after bites by rabid wolves, the proportion of deaths in the 2,634 persons bitten by other animals was between 1 and 1.2 per cent.; a proportion far lower than the lowest estimated among those not submitted to M. Pasteur's treatment, and showing, even on this lowest estimate, the saving of not less than 100 lives.

The evidence of the utility of M. Pasteur's method indicated by these numbers is confirmed by the results obtained in certain groups of his cases.

Of 233 persons bitten by animals in which rabies was proved, either by inoculation from their spinal cords, or by the occurrence of rabies in other animals or in persons bitten by them, only four died. Without inoculation it would have been expected that at least forty would have died.

Among 186 bitten on the head or face by animals in which rabies was proved by experimental inoculations or was observed by veterinary surgeons, only nine died, instead of at least forty; and of the forty-eight bitten by rabid wolves only nine died, while, without the preventive treatment, the mortality, according to the most probable estimates yet made, would have been nearly thirty.

Between the end of last December and the end of March, M. Pasteur inoculated 509 persons bitten by animals proved to have been rabid, either by inoculation with their spinal cords, or by the deaths of some of those bitten by them, or as certified by veterinary surgeons. Only two have died, and one of these was bitten by a wolf a month before inoculation, and died after only three days' treatment. If we omit half of the cases as being too recent, the other 250 have had a mortality of less than 1 per cent., instead of 20 or 30 per cent.

It has been objected that the number treated by M. Pasteur, which, from October 1885 to the end of 1886, included 1,929 French and Algerians, was much greater than could reasonably be supposed to have been bitten by rabid animals. But there had hitherto been no careful registration of such cases, and the numbers that have occurred in the present year are not less than in the same part of last year, when the alarm about hydrophobia was greatest.

From the evidence of all these facts, we think it certain that the inoculations practised by M. Pasteur on persons bitten by rabid animals have prevented the occurrence of hydrophobia in a large proportion of those who, if they had not been so inoculated, would have died of that disease. And we believe that the value of his discovery will be found much greater than can be estimated by its present utility, for it shows that it may become possible to avert by inoculation, even after infection, other diseases besides hydrophobia. Some have, indeed, thought it possible to avert small-pox by vaccinating those very recently exposed to its infection, but the evidence of this is, at the best, inconclusive; and M. Pasteur's may justly be deemed the first proved method of overtaking and suppressing by inoculation a process of specific infection. His researches have also added very largely to the knowledge of the pathology of hydrophobia and have supplied what is of the highest practical value, namely, a sure means of determining whether an animal, which has died under suspicion of rabies, were really affected with that disease or not.

The question has been raised whether M. Pasteur's treatment can be submitted to without danger to health or life, and in answering it, it is necessary to refer to two different methods of inoculation which he has practised, and which are fully described in the Appendix (p. 21).

In the first, which may be called the ordinary method, and which has been employed in the very large majority of cases, the preventive material obtained from the spinal cord of rabbits that have died of rabies derived originally from rabid dogs is injected under the skin once a day, for ten days, in gradually increasing strengths.

In the second or intensive method, *méthode intensive*, which M. Pasteur adopted for the treatment of cases deemed especially urgent, on account either of the number and position of the bites or of the long time since their infliction, the injections, gradually increasing in strength, were usually made three times on each of the first three days, then once daily for a week, and then in different degrees of frequency for some days more. The highest strength of the injections used in this method was greater than the highest used in the

ordinary method, and was such as, if used at first and without the previous injections of less strength, would certainly produce rabies.

'By the first or ordinary method, there is no evidence or probability that anyone has been in danger of dying, or has in any degree suffered in health even for any short time. But after the intensive method, deaths have occurred under conditions which have suggested that they were due to the inoculations rather than to the infection from the rabid animal.

'There is ample reason to believe that, in many of the most urgent cases, the intensive method was more efficacious than the ordinary method would have been. Thus, M. Pasteur mentions that, of nineteen Russians bitten by rabid wolves, three treated by the ordinary method died, and the remaining sixteen, treated by the intensive method, survived; and he contrasts the cases of six children, severely bitten on the face, who died after the ordinary treatment with those of ten similarly bitten children who were treated by the intensive method, and of whom none died; and M. Vulpian reports that of 186 persons badly bitten by animals that were most probably rabid, fifty treated by the intensive method survived, and of the remaining 136 treated by the ordinary method nine died.'

Although several observers have mentioned that they have identified the microbe of hydrophobia in the form of a coccus, this has not been as yet confirmed, and the nature of the virus is still undiscovered.

As to Pasteur's views on the nature of the rabic virus and the methods of preventive inoculation, we quote here from the 'Report of the English Pasteur Commission,' pp. 21 and 22:—

'M. Pasteur believes that the virus of rabies is a living micro-organism, and that, like some others, it produces in the tissues it invades an excretory substance by which, when present in sufficient quantity, its own development and increase are checked, as are those of the yeast ferment by the alcohol produced in the vinous fermentation. In accordance with this theory, he thinks that the spinal cords of animals that have died of rabies contain both the virus and this excretory substance, which, practically, may be deemed its antidote. He believes, therefore, that by injections of an emulsion from such spinal cords into the systems of animals bitten or inoculated with the virus of rabies, the antidote may be able, during the period of incubation, to arrest and prevent the fatal influence of the virus. But, in order to avoid the possibility of injecting a still potent virus, M. Pasteur holds that the virus in the spinal cord must be weakened by drying the cord in a pure and dry atmosphere at a temperature of 20° C., in which drying the efficiency of the antidote may be reduced to a much less extent than the potency of the virus. By such drying this potency may be so reduced that an emulsion of the dried spinal cord may be injected without any risk of producing rabies, and this risk is in no measure increased by the daily injections of emulsions from cords dried during a gradually less number of days, and which, though more virulent than those first used, still contain a larger proportion of the antidote than of the virus.

'In accordance with this theory, the method of the preventive injections first used by M. Pasteur was adjusted in the following manner:—

Days of Inoculation	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th
Days during which the spinal cord had been dried	14	13	12	11	10	9	8	7	6	5

'In consequence of some deaths among those who had been thus treated, M. Pasteur deemed it necessary, in cases of very severe bites, and of persons bitten long before the treatment could be commenced, to increase the intensity of the treatment by more speedily increasing the strength of the injections, by more frequent repetitions of them, and by using on certain days spinal cords dried during only three, two, and one day. Thus in September and October 1886 he adopted the following formula:—

Days of Inoculation	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
Days' drying of the cords	14, 13, 12	11, 10, 9	8, 7	6, 5	4, 3	2	1	6, 5	4, 3	2	1

'In very severe and perilous cases this course was repeated even three or four times. It was distinguished as the *méthode intensive*, and among such severe cases it was followed by a marked diminution of mortality. But when it appeared possible that it might be dangerous, M. Pasteur changed it for that which he now uses, and which may be thus represented:—

Days of Inoculation	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th	12th	13th	14th	15th
Days' drying of the cords	14, 13	12, 11	11, 10	10, 10	9, 9	9	8	8	8	7	7	7	6	6	5

'The material for injection is prepared by crushing portions of the dried spinal cord, and diffusing them in sterilised broth free from all risk of putrefaction, decomposition, or any change due to the presence of other micro-organisms; and the injection is made with syringes through fine tubular needles into the subcutaneous tissue.

'For transmission of rabies through rabbits, in order to obtain the spinal cords required for its prevention in other animals, injections of virus of highest intensity are made through minute holes in the skull into the space under the dura mater or fibrous covering of the brain.

'The materials for the protective inoculations are prepared in the same manner as those for the preventive, from spinal cords dried from ten days to one day.'

CHAPTER XXV

GLANDERS AND FARCY—SYPHILIS—RHINOSCLEROMA

GLANDERS is a subacute or chronic infectious disease of the nasal mucous membrane, respiratory organs, and skin in the horse, leading to death; when the seat of the pathological process is localised in the skin it is spoken of as *farcy*, but both are the same disease. Glanders is communicable to the ass, mule, and man, and leads sooner or later to death; in man it is generally acute, though also in horses it sometimes assumes the acute form. The disease consists of nodular deposits in the mucous membrane of the nose, the septum particularly. The young nodules are grey, deep seated, not larger than a hemp seed or millet seed, and surrounded by congested mucous membrane. Microscopically they consist of an aggregation of round cells of various sizes, and in this phase are in structure not easily distinguishable from young tubercles; they soon enlarge, their central parts becoming purulent, and hereby small abscesses are formed which open on the free surface. Where close together they become confluent and form large ulcers, irregular in shape, with a grey purulent depressed centre, and a raised, injected border, the tissue around them being congested and infiltrated. The mucous membrane of the septum becomes riddled with abscesses, and soon also the cervical lymph glands become swollen and purulent; the process gradually extends to the pharynx, larynx, trachea, and lungs. In this latter numerous nodules appear, at first small (hemp seed) and grey, but gradually enlarging and transforming into abscesses situated in the depth of the lung, as well as on the surface. Neighbouring nodules and abscesses become confluent, and in this way extensive cavities are formed, whose purulent contents discharge themselves into the bronchi, the tissue around them being much congested and infiltrated. In the skin, the cutis and subcutaneous tissue are the seat of the nodules; here also they are at first small and grey, and represent round cell aggregations, which gradually enlarge into the congested and infiltrated surrounding tissue; purulent change is also here the natural

result, and by opening on the free surface they form open sores discharging a little pus; the centre is depressed, covered with greyish purulent matter; the periphery is raised and much infiltrated.

The general mode of infection seems to be that of inoculation. It appears to be doubtful whether the direct transmission of the glanders material on to the intact nasal mucous membrane can produce infection, since such a mode yields experimentally no result; but cutaneous and subcutaneous inoculation in horses and asses is always followed by the characteristic disease of the nasal mucous membrane.

Horses and asses are very susceptible; of carnivorous animals, glanders has been observed in feline animals (lions and tigers fed on flesh of glandered horses); cats, dogs, and sheep are only very slightly susceptible, but in goats, glanders has been observed; in cattle, glanders is unknown. Rodents are easily infected by inoculation (*see* the experiments of Löffler and Schütz).

In man, glanders occurs after infection from the horse, generally through a cutaneous wound; it generally runs an acute course, characterised by the appearance of purulent infiltration about the seat of infection of the skin, particularly the muscular tissue, then in the lung and respiratory mucous membrane; metastatic purulent infiltration occurs also in the joints, the liver, spleen, kidneys, and testes. Löffler and Schütz, as also Israel, were the first to show that in glanders the deposits constantly contain a definite species of bacilli; they occur generally isolated, and in small groups between, and also enclosed in, the cells of the nodules: they are more numerous in the nodules which have not become purulent; after the nodules have become purulent the number of the bacilli in them diminishes. The bacilli are fine rods, of about the same size as tubercle bacilli, but a little thicker, rounded at their ends, straight, or sometimes more or less curved; this latter is especially noticed when they lie in groups; their substance is either homogeneous, or, like that of the tubercle bacilli, shows segregation of the protoplasm into granules within the sheath. The bacilli stain best in alkaline methylene blue and subsequent washing in acidulated water (acetic acid 1 per cent.); also in alkaline fuchsin of Ehrlich, or in gentian violet aniline water. The bacilli are easily cultivated at 35–38° C. on blood serum, agar mixture, and potato. On boiled potato at 35° C., they form a characteristic brownish yellow amber-coloured sticky film. On solid blood serum at 37° C., after three days, one notices small translucent droplets slightly projecting over the general surface. These are the youngest colonies. On agar culture the colonies are also translucent greyish droplets, gradually flattening and becoming dark in the centre (Plate XXXI., fig. 50).

There is no difficulty in obtaining good cultures in the ordinary beef broth peptone gelatine kept at 20–21° C., as also on potato at this temperature. They form on ordinary gelatine whitish-grey, flat, round, disc-shaped colonies. The gelatine is only very slowly liquefied.

According to Raskina the glanders bacilli grow also at 18–20° C., on gelatine, milk, serum, and white of egg. Kranzfeldt grew them also on glycerine agar mixture.

Löffler and Schütz proved that the artificial cultivations inoculated into horses and asses produced typical glanders. On most white mice the bacilli do not act, nor does fresh glanders material directly taken from the horse; ¹ wild mice (field mice), however, are very susceptible to inoculation with the

¹ H. Leo (*Zeitschrift f. Hygiene*, VII., 3) succeeded in giving glanders to white mice after feeding them for days with phloridzine, whereby their tissues contained much sugar.

cultures; they die within eight days, and their spleen and liver are riddled with yellowish-grey minute nodules containing abundant glanders bacilli. In the rabbit, subcutaneous inoculation produces generally a positive result; in most cases, however, only a local abscess is formed which leads to a rapidly healing sore. In guineapigs, both the fresh glanders material and the culture produce a characteristic disease: on the third or fourth day a sore is found at the seat of inoculation, which soon involves the nearest lymphatics, these being found swollen and congested; further, the testes or ovaries become much swollen, congested, and the seat of minute glanders nodules; so does the skin and the nasal mucous membrane, leading to purulent infiltration and, after the discharge of the pus, to ulceration. The spleen contains white nodules. The glanders bacilli are everywhere present in the deposits.

Glanders bacilli of cultures are killed by prolonged drying (in about fourteen days); the glanders material taken direct from the horse becomes innocuous after a few days' drying: these facts seem to indicate that the bacilli do not form spores. With this the observation of Löffler also agrees, that the cultures of the bacilli die after a few months, and Cardéal and Malet found that putrefaction destroys the bacilli, though only after many days. Löffler studied also the resistance of the bacilli to heat, and he found that, for instance, ten minutes' heating to 55° C. completely killed the bacilli of the cultures; in this respect the glanders bacilli are even less resistant than many other non-spore bearing bacilli. Further, Löffler found that perchloride of mercury (1:5,000) kills the bacilli in two minutes, and carbolic acid (8 to 5 per cent.) in five minutes. All these facts strongly indicate that no spore formation takes place.

Within quite recent times it has been shown by a series of observations, carried out by a number of workers,¹ that chemical substances present in the artificial cultures of the glanders bacilli (Mallein) injected into horses produce a definite reaction, viz., a decided rise of temperature, if the animals are affected with glanders; but no reaction follows in healthy horses. So that in doubtful cases the injection of the Mallein determines the diagnosis. The Mallein is prepared in the same way as Koch's tuberculin (*see below*), and is a further instance of the vast importance of the study of the chemistry of pathogenic bacteria.

The literature on the subject of the glanders bacilli is very copious, but the following are the chief works:—

- Löffler and Schütz: 'Deutsche med. Wochenschrift,' 1882, No. 52.
 Israel: 'Berliner klin. Woch.,' 1883, No. 11.
 Kitt: 'Jahreschr. der. kgl. Thierarznei in München,' 1883-4.
 Weichselbaum: 'Wiener med. Wochenschrift,' 1885, Nos. 21-24.
 Löffler: 'Arbeiten aus d. kais. Gesundheitsamte,' 1886.
 Baakins: 'Zeitschrift f. wiss. Mikroskopie,' IV. 1887.
 Kranzfeldt: 'Centralbl. f. Bacteriologie und Parasit.,' II. 1887.

SYPHILIS

The primary syphilitic sore or chancre is a localised inflammation in an abrasion—the seat of infection—with subsequent induration, necrosis, and ulceration of the superficial layer or layers. Under the microscope the connective tissue of the cutis or mucosa, as the case may be, is densely infiltrated

¹ Kalning, *Archiv f. Veterinärwiss.*, I. 1891, St. Petersburg; Preusse, *Berliner thierärztl. Wochenschr.*, No. 29, 1891; Heyne, *Berl. thierärztl. Wochenschr.*, 1891, Nos. 83 and 89; Pearson, *Zeitschr. f. Veterinärk.*, No. 5, 1891.

with round cells; most of these do not differ in size from white blood corpuscles, but some are larger, have two or three nuclei and a considerable amount of protoplasm, and in size of the cell and number of the nuclei correspond to giant cells. The spreading of the induration consists in the infiltration involving larger and larger areas, while at the same time the superficial layers undergo necrosis and disintegrate into a *débris*, whereby an open sore is established. When the chancre heals, the round cells infiltrating the corium are gradually absorbed and disappear, the fibrous connective tissue becomes more abundant, and ultimately by contracting, forms a dense cicatrix. The secondary symptoms are established by acute localised inflammations of the skin, lymphatic glands, and mucous membranes: the tertiary by round cell infiltration, and chronic inflammation in the skin, bones, brain, and viscera. In these inflammations and infiltrations the microscopic appearances do not differ from other non-syphilitic inflammations. Gummata and condylomata, or mucous patches, are localised inflammations, i.e. round-cell infiltrations, and distended and engorged vessels; the epithelium of the surface and the corium or mucous membrane being also the seat of infiltration with round cells; in fact, the disease is similar to what is called in pathology a *granuloma*. The fate of the gummata and condylomata is fatty degeneration, caseation, absorption, and cicatrization.

As to the etiology of syphilis, various microbes have been described. Foremost is the bacillus of Lustgarten,¹ which is by many considered to be the bacillus of syphilis. Lustgarten found in the tissue of primary syphilitic sores certain bacilli, generally in comparatively small numbers: they resemble in size and staining power the tubercle bacilli, are straight or slightly curved, sometimes thicker at the end than in the middle, uniform or granular; they occur chiefly in pairs within the protoplasm of the round cells, but occasionally also between the cells. These bacilli have this peculiarity, that they can be stained by a special method: sections of chancre are placed in gentian violet aniline water, are then decolourised in solution of permanganate of potassium; by this all the elements of the tissue lose the stain except the bacilli. De Giacomini achieved the same result by decolourising the sections (after staining) in solution of ferric chloride. While numerous observers (Doutrelepont and Schütz, Matterstock, E. Fraenkel) have found the bacillus of Lustgarten in the syphilitic foci of the skin and mucous membrane of the mouth, and in the syphilitic gummata of the viscera, others like Alvarez and Tavel, Klemperer, Kübner, Zeissl, Baumgarten, and others have searched for them after the approved methods, but have failed to find them. Besides, Alvarez and Tavel² have proved that bacilli identical with Lustgarten's bacilli in size, aspect, and peculiar staining power occur in the normal sebum or smegma of the genital organs, and this has been confirmed by other observers, so that the claims of Lustgarten's bacilli to be considered as the bacilli of syphilis rest at present on insufficient evidence.

There is also no doubt that though many competent observers have searched for them in the syphilitic tissues in congenital syphilis, they have failed to find them. Kassowitz and Hochsinger described in the cutaneous papules in hereditary syphilitic children, streptococci plugging almost completely the small vessels of the corium; Eve and Lingard³ described, in syphilitic tissues and in the blood, bacilli resembling the tubercle bacilli in size and aspect, and have cultivated them on blood-serum; they do not show any peculiarities in staining.

¹ *Die Syphilisbacillen*, Wien, 1885.

² *Bull. de l'Acad. de Méd.*, August 1885.

³ *Lancet*, No. 15, 1886.

Finally, Disse and Taguchi¹ describe capsulated diplococci, which they found in syphilitic tissues; they cultivated them, and with such cultures produced by inoculation of rabbits, dogs, and sheep, a chronic disease which they consider analogous to syphilis; but all attempts to produce a definite disorder in any animal by means of inoculations of veritable syphilitic tissue, hitherto attempted by many competent observers, have yielded only negative results.

RHINOSCLEROMA

A peculiar chronic disease, starting in the nasal and pharyngeal mucous membrane and extending to the outer nose (skin) and downwards into the larynx and trachea, prevalent in Austria and in some Eastern countries. It is essentially a firm, nodular swelling, consisting in a circumscribed infiltration with round cells of the mucous membrane and skin; in the former it leads to degeneration of the superficial layers, including the epithelium of the surface, and to ulceration. In the skin the nodular swelling of the corium persists for a long time without leading to ulceration. In a microscopic section the connective tissue is densely infiltrated with round cells; among these there occur peculiar hyaline globules, larger than the round cells. These were first recognised by Mikulicz,² and are known as Mikulicz' cells. Mikulicz, Dittrich, Walkanitsch, and others consider that these Mikulicz cells are produced by hyaline degeneration and swelling, with solution of the nucleus, of certain epitheloid cells of the tissue, not the ordinary round cells. In sections through rhinoscleroma, stained with methyl blue, the Mikulicz cells are easily recognised by their deep blue staining; some are many times larger than the ordinary round cells of the tissue, others are much smaller, and of no greater diameter than a leucocyte. Some are only a single, large, homogeneous sphere; others are made up of two or three small homogeneous spheres, but one can distinguish in all of them a thin pellicle of granular investment, including at one place a flattened nucleus; where the cells are made up of two or three smaller spheres, these latter have only a common investment. These Mikulicz cells resemble, therefore, fat cells; but instead of a fat droplet or fat droplets, there is inside them a homogeneous colloid material. We therefore think that the general statement, that these Mikulicz cells are hyaline degenerated cells, is only partially correct, but think rather that some of the cells of the scleroma tissue form at the expense of their protoplasm hyaline globules, in the same manner as certain cells form fat droplets; the nucleated investment would represent the remnant of the original cell protoplasm. A. von Frisch³ was the first to show that in the tissue of rhinoscleroma, particularly in the Mikulicz cells, there occur small oval bacilli, either single or as dumb-bells. He cultivated them and used them for inoculations on animals, but without result. Cornil and Alvarez⁴ then showed that the rhinoscleroma bacilli possess a gelatinous capsule, and therefore resemble the pneumonia bacilli of Friedländer (see a former chapter). Dittrich has made extended experiments and observations on these rhinoscleroma bacilli, and showed that morphologically and culturally they are distinguishable, but only with difficulty, from Friedländer's bacilli; though he maintains that in some minute details as to staining and as to appearance in gelatine cultures the one can be distinguished from the other. This is, however, not admitted

¹ *Deutsche med. Woch.*, No. 14, 1886.

² *V. Langenbeck's Archiv f. kl. Chir.*, XX., p. 485.

³ *Wiener med. Wochenschrift*, No. 32, 1882.

⁴ *Archives de Physiologie normale et path.*, VI., 1885.

by many observers. Alvarez, Paltauf and Von Eiselsberg, Wolkowitsch, and Dittrich found these bacilli also in the lymphatics of the surrounding tissue. Paltauf and Von Eiselsberg,¹ then Dittrich, Babes, and others, produced in guineapigs, mice, and rabbits a septicæmic infection similar to that producible by Friedländer's bacilli, but no chronic nodular disease.

The constant presence, then, of the capsulated rhinosclerom bacilli in the scleromatous tissue, particularly the Mikulicz cells, is a fact of which there can be no doubt, but it is equally a fact that they are identical with the bacilli of Friedländer; their causative relation to the rhinoscleromatous process is, therefore, more than doubtful, or at any rate not sufficiently supported.

CHAPTER XXVI

TUBERCULOSIS

TUBERCLE is, after Virchow, a granuloma or a round-cell new growth, at first grey and translucent, then undergoing from the centre outwards a necrotic change leading to degeneration or caseation; the part degenerated is yellow. This necrotic or caseous change extending from the centre, while the periphery increases by the deposit of new round-cell elements, wider and wider areas of tissue become involved, and thereby both the grey periphery and the yellow or caseous centre enlarge in diameter. The caseous portion softens by suppuration, so that the tubercle becomes gradually changed into an abscess, the suppuration being sustained by the peripheral portion of the tubercle. Tubercles differ in structure: some, e.g. those of the lung, liver, and lymph glands of man, are in most instances, in the early or grey stage, typical granulomata or round-cell new growths in the connective tissue; then they become reticulated, and, instead of finding dense aggregations of round cells between delicate bundles of fibrous tissue, we find a more or less regular reticulum of cells; in the peripheral part the reticulum is somewhat arranged like a capsule of concentric layers; in the reticulum of the centre we find large irregularly shaped, cylindrical or spherical cells, containing numerous nuclei, generally disposed in an annular zone in or near the periphery of the cell protoplasm; in such reticular tubercles it is the central portion containing the giant cells that first undergoes necrosis and caseation. Some tubercles never assume this reticular condition, but pass directly from the grey stage, or granuloma, into the caseous or yellow tubercle. Then there are tubercles, e.g. some forms of acute miliary tubercles in children, when the lung is found studded with grey tubercles, which do not at all resemble granulomata, being in reality groups of air cells filled with, and distended by fibrinous deposits with leucocytes in the centre. There are sometimes tubercles in which few or no giant cells are found, e.g., tubercles in the brain, leading to suppuration and formation of abscess, tubercles of the spleen and of the liver; while in other cases the tubercles in the same viscera contain abundance of giant cells, chiefly in connection with the reticular form. In bovine tubercle, in tubercle of the fowl, and in artificial tuberculosis in guineapigs, giant cells are abundant in the tubercles, which do not present the reticular form; they are present in tubercles of man in the lung, liver, membranes of the brain, spleen, and marrow of bone. Most tubercles are at first granulomata in the connective tissue; i.e. derived from connective-tissue

¹ *Fortschr. d. Med.*, No. 19, 1889.

cells (Virchow), as well as white blood corpuscles (Cohnheim). This is the case with the tubercles occurring in the lymph follicles of the lymph glands, and of the intestine (Peyer's and solitary glands), in the Malpighian corpuscles of the spleen, in the peribronchial lymph follicles, in the connective tissue of the pia mater of the brain, and in the interlobular connective tissue of the liver: other tubercles owe their origin partly to round cells, and partly to a multiplication of epithelial or endothelial cells; such is the case with the tubercles in the alveolar cavities of the lung, in the interlobular bile ducts, in the lymphatic vessels and lymphatic sinuses, in the intima of large blood-vessels (aorta), and in the glands of the mucous membranes and skin. Amongst the cells constituting a tubercle the gradual enlargement of at first small round cells (lymph cells or leucocytes) into larger epithelial-like cells, with one or more large nuclei, can be distinctly inferred from all transitional forms between the two, and therefore the large cells with large nuclei, though resembling in aspect epithelial cells, cannot be declared to be epithelial in origin; but in tubercles formed in connection with epithelial and endothelial tissues (*see above*), epithelial cells do contribute to the formation of the cells constituting the tubercle.

Then, again, while in some tubercles, necrosis and caseation, though extending to a large extent, do not lead to suppuration—e.g. in the liver and spleen—there are others which, sooner or later, after caseation has set in, commence to soften and suppurate, e.g. the tubercles of the lung in man, in cattle, in artificial tuberculosis of animals, the tubercles of the lymph glands, and the tubercles of bone-marrow in man. All tubercular masses (nodules, patches, and streaks) sooner or later caseate, i.e. the tissue undergoes necrotic change; the cells become opaque and change into granular *débris*, not taking a stain; no nuclei are visible in them: fatty and other coarse granules are, however, numerous; further, calcareous deposits may take place in these caseous masses if they do not become softened and absorbed.

It is impossible to recognise by their structure under the microscope some tubercles as 'tubercles,' if they are only in the condition of granulomata, or if they are suppurating and without any giant cells; but if reticular and with giant cells, the presence of these latter would generally decide the diagnosis. As we shall presently see, however, there is an infallible test for diagnosis, and that is the presence of the tubercle bacilli of Koch. But from microscopic structure alone some tubercles, notably those without giant cells, cannot easily be identified. Of course there are in many cases sufficient indications of a gross anatomical nature, e.g. the disseminated nature in the different lymph glands and viscera, the condition of caseation, &c., which enable us to form a fairly correct diagnosis, but in cases in which only in one or the other organ we find a nodule, caseous or not, the diagnosis is very difficult, in fact in many such instances it cannot be made; then the bacteriological examination alone is decisive. No doubt many pathologists have had not a few times to examine deposits, few in numbers, in the lungs and liver of man and animals (fowls, cats, sheep) which were firm and caseous, some even calcareous, and where the histological examination was insufficient to permit of a correct diagnosis; it is an every-day experience of those whose practice lies more in the direction of diseases of the chest that it is difficult in some cases to make a correct diagnosis from the symptoms and physical appearances of the lung affection; yet the test for the tubercle bacilli at once makes the diagnosis certain. Not long ago a case of multiple brain abscess occurred, and the patient died. There were no tubercles in any of the viscera of the chest or abdomen. The histological examination of the infiltrated tissue forming the wall of the abscess did not show any appearance.

characteristic of tubercle—no giant cells, no localised granulomata ; the tissue was uniformly infiltrated with round cells, gradually becoming denser towards the interior ; no diagnosis of tubercle could be made from the histological characters, and yet by testing for the tubercle bacilli, and finding them in large numbers in and amongst the round cells, the diagnosis was at once made certain.

The first decisive experimental proof that tuberculosis is a communicable disease has been given by Klencke and Villemin,¹ the latter showing that if guineapigs are inoculated with tubercular matter, such as sputum derived from a tuberculous patient, a chronic disease is produced, which has the distinct characters of disseminated tuberculosis in the lymph glands, the lungs, the serous membranes, the liver, and the spleen. The deposits are at first minute and grey, not larger than a pin's head ; they gradually enlarge and caseate in the centre, the caseation spreading over the whole tubercle. Wilson Fox, Burdon-Sanderson, Klebs, Cohnheim, Chauveau, and others have repeated and confirmed these experiments. Inoculations with bovine tubercular matter were also made on guineapigs and rabbits, and true disseminated tuberculosis produced. The feeding of pigs, guineapigs, and rabbits with tubercular matter, both human and bovine, produced disseminated tuberculosis. The tubercular deposits of all such experimental animals when transferred to normal animals again produced tuberculosis.

When inoculation into the subcutaneous tissue of the groin of guineapigs is carried out with a minute particle of human tubercular material,² after a lapse of about fourteen days, more or less, the lymph gland nearest the seat of inoculation can be easily felt as a firm swollen nodule of the size of a pea ; after a lapse of a further ten or twelve days the first gland is much enlarged (size of a bean or filbert) and may already have become changed into an abscess firmly fixed to the skin, whilst one or other lymph gland near it can now be felt as a firm swollen nodule. The abscess soon opens, and discharges thick creamy pus, a persistent sore is established, and though it may from time to time become covered with a scab or crust, the accumulation of thick pus underneath soon causes it to be again opened. The other enlarged lymph glands about the seat of inoculation also become converted into abscesses. On killing the animal after four to six weeks, we find at the seat of inoculation an open sore discharging thick pus, the subcutaneous connective tissue around and for some distance being hyperæmic and œdematous. In connection with the sore we find a chain or a packet of swollen firm lymph glands (from the size of a split pea to that of a bean) containing cheesy, yellow deposits. On cutting into such a gland we find it very juicy, and containing larger or smaller yellowish masses ; in the largest glands some of these masses are found changed into thick creamy pus. At or about this stage, i.e. after four to six weeks, in most instances either no tubercles visible to the unaided eye are as yet found in the lungs, or only very few minute punctiform nodules ; in the enlarged spleen we find numbers of minute granules projecting above the surface of the capsule, thus making the surface uneven and rough. In the liver there are numerous minute grey, punctiform nodules, which in some places have a tendency towards confluence ; on section greyish streaks are recognised under a lens between the normal red liver tissue, and the whole organ is slightly enlarged. The omentum also shows numerous minute opaque patches, which are more numerous and larger

¹ *Etude sur la Tuberculose*. Paris, 1868.

² Any material derived from tubercle, no matter whether grey, caseous or purulent : caseous or purulent lymph gland, sputum or purulent matter of the caseous tubercles of the lung, tubercle of the spleen or liver, may serve the purpose.

than those normally found. The lymph glands in the porta hepatis are large and firm, so also those in the hilum of the spleen; the mesenteric glands are large and firm. In the marrow of long bones, grey and even caseous tubercles can be distinguished. If the animal is allowed to live, it will be found gradually getting thinner towards the third or fourth month, and it does not generally die before the end of the third or later than the end of the fifth month, the average duration being 100 to 120 days after inoculation. On *post-mortem* there is found a large discharging sore at the seat of inoculation, from which extends a chain or packet of lymph glands from about the size of a bean to a large filbert; on cutting into such a gland numerous yellow irregular cheesy deposits are seen, some softened and purulent. The lymph glands behind the sternum, at the root of the lung, of the porta hepatis, at the hilum of the spleen, of the mesentery, of the pelvic cavity, and of other localities are swollen, firm, and contain larger or smaller irregular patches of caseous deposit. The lungs are enlarged, and sometimes crowded with, at other times containing a limited number of, tubercular deposits of all sizes—from that of a millet seed to that of a pea—and in all stages; some grey with the beginning in the centre of yellow caseous change, others entirely yellow and caseous, most of them firm, the largest softened in the centre and representing the first trace of cavernæ; where crowded they become confluent. The spleen is greatly enlarged, of from four and five times to ten times its original size, riddled with caseous tubercles, in many places confluent into irregular yellow firm patches. The liver is permeated with greyish white to yellow nodules, streaks, and patches, the larger ones of a greenish yellow colour and firm. The omentum is one mass of confluent tubercular nodules and patches. The diaphragm contains whitish-grey streaks and patches of tubercle. So also the pleura. Rarely is there tubercle in the kidney.

Rabbits, inoculated subcutaneously in the inguinal region with human tubercular sputum, show very much less pronounced disseminated tuberculosis than guineapigs; after twelve to sixteen or more weeks the animals are found much emaciated; the lymph glands of the inguinal region are enlarged and caseous; in the lungs are few or no tubercles, in the liver a few tubercles, some grey, others yellow; the spleen is enlarged, and contains many tubercular deposits; the mesenteric and other abdominal lymph glands are swollen, firm, and caseous; and the process, on the whole, is very distinctly less intensive and extensive than in the guineapig. The writer has seen numerous cases in which, after twelve to sixteen weeks, the only organ containing numerous tubercles was the spleen, the liver contained only few, the lungs none.

The feeding of guineapigs and rabbits on human tubercular matter produces tuberculosis, but with this difference, that while in the guineapig it leads to general disseminated tubercular deposits, this is far less so in the rabbit. In the guineapig, if the animal is killed after six to eight weeks, we find distinct tubercular deposits in the wall of the small intestine, the tubercles being situated in the Peyer's glands of the ileum and the ileo-cæcal valve; they are of various sizes, and more or less caseous in the centre; the mesenteric glands are always enlarged, and contain firm caseous deposits. The liver also shows grey tubercular nodules and streaks; the spleen is slightly enlarged and granular. The whole process, judging from the amount and progress of the changes, starts in the lymphatic follicles of the ileum, and spreads thence into the mesenteric lymph glands, liver, and spleen. The lungs as yet show no tubercles. If the disease is allowed to run its course, the animal becomes greatly emaciated, and dies in about four or five months or earlier; we then find tubercles in all the lymph glands, in the viscera,

in the marrow of bone, and in serous membranes ; but the changes in the abdominal viscera are the most extensive, those of the thorax considerably less.

In the rabbit, on the other hand, feeding with human tubercular matter produces considerably less result ; in a large percentage of cases, even after many weeks, caseous tubercles are found only in the lower ileum and mesenteric glands. The spleen, liver, and lungs appear free, and only in a few cases are these organs involved, and then but to a small degree, viz. containing only few tubercles.¹

In the fowl, tuberculosis can be produced both by subcutaneous inoculation and by feeding with human sputum, although all animals are not equally susceptible. In most cases tubercles of the spleen, in others of the spleen and liver, are the result ; the intensity of the process in both these organs is striking only in a few very successful cases, and in these we find both those organs enlarged and containing numerous spherical, firm, white nodules, from the size of a millet seed to that of a pea. When superficial they project over the capsule. In many other cases tubercles are found only in the spleen. The remarkable fact is that in most instances, notwithstanding the tuberculosis going on in their spleen, the animals are very fat ; when, however, the liver becomes involved to a large extent, the animal is found emaciated.

In guineapigs, rabbits, and fowls, in all tubercular deposits giant cells are numerously met with. Tuberculosis can be produced in animals (guineapigs, rabbits) by intraperitoneal injection, and by inhalation. By a spray-producer, finely divided tubercular matter can be distributed in the air in which guineapigs sojourn ; the majority of these will become affected with general tuberculosis after the usual lapse of time, the lungs being here most advanced in the tubercular process. The writer has had guineapigs kept in their cages in the ventilating shaft at Brompton Hospital, and thereby general tuberculosis was produced in the great majority of these animals : caseous tubercles in the lungs, in the lymph glands, spleen, liver, and pelvic glands, was the result ; proving that the air of any place in which tuberculous persons sojourn contains the tubercle virus, and must therefore be considered as not free from danger.

BOVINE TUBERCULOSIS

Tuberculosis is a common disease of the bovine species : the number of tubercular animals is astonishingly great. In many instances, on slaughtering them, only the lungs are found diseased, presenting a peculiar and characteristic appearance, viz. the surface of the lung, pleura, and diaphragm presenting numerous flat oval, spherical or irregular shaped nodules, some with short broad basis, others with long thin basis or stalk fixed on to the organ. Sometimes clusters of them project from the general surface ; these appearances have caused the disease to be called 'the grapes,' in German 'Perlsucht.' Not only the surface of the lung, but also the interior contains numerous such nodular deposits. They differ considerably in size, some being not larger than a split pea, others as large as a filbert or walnut, or larger. Some of these nodules are filled with thick, creamy pus, others are yellow and caseous but firm, and others again contain calcareous matter. Under the microscope the nodules contain in the periphery, round cells in a fibrous

¹ Eisenhardt (*Centralbl. f. Bact. und Parasit.*, X., No. 18) found in 1,000 *post-mortem* examinations of human tubercular bodies, children under sixteen excluded, only one solitary instance of primary tuberculosis of the intestines.

matrix, and amongst them very numerous giant cells of different sizes, from that of only twice or thrice the size of an ordinary leucocyte to that of a real giant cell with twenty to thirty and more nuclei all regularly disposed near a peripheral zone of the cell. Near the caseous portion these huge giant cells are very conspicuous; the caseous part may still show the outline of the giant cells, but their nuclei do not take the stain, and the whole tissue of the caseous portion is a granular *débris*. In early cases of bovine tubercle the development of the tubercles in and from the lymphatic vessels of the pleura can be readily followed. This has been correctly described by Sibley. In pronounced and advanced cases, freely projecting nodules, as also nodules within the substance, having a great tendency to suppurate, are met with in the lymphatic glands, in the spleen, liver, and even in the milk glands. In these latter, the condition assumes an important practical aspect, since the udder of a cow may contain tubercular nodules without these being easily diagnosed, and may give to the milk infective properties. Although in light cases the milk gland is found free from tubercles, yet in many advanced cases purulent tubercular deposits have been demonstrated in the udder.

In the lymph glands, spleen, and liver, the character of the nodules is the same as in the lung, and giant cells form a very conspicuous feature.

Tubercular nodules of large size (about the size of a bean or filbert), caseous in the centre, are occasionally found in the spleen of young calves; this would suggest that the process is inherited, for it can hardly be assumed that in an animal six to eight weeks old tubercles already caseous had been acquired after birth: in fact, if careful search is made for these tubercles in young calves derived from tubercular cows that have no tubercles in the udder, it will be found that they are not at all of such rare occurrence as is sometimes assumed. On the other hand, calves fed on milk derived from tubercular cows, in whose udder tubercles can be demonstrated (by the tubercle bacilli test), develop tuberculosis of the intestine, the mesenteric glands, spleen, and liver; but for the development of these many weeks and months are required. While, then, calves acquire tuberculosis by feeding on milk tainted with the tubercular virus, some undoubtedly inherit tuberculosis directly from their tubercular mother.¹

Infection with general tuberculosis of guineapigs and rabbits by bovine tubercular matter, by feeding, or by subcutaneous or intraperitoneal inoculation, is easily achieved; the result is more intensive and much more rapid than by infection with human tubercular matter. Guineapigs, subcutaneously inoculated, develop disseminated tuberculosis of the lymph glands, lungs, liver, spleen, serous membranes, and marrow of bone in less than half the time; in some cases the animals die in about five to six weeks with remarkably widespread and advanced tubercular deposits. Also as regards rabbits, the process is much more rapid and more intensive; for while these animals, as mentioned above, after inoculation with human tubercular matter, develop, as a rule, only a more or less mild form of tuberculosis, limited chiefly to some lymph glands, spleen, and perhaps the liver, after inoculation with bovine tubercular matter they show very numerous tubercular deposits in the lungs, liver, spleen, all lymph glands, and even in the kidneys. The same results are obtained by feeding rabbits and guineapigs with bovine tubercular matter. Here also the process starts with tubercles of the ileum,

¹ In the Second Congress on Tuberculosis in Paris (*Semaine Médicale*, 1891), Vignal, Bernheim, Hutinel, from experiments and observations made on guineapigs and rabbits, conclude that transmission of tuberculosis from the mother to the offspring is of very rare occurrence.

then spreads to the mesenteric glands, the pelvic glands, the omentum, spleen, and liver, and finally the lungs and sternal and bronchial lymph glands. The difference in the intensity and duration of the process is decidedly more pronounced with bovine than human tubercular matter, and also in the rabbit the difference between feeding with bovine and with human tubercular matter is striking; so that there can be no question that bovine tubercular matter acts in a conspicuous degree more virulently than human tubercular matter, both in guineapigs and rabbits.

The feeding of calves with milk derived from an udder containing tubercular deposits produces tuberculosis in these calves; but milk coming from a healthy udder (though the cow has tubercles in the lung) fails to produce tubercle.

Hirschberger ('*Experim. Beiträge zur Inf. der Milch tuberculöser Thiere*, München, 1889) finds that at least five per cent. of milch cows are tubercular, and though in many cases their milk is not different from the milk of normal cows, and no tubercle bacilli can be detected, yet the same milk injected into the peritoneal cavity of guineapigs produces miliary tuberculosis in the peritoneum, spleen, and liver. Out of twenty series of experiments only once could the tubercle bacilli be demonstrated in the milk, and yet in ten such experiments in which the milk did not show tubercle bacilli, it nevertheless produced tuberculosis on intraperitoneal injection. Hirschberger explains these results by assuming that though tubercle bacilli were not present in the milk as bacilli, their spores must have been present. Bollinger ('*Münchener med. Zeitschr.*, No. 48, 1889) shows that milk from the healthy udder of tubercular cows loses its infectivity after dilution (1 : 40, 1 : 50, or 1 : 100). Therefore, he warns against the consumption of milk coming from one particular cow, but encourages the mixing of milk from many cows, since thereby the tubercular milk would become greatly diluted by normal milk, and thereby lose its infective power; a conclusion that must be admitted as quite possible from the observations just stated, though it is contrary to what one would *a priori* have expected, viz. one would have thought it better not to mix, in order to avoid possible contamination of normal milk.

TUBERCULOSIS IN FOWLS

Koch first pointed out (International Medical Congress, Berlin, 1890) that the tubercular process naturally occurring in fowls is different from human or bovine tubercle, the tubercle bacilli of the fowl behaving differently in culture and in the animal experiment from those derived from human or bovine tubercle. A. Maffucci ('*Zeitschr. für Hygiene und Infect.*, Band XI., 8, p. 445) has minutely studied and described these differences.

The important question that is constantly being asked is this: Can bovine tubercular matter produce tuberculosis in man? No direct evidence of this has as yet been given, but there are a number of ascertained facts which make it extremely probable that bovine tubercle is so communicable. On the one hand, the pathology of bovine and human tubercle makes it quite acceptable that the two are variations of one disease. Although in some minute details they differ from one another, e.g. in the bovine species, tubercle of the lung assumes the form of the so-called grapes, which it does not in man, they nevertheless must both be regarded as the disease tuberculosis (and as we shall presently see, both are caused by the same kind of bacillus), i.e. a granuloma in the lymph glands, viscera, and marrow of bone, leading to caseation and associated with the same kind of microbe; it is also true

that in the guineapig and rabbit the result of inoculation and feeding with the two viruses is not quite identical, but they have, nevertheless, this in common, that by subcutaneous inoculation and feeding, typical tubercle can be produced. These differences may be and probably are entirely due to the fact that the tissues of the bovine species are not identical with those of man, and that the activity of the virus is different according as this is bred on bovine or human soil, that is to say, that the degree of virulence of the two is not the same. As we shall see below, there is other evidence to show that bovine tubercle is of greater virulence than human tubercle.

On the other hand, the numerous cases of acute miliary tuberculosis in children, not derived from tubercular parents, become otherwise very difficult of explanation. It will not be contested that tuberculosis differs from some other infectious diseases in this: that direct communicability from person to person, though proved, is yet of rare occurrence. Villemin himself described cases where a husband communicated tuberculosis to his wife, and *vice versa*; other cases are on record where a phthisical person (a midwife) infected new-born children with tuberculosis; but on the whole, taking into account the comparatively extremely rare occurrence of tuberculosis in persons who, coming from a healthy stock, have been in communication with a tubercular patient, it is quite justifiable to assert that direct infection of the healthy from the tuberculous is a rare event.

So that on this assumption the comparatively numerous cases of miliary tuberculosis in children are difficult of explanation, and we are perhaps not far wrong in attributing such cases to the consumption of cows' milk containing the tubercular virus derived from a tubercular udder. Likewise, Dr. Sims Woodhead states that the numerous cases of *tabes mesenterica* (tuberculosis of the intestines and mesenteric glands) of children are attributable to the consumption by these children of milk derived from the tubercular udder of the cow. But it must not be forgotten that tuberculosis of the human species is in the majority of cases directly inherited, though the disease as a rule lies latent till the person attains the teens or twenties.

A good many pathologists still maintain with Virchow that heredity means only a greater predisposition, a weakness of the lung and other tissues, and that if tuberculosis actually makes its appearance in such a person, say when in the teens or twenties, it probably is due to infection with tubercle. Now, this view dates, as Baumgarten¹ very correctly points out, from a time when tuberculosis was not yet known to be an infectious disease; at the present time it has no clear meaning, because in no other infectious disease is anything so vague admitted. Heredity of disease always means, and can only mean, the transmission of the disease germ directly from the parent to the offspring. In inherited tuberculosis the disease germs lie dormant during the period of the active growth of the tissues, to start multiplying when this has become less active. The cases of latent tuberculosis are not quite so rare as has been represented, since on the *post-mortem* table one has not so very unfrequently the opportunity of finding a calcareous or caseous nodule that is really a solitary tubercle in a lymph gland, in the lung, in the liver, or in the spleen.

Direct infection by inoculation is known to occur on the hands of butchers, cooks, and others handling carcasses of tubercular cows, and also amongst those who make *post-mortems* on human tubercular corpses (*tuberculosis cutanea verrucosa*); but in all these instances inoculation is followed by local tubercle, which after suppuration leaves no further mischief behind.

A further point in connection with bovine tubercle is the question

¹ *Lehrbuch d. pathol. Mycologie*, p. 626.

whether in a tubercular cow the whole carcass should be declared dangerous for consumption, or, after the removal of the tubercular organs, should the consumption of flesh be considered as of no danger? If, in a tubercular cow, the tubercular viscera or any part thereof be considered dangerous for human consumption, then the whole animal must be considered dangerous. Tuberculosis being disseminated and involving in the cow such very different and distant organs as the lungs, the lymph glands, the abdominal viscera, and the marrow of bones (MacCall), it must be clear that the tubercular virus, though it has not its habitat in the blood, is nevertheless being disseminated by the blood to the different organs, or is carried from one part to a distant one by means of the blood-vessels. True, Klebs, Cohnheim, Sanderson, and the writer himself, have shown that in artificial tuberculosis in guineapigs infected by way of the peritoneum, the process spreads by means of the lymphatics from the peritoneum through the lymphatics of the diaphragm to the organs of the thorax; also that after subcutaneous infection the virus spreads along the lymphatic vessels and lymphatic glands, but on the other hand, tubercular deposits in the intima of the carotid artery, the aorta, and other large arteries have also been recorded. Besides, the early appearance of tubercles in the red marrow of the bones in guineapigs (Klein and Lingard), after subcutaneous inoculation, and in the bone marrow of the cow in general tuberculosis (MacCall), conclusively show that the tubercular virus becomes disseminated through the general blood circulation. By injection of tubercular virus (artificial cultivations of tubercle bacilli) into the veins of the ear in rabbits, the writer has, like Koch, produced most marked miliary tuberculosis in the lungs and liver: both organs were riddled with vast numbers of minute grey tubercles (fig. 141). After three weeks this condition was established, and in the lung the formation of the tubercles in connection with blocking of capillary blood-vessels could be clearly demonstrated. Such being the facts, we are justified in saying that the dissemination of the tubercular virus by the vascular system is proved, and under this aspect all organs may, at any time, harbour in their blood-vessels the tubercular virus, though this may be only on transit to a distant place, and though a particular organ (e.g. muscle) does not contain any discernible tubercular deposits.

Kastner ('*Experim. Beiträge zur Inf. des Fleisches perlsüchtiger Rinder*,' München, 1889), it is true, has failed to produce tuberculosis in guineapigs into whose peritoneal cavity the juice of the muscular tissue of bovine animals affected with tuberculosis was injected, but such negative results do not justify us in denying the possibility of infectiveness of the flesh at one time or another. Besides, more recently Steinheil ('*Münchener med. Woch.*' 1889, Nos. 40 and 41) has shown that injection of muscle, in which no tubercle could be detected, but taken from persons dead of phthisis, into guineapigs produced well-marked tubercles in fifteen out of eighteen animals so inoculated.¹

Cohnheim and Salomonson² were the first to show that in all tubercular material a specific virus is present. On injecting a small particle of such matter into the anterior chamber of the eye, they noticed that after the first result due to the injury has passed off, the introduced particle gradually undergoes diminution to almost complete disappearance, but in about a fortnight or three weeks a crop of minute grey nodules which in reality are typical young tubercles are found in the iris; these gradually enlarge,

¹ See also the discussion on this subject by Bang, Arloing, Nocard and others at the International Congress on Hygiene, London, 1891.

² *Uebertragbarkeit d. Tuberculose*, Berlin, 1877.

and like all ordinary tubercles undergo caseation. The tubercular process is at first localised to the iris, but it gradually spreads to the cervical lymph glands, and ultimately leads to general tuberculosis of the other lymph glands and viscera exactly as after subcutaneous inoculation. This typical production of a crop of grey tubercles on the iris by tubercular matter enabled Cohnheim and Salomonson to differentiate tubercular from non-tubercular matter, and they have formulated from this the axiom that only matter derived from tubercle is capable of producing tubercle, and that whenever any substance is found capable of producing this iris tuberculosis, it is derived from tubercle. By this clear proof for the first time a means was offered of making an easy differential diagnosis between tubercular and non-tubercular matter, a diagnosis which those who, at former times, were engaged in work on tubercle found extremely difficult. As is well known from clinical observation, the diagnosis of tuberculosis of the lungs sometimes associated with difficulties: the physical examination and symptomatology do not always insure a correct diagnosis. It is true that Villemin had proved by experiment that tuberculosis is inoculable, and Wilson Fox had insisted on the specificity of tuberculosis by numerous experiments which he himself had carried out, yet there were authorities who did not draw this sharp distinction, but were rather inclined to the view that artificial tuberculosis is due to matter derived from a variety of sources not necessarily always tubercular, having undergone some process of intensification, and become infective. For that period, therefore, the exact proof given by Cohnheim and Salomonson marked a very important step, though the exact nature of this specific tubercular virus remained undetermined. The next discovery was that of Koch,¹ who showed what this nature is; he demonstrated a particular species of bacilli, now familiar to all pathologists as the *tubercle bacillus*, which he found only in tubercle and in no other disease, a bacillus so peculiar and so constant that its important diagnostic value was at once recognised. No matter whether it is a nodule in any tissue or organ that does or does not present the typical pathological (gross and minute) characters of the classical tubercle; no matter whether in man or the bovine species, in the sheep, in the monkey, dog, cat, rat, mouse, rabbit, guineapig, fowl, or ostrich, if in such a nodule the bacilli characteristic of tubercle can be demonstrated, *that nodule is tubercle, and the disease is tuberculosis*. The discovery by Koch of this fundamental fact marks one of the most brilliant and most practical discoveries of modern medical science; the diagnosis of tubercle, once so difficult to make with certainty, is now, by means of the demonstration of the presence of the tubercle bacilli, one of the easiest and at the same time one of the most important helps in the formation of a correct diagnosis in some otherwise doubtful cases. Koch further proved that not only are these particular bacilli present in all and every tubercle of man and brutes, but he also showed that these bacilli can be artificially cultivated outside the animal body, and with such cultures typical and general tuberculosis can be produced by inoculation, the tubercles thus produced again containing the same tubercle bacilli; in short, *he conclusively established that these bacilli are the vera causa of the disease tuberculosis*. The whole problem concerning one of the most widespread, fatal, and at that time little understood diseases of man and animals was by these researches at once cleared up, and considering the difficulties in the solution of the problem and the necessity of having had to invent special methods by which this research was carried to a successful issue, we have no hesitation in saying that this discovery of Koch marks one of the most important, if not the most important,

¹ *Berliner klin. Wochenschr.*, 1882.

epoch in pathology. Many are the workers who since Koch have contributed towards further details as to this matter; but without any intention of minimising the importance of any and every contribution of fact towards a clear understanding of this disease and its prevention, we are, we think, within the limits of absolute correctness in saying that Koch's publications on tuberculosis ('Berliner klin. Woch.', 1882; and 'Mitth. aus d. k. Gesundheits-amte,' II.) contain almost the complete solution of the problem. Koch showed that by staining tubercular materials (in cover-glass specimens or in sections) with aniline dyes (methyl blue) to which a little alkali is added, and then subjecting them to decolourising agencies (nitric acid, hydrochloric acid), all the colour is lost from the elements of the tissue with the exception of the tubercle bacilli themselves. Ehrlich then showed that by staining tubercular material in fuchsin aniline oil and then treating the specimen with one-third nitric acid (ordinary nitric acid one part, water two parts), all the colour is removed except from the tubercle bacilli, these retaining the red colour; by after-staining with vesuvin or methyl blue the tissue is again stained brown or blue respectively, while the tubercle bacilli retain the beautiful bright red colour. The leprosy bacilli, the syphilis bacilli, and the smegma bacilli have the same power of withstanding the action of nitric acid after fuchsin staining, but other bacteria become decolourised. The best method, and one always used with success, is to stain with Ziehl's carbol fuchsin from twenty to thirty minutes at 85° to 40° C., then washing in water for a second or two, then for a few seconds (five to ten) in 83½ per cent. nitric acid, washing again in water and placing in methyl blue aniline oil for five to ten minutes, washing and treating in the usual way, according to whether a cover-glass specimen or section is being dealt with. The writer has never seen failure with this method: the tubercle bacilli are always brought out with striking clearness.

A great many modifications for staining the tubercle bacilli have been published, all good to a lesser or greater degree, but the one just mentioned is as good as any and in many instances has proved simpler and better. The tubercle bacilli always occur in tubercular nodules, sometimes more-numerously where caseation has already set in than in the earlier stages. They occur isolated or in groups between the cells constituting the tubercle, or they are found singly or in small groups within the larger cells; when present in giant cells they are found in large numbers, forming a sort of annular zone around the central portion. In some giant cells their number is at times very limited, and Koch has concluded from this fact that the tubercle bacilli suffer death in the giant cells, and hence disappear from them. In human tubercle, the tubercle bacilli are, as a rule, between the elements constituting the tubercle; but, as just mentioned, they also occur within the uni- and multi-nucleated cells. In bovine tubercle, however, the rule is that they are mostly present in the uni- and multi-nuclear cells, and only when these degenerate and break up do they become free; in the caseous matter they are present in groups in the granular *débris*. In tubercle of rabbit (lung and liver), produced by inoculation with bovine tubercular matter, or with artificial culture derived from bovine tubercle, the presence of tubercle bacilli within the cells—small, large, and giant cells—is very conspicuous, and yields very remarkable specimens (*see* fig. 110).

The tubercle bacilli in human tubercle are delicate cylindrical rods: measuring 1·5–4 μ ; many are straight, with rounded ends, but others are slightly curved; in preparations (sputum, purulent matter, or sections) stained in the above manner the bacilli always appear composed of granules, that is to say, within a faintly stained sheath the protoplasm is segregated into-

deeply stained, cubical, spherical, or rod-shaped granules; between the granules the sheath is empty, but these empty places are not to be taken for bright spores, as is done by some observers, nor is it proved that the above granules are spores. That the tubercle bacilli contain spores is proved by numerous experiments of drying and heating, to be detailed below; but what the character of these spores is, and how they appear in the bacilli, has not been satisfactorily shown. In bovine tubercular matter prepared in the same manner the tubercle bacilli are, as a rule, shorter and thinner, and though we do not for a moment question the fact that some tubercle bacilli of human tubercle are as short and thin as those of bovine tubercle, we are confident from numerous observations that the majority of the human tubercle bacilli are longer and thicker than those of bovine tubercle; besides, in preparations stained in the above manner, the segregation of the protoplasm within the sheath, though also present in many tubercle bacilli of bovine tubercle, is not so general and uniform as in those of human tubercle. But these minute differences need mean nothing more than differences due to the different soils on which the bacilli were reared. Such morphological differences in size and aspect in one and the same species of microbes are well known to occur in other instances if the microbe be cultivated in different soils. When the tubercle bacilli from any source (bovine, human, or from artificially infected animals) are passed through the rabbit or the guineapig, in these animals the new crop of bacilli all appear to be morphologically the same.

The tubercle bacilli show definite characters in cultivation. Koch succeeded in cultivating them on solid blood serum. On inoculating the slanting surface of the solid serum with tubercular matter, and provided no other bacteria are introduced, Koch noticed the first signs of growth in ten to fourteen days. Koch used for inoculations of serum tubes the tubercular deposits of a swollen lymphatic gland of a guineapig, three to four weeks previously inoculated with tubercular matter, clean sterile instruments being used. After ten to fourteen days the first signs of the growth of the tubercle bacilli showed themselves in the form of whitish points and patches, resembling dry scales. On further growth they enlarged, and where close together they coalesced into dry whitish scaly masses with irregular outline. From such primary cultures, sub-cultures on serum were then carried out. But under a magnifying lens, or better under the microscope, the growth and multiplication of the tubercle bacilli can be seen before the end of the first week. Peculiar curved, or convoluted, or S-shaped whitish lines, which prove to be strands of tubercle bacilli, are noticed even at this early stage. On agar broth the growth is very limited, and also in broth. But Roux and Nocard showed that by adding 8 per cent. of glycerine to agar meat infusion, or to meat broth, the tubercle bacilli can be brought into rapid and extensive multiplication. On glycerine-agar-beef infusion the tubercle bacilli grow very rapidly, the growth being visible after six to eight days, and after several weeks to several months covers the whole surface as a whitish, peculiarly wrinkled, dry film, extending as a white thin pellicle over the condensation water at the bottom of the tube. In order to obtain good and copious growth it is necessary to keep the tubes capped from the outset. The writer has obtained very copious growths in alkaline broth, to which a piece of boiled white of egg was added. In such tubes the broth kept at 87° C. remains clear for four or five days, then minute flocculi and granules appear at the bottom and along the wall of the tube, where it is in contact with the broth; after a fortnight the growth becomes abundant, and on shaking the broth is made turbid by the numerous flocculi. On potato moistened with broth it is likewise possible to obtain growth. Temperatures

of 36° to 38° C. are most favourable for the growth ; below 30° or above 42° C. no growth can be noticed.

Koch has shown that by subcutaneous or intravascular injection, by inhalation, and by inoculation into the peritoneum or the anterior chamber of the eye, &c., of artificial sub-cultures removed by many generations from the original source, typical tuberculosis is produced in all animals susceptible to tubercle (guineapigs, rabbits, dogs, rats, mice), and that the tubercular deposits in these experimental animals again contain abundantly the tubercle bacilli ; thus, the final and exact proof that the tubercle bacilli are the *vera causa* of the tubercular process was definitely established. The intravascular and intraperitoneal injection produced the most striking and rapid results.

Although the growth on glycerine agar mixture is copious, it yet has this drawback, that by continued sub-culture the virulence of the bacilli is diminished. The first sub-cultures act virulently, inasmuch as they produce general and fatal tuberculosis on inoculation into guineapigs ; thus even with a fourth and fifth sub-culture the writer has succeeded in producing the same results as by directly using sputum or bovine tubercle, but after the eighth or tenth generation it has not been possible to produce general tuberculosis and death of the guineapigs by inoculation. The writer has found that if from an agar-glycerine culture, which, owing to age or sub-cultures, has completely lost its virulence, new cultures are established in alkaline beef broth, to which a piece of boiled white of egg is added, these sometimes again acquire a somewhat virulent character.

By injection of large quantities of cultures (a full Pravaz syringe of a milky salt mixture) directly into the ear vein of the rabbit the writer has produced in two weeks extensive tuberculosis in the lung, both lungs being riddled with minute tubercles, the small ones (varying in size from about the point to the head of a pin) being grey, the larger ones yellow and caseous in the centre (*see also* Koch). In guineapigs inoculated with considerable quantities of late sub-cultures on glycerine agar, firm-swelling and purulent change in the nearest lymph gland can be produced, leading to abscess and ulceration ; in such glands the presence of crowds of tubercle bacilli can be easily demonstrated in cover-glass specimens ; but the glands gradually diminish in size, the ulcer heals, and after eight to ten weeks little can be noticed ; the animals do not grow thin and do not die.

Eight such guineapigs were after six months re-inoculated with virulent tubercle (tubercle of bovine lung) ; they all developed in the normal time general tuberculosis, and died. On the *post-mortem* being made no difference could be detected between them and control animals. But it is justifiable to suppose that with the proper degree of virulence, i.e. the proper stage of attenuation, the cultures might produce a more severe effect than the above cultures, an effect which, though not leading to the development of general tuberculosis, might yet protect the animals against a second inoculation of virulent material.

As a rule, cultures made in alkaline beef broth to which a piece of boiled egg albumen has been added behave in the same manner as glycerine agar sub-cultures ; that is to say, in the former the virulence of the tubercle bacilli becomes gradually attenuated by sub-cultures, so much so that injecting a considerable quantity of a late broth sub-culture into rabbits (the ear vein), or, better still, into the muscle of the abdominal wall, only a local abscess is slowly developed ; the pus and particularly the wall of the abscess contain the tubercle bacilli. After the abscess is opened, spontaneously or by incision, the wound heals and the animal completely recovers. With Dr. Schorstein the writer has made experiments with the pus of such a local

abscess, and it was found that on subcutaneous inoculation into guineapigs a much more marked result is produced; in fact, general tuberculosis ensues, thus proving that the tubercle bacilli, attenuated in the broth culture, when passed through the guineapig regain their virulence. A condition similar in principle to this will be mentioned of the tubercular virus of human scrofula (*see below*), this being an attenuated form of the tubercular virus, but gaining in virulence as it is passed through the guineapig.

The sub-cultures on glycerine agar show after several months besides the typical forms of cylindrical and granular tubercle bacilli also some filaments made up of rods and granules. Some of these filaments are remarkable by their being undoubtedly branched like the mycelium of a hyphomycetes, and, further, by some being club-shaped at the end or beaded in their course; these club-shaped and branched filaments are the more numerous the older the culture. Although the club-shaped and beaded condition might correspond to involution of the threads, the branched condition cannot, and therefore the club-shaped forms may well represent the growing ends of the threads of a mycelium; the two together, i.e. club-shaped and branched threads, would, therefore, indicate that the typical tubercle bacillus is a phase only in the development of an organism, which under certain conditions (glycerine agar) declares its true nature and origin, being, namely, comparable to a fungus having a mycelial stage (*see Klein in the Report of the Medical Officer of the Local Government Board, 1889, Plate XXVII., figs. 61, 62, 68*).

SCROFULA AND LUPUS

Koch and many other observers have shown that, both in scrofula and lupus, tubercle bacilli occur, and that with both these materials general tuberculosis can be induced in guineapigs. But since these two diseases are in the human subject well-marked disorders, distinct from pulmonary tuberculosis, it is necessary to assume that the tubercle bacilli in the three diseases possess some functional differences. To say that lupus is a form of tuberculosis of the skin does not cover the facts, since real tuberculosis of the skin does occur, and is totally different from lupus; so also scrofula is not merely tuberculosis localised in the cervical lymph glands, since, in many instances, it does not lead to pulmonary and general tuberculosis, whereas the true tuberculosis of lymph glands does so. It is quite feasible to assume that both lupus and scrofula are tuberculosis, but that in origin and virulence their tubercle bacilli are different from the bacilli causing true tuberculosis. That the virulence of the virus of lupus and scrofula cannot be the same as that of the material of human and bovine pulmonary tubercle is proved by experiments of Dr. A. Lingard,¹ who showed that the duration and extent of the disease induced by inoculation of lupus or scrofula into guineapigs is quite different from that induced by pulmonary tubercular matter, and, further, that if a guineapig is made tubercular with scrofulous matter, and the tubercle of such an animal is again transmitted by inoculation through several generations of fresh guineapigs, the disease thus produced gains gradually in shortness of duration and intensity, until after several generations the same effect of general tuberculosis is produced as that produced directly by matter of pulmonary human tuberculosis.

That the tubercle bacilli in one phase or another do contain spores has been shown by Koch, who found that tubercular sputum when thoroughly dried maintains its virulent character. The writer has repeated these experiments, and can say that tubercular matter (from human tubercles of the

¹ Report of the Medical Officer of the Local Government Board, 1888, p. 462.

lung and lymph glands, from bovine tubercle of the lung, tubercular matter from lymph glands, lung, liver, and spleen of guineapigs dead of general tuberculosis artificially produced) bears thorough drying without losing its virulence. The writer has also repeated these experiments with regard to artificial cultivations: a small particle of culture is mixed well in sterile water; of this mixture a thin film is spread out on a glass slide and this is thoroughly dried, then the film is again scraped off, mixed with a drop of water, and with this mixture guineapigs are subcutaneously inoculated: these become affected with general tubercle in the usual way.

Now, if the tubercle bacilli had no spores, they would not in all cases survive thorough drying; no sporeless bacillus is known that can survive thorough drying; whereas all bacilli in the stage of spore-bearing survive this process. Further, tubercular matter and cultures of tubercle bacilli survive temperatures up to 100° C. Non-spore-bearing bacilli and micrococci are killed by being exposed for five minutes to a temperature of 65–70° C., whereas spores of other bacilli withstand much higher temperatures. Tubercular sputum distributed in salt solution does not lose in the least its virulence by being kept at 100° C. for one to two minutes. Nor does a solution of perchloride of mercury kill the tubercle bacilli in the way it does sporeless bacilli. Dr. Lingard found (Report of Medical Officer of Local Government Board for 1885, p. 188) that solution of perchloride of mercury, one grain of mercuric bichloride to 960 grains of water, that is to say, about one in 1,000, although it kills the bacilli in human tubercular matter when acting on it for four hours, does not do so in the case of bovine tubercular matter, since not even eight hours' exposure to the solution is sufficient to destroy the infective power of that material. This also shows, as has been mentioned already on a former page, that bovine tubercular matter is of a higher degree of virulence than human tubercular matter.

The important observations of Schottelius ('*Centralbl. f. Bact. und Parasit.*, VII., 9), that tubercle bacilli taken from the lung of phthisical persons, buried for years, still possessed on inoculation into rabbits and guineapigs the capability of producing artificial tuberculosis, have been already mentioned.

Schottelius further shows that the tubercular lung kept in soil (enclosed in a wooden box) shows an incomparably large increase in temperature, amounting in some experiments to a rise to 84° C. This would indicate that under these conditions the tubercle bacilli in such a lung are capable of continuing to live and multiply. From these experiments the conclusion must be drawn that the tubercle bacilli are not true parasites, but belong to the ectogenic microbes, which can live and thrive independent of a living host.

In August 1890, on the occasion of the International Medical Congress held in Berlin, and in subsequent publications, Koch announced that by experiment he had ascertained that when guineapigs, previously made tubercular by subcutaneous inoculation, are inoculated, at a date when the animals show the signs of tuberculosis, with extract (glycerine extract) of dead tubercle cultures, the growth itself from the surface of serum or glycerine agar being rubbed down and extracted with glycerine, the tubercular glands of these animals undergo a rapid necrosis and elimination, brought about by an acute reactive inflammation beginning in the tissues around the tubercle, but the tubercle bacilli themselves are not affected by it. He then applied this method of injecting glycerine extract of dead tubercle cultures—tuberculin—in very small doses, 0·001–0·01 gramme, into the human subject: lupus, bone tuberculosis, and early pulmonary tuberculosis. The result was remarkable, since most patients affected with one or another form of tuberculosis reacted very conspicuously to such injection: high

temperature, great local congestion and inflammation in lupus, and bone tubercle. Persons not affected with tubercle do not, as a rule, show any reaction to such small doses. In lupus, and bone and joint tubercle, the tubercular tissue becomes necrotic, is either spontaneously eliminated, as in lupus, by the reactive inflammation of the surrounding tissue, or can be removed by surgical aid, as in tuberculosis of bone. Tuberculin is, then, a distinct means of diagnosing tubercle in the living subject, otherwise not easily diagnosed. Also with regard to bovine tubercle, the injection of tuberculin has proved of great value, since early tuberculosis, not otherwise apparent, can be diagnosed by the definite reaction (rise of temperature) that occurs in such animals, but not in animals free from tubercle. These observations have been recorded by a number of accurate experimenters on the Continent, in England, and in America.

Weyl has analysed the tuberculin, and found that the essential portion of it is a substance related to mucin, not to albumin. Dr. W. Hunter ('Brit. Med. Jour.', 1891) has more accurately determined the various principles present in tuberculin.

Good therapeutic results have been obtained with the tuberculin in lupus, bone tuberculosis, and in early pulmonary tuberculosis; in advanced pulmonary tuberculosis the injection of tuberculin has in some cases produced a dissemination of the tubercle bacilli and acute miliary tuberculosis in the lung and other viscera (Virchow). The same or similar results have been obtained in tuberculised guineapigs by Baumgarten after injection of tuberculin.

CHAPTER XXVII

LEPROSY—ACTINOMYCOSIS—TRACHOMA

LEPROSY

LEPROSY belongs to the group of granulomata, inasmuch as in the majority of cases—the tubercular form—the connective tissue of the skin, and to a smaller extent of the mucous membrane of the mouth, pharynx, larynx, cornea, testes, parenchymatous organs, and marrow of bone, are the seat of nodular deposits made up of cells. These cells are spherical, oblong, spindle-shaped, or irregular; in the central part of the nodules or tubercles they are more closely placed side by side; in the peripheral, i.e. towards the surrounding portions, they are less crowded; few of the cells are small like leucocytes; the majority are twice or thrice or more times this size. But whether small or large they generally possess a single nucleus; the large cells have received from Virchow the name of *leprosy cells*. Where the cells are closely aggregated there is very little other tissue between them. In the peripheral parts, however, there are connective tissue bundles between them. The tubercle or nodule enlarges by masses of cells becoming deposited in the surrounding tissue—fibrous tissue, muscular tissue, cartilage, &c. The nodules may also enlarge towards the surface, i.e. involve the epidermis or epithelium; and this latter then ultimately sooner or later becomes destroyed, and a discharging, ulcerating sore is established; but this is not the necessary fate of the tubercles, since some, having reached their maximum growth, dwindle down and become absorbed, leaving an œdematous, puffy, discoloured patch of the skin behind. In all the nodules, however, numerous cells are constantly disintegrating, the nucleus swells up and disappears, and then the cell substance disintegrates. In the second form, the

anæsthetic form, there are no nodules formed, but the nerves undergo a fibrous degeneration; in the early stages the connective tissues of the nerve—the general epineurium and the perineurium—contain in their lymph channels and lymph spaces cells which also gradually invade the connective tissue within the nerve bundles, i.e. the endoneurium; gradually the connective tissue thickens, and in the nerve bundles the nerve fibres undergo degeneration leading to the disappearance of the axis cylinders and medullary sheath and the transformation of the whole nerve fibre into a connective-tissue bundle.

That leprosy is a communicable disease is proved in several instances: Europeans living with lepers (Father Damien), sexual connection of Europeans with lepers (in India and other Eastern countries); but by what ways the leprosy virus enters the body is unknown. Likewise it is not known why the disease is, on the whole, so rarely communicated to healthy persons repeatedly in contact with lepers, for the fact that the disease is one of the least easily communicated is a conspicuous feature in all countries where leprosy prevails. Occasional cases of leprosy occur in England and in America nowadays, some of which have contracted the malady in the East: they present sometimes a very severe form, but no communication to other persons has been known to occur. The writer has seen a very severe case of leprosy in a young man, who contracted the disease in India: the patient's face and ears were really one large sore discharging purulent matter very copiously; every trace of this discharge was crowded with round cells and large leprosy cells, all of them crowded with the leprosy virus, i.e. the *lepra bacilli*, numerous cells in a state of disintegration, and hereby the bacilli becoming free and scattered in the fluid part. Fig. 147 is a representation of this discharge, and yet, though the patient voided many thousands of the bacilli in every drop of the discharge, no case of leprosy was known to have been communicated by it. But though the disease seems to be communicated with difficulty, it behoves us in the face of the copious crop of *lepra bacilli* yielded by the leprosy ulcers to be on our guard and to keep lepers as much isolated as possible; and this wise precaution is, as is well known, practised in Norway, as also in many places in the East and West, where leprosy prevails.

A microscopic section through a leprosy tubercle (figs. 145 and 146) shows the tubercle to be a granuloma, i.e. an aggregation of cells, each and all of them literally filled with minute bacilli. A. Hansen ('Virchow's Archiv,' Band LXXIX. and XC.) was the first to have observed this fact, and he justly regarded these bacilli as the virus, i.e., as the *bacillus lepræ*. Neisser ('Breslauer ärztl. Zeitschrift,' XX. and XXI., 1877; 'Virchow's Archiv,' LXXXIV.) then showed that these bacilli have peculiar characters of staining, and he also claims to have succeeded in artificially cultivating them. Cover-glass specimens are made from a scraping or part of a leprosy nodule, or the discharge of a leprosy ulcer, by spreading it out in a thin film on a cover-glass, drying and heating, then staining after Ehrlich's method of staining for tubercle bacilli (in carbol fuchsin for twenty to thirty minutes at 85° C., then washing in water, then for a few seconds in 88½ per cent. nitric acid, washing in water, drying, and mounting); such specimens show the leprosy cells, some small, some very large, all crowded with minute stiff, thin, and relatively long bacilli. Many cells are in a state of disintegration, or broken down into granular *débris*, and in accordance with this, numerous bacilli are found free, isolated or in groups. The large and middle-sized cells are particularly interesting, since their substance is almost entirely occupied with the bacilli arranged in bundles, which bundles often lie towards one another at sharp angles, and hereby produce a very striking effect. Sections through a

leprous nodule stained in the above manner (carbol fuchsin, 39½ per cent. nitric acid, washed in water, then counter-stained in methyl-blue aniline water for fifteen to thirty minutes) shows the nuclei of the tissue blue, the cells forming the leprous nodule red, owing to the fact that their substance is crowded with the (red) leprosy bacilli; in such sections nothing can be seen of the nuclei or substance of the leprosy cells, the cells being marked merely as groups of densely aggregated leprosy bacilli (fig. 146). While, then, the lepra bacilli have characters in staining by which they resemble the tubercle bacilli, they differ according to Baumgarten and others in this, that they stain in alkaline methylene blue with conspicuously greater difficulty than the tubercle bacilli. The lepra bacilli are on the average 4–8 μ long, and about 0·8 μ thick; in well-stained and well-washed specimens they, like the tubercle bacilli, show segregation of the protoplasm (deeply stained granules) in a faintly stained sheath; the bacilli appear straight or slightly bent and more or less possessed of pointed ends. The remarkable constancy with which the lepra bacilli occur in dense crowds in the leprosy cells, and their peculiar characters, are striking. Neisser maintains that he has succeeded in cultivating the lepra bacilli, but the evidence adduced is not deemed sufficient. Bordoni-Uffreduzzi ('Zeitschrift f. Hygiene,' III., p. 178) maintains, however, that he has produced artificial cultures from the leprosy nodules of bone marrow, on glycerine serum to which peptone and salt had been added, kept at 85–87° C. The line of inoculation became marked as a yellowish irregularly outlined band; the serum was not liquefied. On glycerine agar, inoculated with considerable quantity of leprous material, the same kind of growth took place. In glycerine agar plates the colonies that grew on the surface and in the depth, seen under a magnifying power of 100–200, were rounded reticulated patches, with dark thick centre.

Inoculation experiments on animals, though repeatedly tried by a large number of observers—Damsch, Vossius, Melcher, Ortmann, Wesener, Thin, and others—by the introduction of large bits of leprous nodules into the subcutaneous tissues, the peritoneum, and the anterior chamber of the eye in various animals, have yielded only negative results, so that the conclusion is justified that leprosy is not transmissible to the lower animals.

Dr. Arning inoculated in November 1885 a Hawaiian man named Keanu with leprosy, and the disease (typical tubercular leprosy) subsequently developed. But Dr. Sidney Bourne Swift, resident physician, leper settlement, Molokai, Hawaiian Isles, states, in an article to the 'Occidental Medical Times,' April 1890, that this man Keanu is one of a family of lepers (Keanu's son, sister's son, and wife's brother are, or were, lepers), and that the disease as it appeared in Keanu was too rapid to have been the result of the inoculation by Dr. Arning ('Brit. Med. Journal,' I., 1890, p. 917).

ACTINOMYCOSIS

Bollinger¹ was the first who showed that various tumours in cattle, leading to chronic suppuration, e.g. in the jaw, tongue, pharynx and skin, are one and the same disease, due to a parasite which he constantly found present in those tumours in the shape of yellow granules. These granules, when examined under the microscope, appear to be made up of radially arranged fibres and clubs, which Professor Harz designated *actinomyces*, or *ray fungus*; and the disease it causes is therefore called *actinomycosis*. Israel² next observed a

¹ *Centralbl. f. d. med. Wiss.*, 1877, No. 27.

² *Virchow's Archiv*, Band LXXIV., 1878.

disease in the human lung, in which he found a mycelial fungus that was afterwards identified by Ponfick as the ray fungus seen by Bollinger in the tumours of cattle. Ponfick himself published several cases of actinomycosis in man.¹ Since these observations, the reports of a large number of cases, in cattle, pigs, and man, have been published, in which tumours, abscesses, and suppurations, &c., were found in one or the other of the following organs: the jaw, skin, tongue, pharynx, larynx, lung, intestine, liver, and brain, and proved to be due to the same parasite—actinomyces.

Pathology.—As regards the pathology of the nodules produced by the ray fungus, these belong to the granulomata, i.e. they are in the first place aggregations of round cells in the connective tissue; around the fungus which forms the centre of the new growth the cells constituting the infiltration are ordinary lymphoid cells, some are several times larger than the rest, and possess several nuclei; these latter, viz. the multi-nucleated cells, are generally found in the immediate neighbourhood of the fungus; many of the round cells contain coarse granules, which gradually change into fat globules. As the central fungus mass enlarges by active growth, so the nodule enlarges by the infiltration with round cells spreading into the surrounding tissues. In a later stage the central portion softens and becomes purulent; an abscess is thus formed which, opening on to the surface, or into the nearest cavity, soon discharges copious pus; when the abscess opens on to the free surface, e.g. jaw, skin, pharynx, larynx, lung, or intestine, an ulcer is established, which enlarges as the infiltration in the periphery proceeds. In the discharge of the abscess a number of yellowish minute granules can be found; these granules looked at under the microscope are a mass of the ray fungus.

It has now been established that carious teeth in some cases represent the point of entrance for the fungus; in these cases the alveolar process of the jaw becomes the place for the growth of the fungus, leading to the formation of a hard tumour, gradually becoming converted into an abscess and ulcer; the infection, i.e. invasion of the fungus, then spreads to the lymph glands and skin nearest to the affected jaw, and there produces a tumour, then suppuration and an ulcer. Or it invades the tonsils and the pharynx, either primarily or after it has once taken root in the jaw, tongue, or cheek. Or it appears primarily in the larynx, trachea, and lung; and in these cases the fungus has evidently been introduced by the air during inspiration; in the case of the lung, extensive interstitial inflammation is set up, leading to abscesses perforating into a bronchus. Or it primarily invades the alimentary canal and leads to abscess and copious suppuration there, and even to perforation of the part; in the case of the alimentary canal evidently the fungus has entered with the food. From the alimentary canal the disease spreads to the mesenteric glands and the liver; and in this latter organ it produces abscess, which may open through the peritoneum into the peritoneal cavity, or, if previously an adhesion with the abdominal wall has been established, may perforate outwards. In all these instances the discharged pus contains the yellow granules, i.e. groups of the ray fungus. In the case of the skin the fate of the tumours is suppuration and formation of abscess, and this opening on the surface leads to the formation of a sore. The primary infection of the skin by actinomyces has been proved (E. Müller, 'Mitth. aus d. chirurg. Klinik,' Tübingen, Band III., 8) in a case in which a wood splinter in the skin had evidently been the means of providing an entrance for the fungus. Both in man and cattle these various ways of infection with actinomyces have been observed in many cases.

¹ *Bresl. ärztl. Zeitschrift*, I., p. 117. *Berl. klin. Woch.*, 1879. 'Die Actinomykose.' Berlin, 1881.

Now the important question that presents itself is this, Where does the fungus generally live? The various ways above mentioned in which it invades the organism, at once suggest that it has its usual habitat in the outside world, i.e. that it is an organism which is introduced into the animal body from without, and is not directly derived from an infected animal or man. It is a prevalent opinion that the natural habitat of the ray fungus is on cerealia, that it lives on these parasitically, and through and from these enters the animal body through wounds, abrasions, &c. Johne ('*Centralbl. f. d. med. Wiss.*,' 1881, No. 15) has shown that actinomyces occurs normally in the pits and the loculi in the tonsils of the pig; in these instances there were always present bits of ears of barley covered with what appeared to be ray fungus. Jensen ('*Deutsche Zeitschrift f. Thiermed.*') observed an epidemic of actinomycosis in cattle fed on barley; and Piana described actinomyces nodules in the tongue of cattle, where in the midst of some of the nodules there were present portions of vascular fibre-tissue of corn surrounded by ray fungus. Finally, Soltmann ('*Breslauer ärztl. Zeitschrift*,' 1885, No. 8) made the remarkable observation of an actinomyces abscess in the region of the dorsal vertebral column, caused by the perforation (during swallowing) of an ear of barley; the abscess opened and the ear was discharged. Fischer ('*Centralbl. für Chirurgie*,' No. 22, 1890) describes a similar case: a labourer on chewing barley pierced into his tongue a portion of the awn. Eight days after, a swelling appeared on the punctured spot, and a fortnight later a tumour of the size of a filbert could be distinctly felt. After eighteen days an incision was made into the tumour, and the examination of the scanty pus and the tissue of the tumour revealed the presence of numerous yellow granules—actinomyces. A fragment of the awn was also removed from the interior of the tumour, and on examining it under the microscope was found covered with clumps of actinomyces. So that from all this the conclusion appears justified that actinomyces is a fungus having its habitat on certain cerealia, and with and by them is introduced into cattle and man.

As mentioned above, the tumours and abscesses occurring in one or the other organ contain peculiar minute granules and clumps, visible to the unaided eye, generally of a yellowish, but occasionally of a greenish-yellow tint. Under the microscope they appear made up of a central mass of fine granules, or of a distinct trellis-work of fine branched threads; next is a zone of coarser granules, which look not unlike cocci; but when this or the central zone is teased out, it can be shown that the granules are not really granules, but in reality are densely aggregated and twisted branched fine fibres, the 'granules' being only due to optical sections of the fibres; at the periphery of the mass are glistening, densely and radially aggregated, flask-shaped or club-shaped bodies called the 'clubs' (fig. 117). The central mass is occasionally found in a state of calcification: this is not seldom the case in cattle.

That these clubs are an important and characteristic feature in the morphology of the fungus is shown by the name of ray fungus and by the fact that, as is commonly observed—at any rate in cattle it is common—all the actinomycosis nodules and abscesses contain one or more central masses of these radiating aggregations of clubs. But also in the human disease the clubs are with few exceptions present, though there are cases described in which the fungus was said to have been represented only by a dense felt-work of branched fine threads. The first question that offers itself for answer is, What is the significance of these different morphological features, and what is the life history of the fungus?

In sections through the affected tongue of cattle we find numerous round-

cell granulomata between the muscular bundles and in the mucosa, some quite superficial and penetrating from the papillæ into the surface epithelium ; in all of these there are one or more groups of the ray fungus ; in suitably stained sections (rubin, 2 per cent. watery solution for several hours, then washed in water and stained in methyl-blue aniline water for fifteen to thirty minutes) the ray fungus group appears as an irregularly spherical mass or more commonly, particularly when large, as a lobed mass, composed of a central faintly stained homogeneous, or only faintly granular mass ; around this is a zone deeply stained in blue, and owing to its being composed of densely aggregated and twisted branched threads, it looks not unlike cocci. The peripheral part is made up of conical, or cylindrical, or club-shaped corpuscles of different lengths and thicknesses, deeply stained pink, closely placed side by side, and all radiating by longer or shorter thin, pink, filamentous stalks from the next, the blue or 'granular' zone ; each of the 'clubs' possesses a faintly stained homogeneous sheath. In human actinomycosis, and also in actinomycosis of cattle, the central mass is sometimes recognisable as a dense felt-work of fine branched threads ; from the periphery of the mass longer threads project, each or only some of them possessing a terminal enlargement ; in other cases the terminal club-shaped enlargement is the only distinct portion.

Now, some observers consider the clubs as indicating an involution or a degeneration-phase of the threads, and, further, the above granules of the second zone as indicating a coccus phase of the threads, and for this reason consider the ray fungus as belonging to the species of cladothrix, a polymorphous fungus, in which the threads may break up into, or develop from cocci and shorter or longer rods or bacilli. Now, we are opposed to this view, for we constantly find in actinomycosis of cattle some of the smaller, i.e. younger tumours, contain fine clubs in isolated examples or in small groups, without any filamentous or granular centre ; in the preparations stained successfully as above we find appearances which place us in full agreement with Crookshank ('Transactions of the Med. Chir. Soc.', 1889) : single clubs very conspicuous by their deep red staining attached to a short single or branched stalk, free or enclosed within a nucleated cell. Further, we can find, free or enclosed within a larger mass of protoplasm, a small homogeneous mass from which are budding out two, three, or four clubs of different lengths and with very short stalks ; these structures being stained bright pink stand out very conspicuously from the blue ground.

Further, we find spherical or oval globules recognisable by their deep pink staining becoming constricted off from the free end of the clubs. Putting these features together, there can be no difficulty in recognising a striking likeness between the ray fungus and a mycelial fungus : the fine branched threads being the mycelium, and the clubs being the growing ends of the hyphæ, such as are common to some hyphomycetes ; these clubs, with their power of sprouting and giving off conidia (the above spherical or oval globules), would render the appearances easily intelligible. Further, the central part is the only part which in any way can be said to represent the portion which is actually degenerating, since it often contains calcareous deposits. This view of considering the clubs as the sprouting parts and conidia-bearing ends, and the threads as analogous to the mycelium of an oidium-like fungus (Bollinger, Israel and Bujwid), is the one which stands better in harmony, we think, with the actual facts than the view that it belongs to a species of cladothrix (Boström, Paltauf, Afanassiew¹). Israel has shown that the ray fungus can be artificially cultivated, but Boström was the first who succeeded in artificially

¹ Sauvagean and Radais (*Annales de l'Institut Pasteur*, vi., 4, p. 242, April 1892) consider actinomyces as a mycelial fungus, *Oospora bovis*.

producing good cultures of this fungus. On blood serum, and on agar at 88–87° C., the fungus forms whitish granules, which rapidly enlarge; they show a yellow or reddish, round, knobbed centre, from which start fluffy nebulous branched masses; after five to six days the growth has reached its height. The presence under the microscope of the branched mycelial threads and of the clubs was established in these cultures. Paltauf and Afannassiew have confirmed these observations. As a rule, however, the granules in the cultures are made up entirely of dense felt-works of branched threads (*see* fig. 118).

On glycerine agar the actinomyces forms a film composed of distinct yellowish-brown round granules; in broth it also forms distinct granules, varying in size from that of a millet seed to that of a small pea; on gelatine good growth can be obtained at 20° C., in the form of a whitish-yellow pellicle made up of granules; the gelatine is liquefied, but remains quite clear.

An important fact, established by Israel, Boström, Rotter and others, is this, that the ray fungus of man can by inoculation produce typical actinomycosis in cattle, and there is therefore the greatest probability that the converse also holds good.

The chronic necrotic disease occurring in India, and known as the madura disease, or the fungus disease, has been shown by Kanthack to be due to, and caused by, a fungus resembling in most respects actinomyces. This was found to be the case in the yellow or pale variety, as also in the black or melanoid kind (Pathological Society of London, January 19, 1892).

TRACHOMA

Another form of chronic round-cell growth, i.e. granuloma, is the trachoma conjunctivæ (also called Egyptian ophthalmia): it is a chronic diffuse infiltration of the conjunctiva with round cells, and at the same time a hyperplastic enlargement of the normal lymph follicles of the conjunctiva. The surface of the conjunctiva, besides being abnormally injected, has instead of its smooth, a finely granular character. This chronic process, the trachoma, does not lead to suppuration, but the infiltration and the swelling of the lymph follicles subside, and after fatty degeneration and absorption of the round or lymph cells, a shrinking and cicatrization of the conjunctivæ take place. Sattler maintains that he discovered in the secretion of the conjunctiva, and in the swollen lymph follicles, a coccus which he cultivated in gelatine, and which he compared to the gonorrhœa coccus. (This last microbe, as has been stated on a former page, does not grow on gelatine.) He also maintains that he produced typical trachoma by inoculation of his cultures into the conjunctiva. The description of what Sattler calls trachoma, produced by inoculation of his trachoma coccus, cannot be accepted as showing that it is genuine trachoma.

Michel described ('Archiv f. Augenheilkunde,' XVI., 1886), and grew in artificial cultures, this trachoma coccus, and maintains that he succeeded by inoculation in producing a swelling of the lymph follicles.

As has been mentioned on a former page, Koch, and also Kartulis, have definitely settled that what is called Egyptian ophthalmia is one of three things: (a) an acute blennorrhœa identical with gonorrhœa and caused by the gonococcus; (b) an acute purulent ophthalmia caused by a minute bacillus to a certain extent similar in morphological and cultural characters to the bacillus of Koch's mouse septicæmia; or (c) the chronic granular ophthalmia known as trachoma. Kartulis has shown that either of the first

two diseases becoming chronic leads to trachoma, and since the specific organisms causing the first two diseases only exist in the conjunctiva during the acute stages, it follows that the result of the disease, i.e. the granular chronic stage, the trachoma, is not directly caused by, or associated with, any organism of its own. This view of Kartulis seems to commend itself more than that of Sattler and Michel. Kartulis failed to obtain any organisms from the tissue of the swollen lymph follicles in trachoma. And we ought to add that Koch also failed to find bacteria of any kind in the lymph follicles in trachoma.

CHAPTER XXVIII

DISEASES DUE TO FLAGELLATE PROTOZOA

IN the chapter on malarial fever mention was made of the presence of crescentic or spherical flagellate bodies, Laveran's corpuscles, which, however, occur only in fever of irregular type. Many species of similar flagellate infusoria are known to inhabit the bodies of invertebrate and vertebrate animals; of these the group known as Monadinæ are in so far of interest that some of them have been found in vertebrates in connection with disease. The genus *Trichomonas*¹ has been found by Pfeiffer in the oral and pharyngeal mucus of pigeons affected with the chronic necrotic thickening of the mucous membrane, called also 'diphtheria,'² the genus which this observer considers to be connected with the cause of the disease. But Löffler (*see* the chapter on DIPHTHERIA) has shown that the disease in the pigeon is due to a specific bacillus, and in this he is fully confirmed by Babes.³ Pfeiffer in the monograph just quoted still maintains his original assertion, that the disease is due to trichomonas invading and ultimately destroying the epithelial cells. Pfeiffer, however, differs, as regards the life history of this protozoön, from all other observers and writers on protozoa (Leuckart, Bütschli, Dallinger and Drysdale), inasmuch as he describes the formation of spores within the substance of the trichomonas.⁴ It ought also to be mentioned that a seemingly identical trichomonas is frequently found in the pharyngeal mucus of perfectly normal pigeons. The writer has had the opportunity of examining a case of this so-called diphtheria in the pigeon, and he found the presence of the parasite in the pharyngeal mucus, but comparing with it a perfectly healthy pigeon, the same trichomonas was also found abundantly in the pharyngeal mucus. Davaine⁵ mentions the genus *Circomonas*, much smaller than *Trichomonas*, a minute club-shaped, ciliated protozoön, possessed of no envelope, having a pointed prolongation at one end, and a long fine flagellum at the other (*Circomonas intestinalis hominis*), as occurring in the stools of cases of acute Asiatic cholera; and once he also found them in the stools of a patient in typhoid fever. Lambl as long ago as 1859 described them as occurring in the stools of children in diarrhoea, and Lösch found them also in the stools in cases of dysentery. The writer has had the opportunity of finding in a dead mouse the peritoneal cavity and almost the whole of the intestine distended by, and filled with, a grumous

¹ Leuckart, *Die Parasiten des Menschen*, 2nd edition, p. 311.

² L. Pfeiffer, *Die Protozoen als Krankheitserreger*, 1890 (Jena), p. 85.

³ *Zeitschr. f. Hygiene*, Band X.

⁴ *Loc. cit.*, p. 85, fig. 26.

⁵ *Traité des Entozoaires*, p. xxiii.

milky fluid, in which, besides leucocytes and micrococci, there were present trichomonas and innumerable circomonas; in fact, the main part of the corpuscular elements was made up of circomonas, many of them moving very rapidly.

A species of flagellate monadinæ was first described by T. Lewis¹ in 1878 as occurring in the blood of normal horses, dogs, and rats; by Evans in 1880 as occurring in the blood of horses in Madrid; by Wittich and R. Koch in 1881 as occurring in the blood of normal badgers. These protozoa are known as the *Herpetomonas Lewisii*: the body is cylindrical, and often spiral, the flagellum extends as a delicate membrane all along the body of the creature, anteriorly the body terminates as a pointed rigid process. A hæmatozoön which, according to Lewis and Crookshank,² is identical with that occurring in the healthy rat was discovered by Evans, but first assumed to be a spirillum, and considered by this observer as the cause of the surra disease, a deadly malady affecting in India horses, mules, and camels in an epidemic form. T. Lewis and afterwards Crookshank³ gave good photographs of them: the former believes them to belong to the genus *Trichomonas*.

CHAPTER XXIX

DISEASES ASSOCIATED WITH COCCIDIA

THE class of protozoa known as sporozoa—unicellular parasites of fixed form of body, surrounded by a capsule, forming within their body a number of spores, each surrounded by a cuticle, the spores becoming free after the bursting of the capsule, and giving rise to a new parasite—comprises a group which is important to the pathologist, viz. oval coccidia or psorospermia. These are capsulated, uninuclear, oval, protoplasmic corpuscles, in the interior of which a number of spores are developed out of the protoplasm; many of these coccidia are endo-epithelial parasites, and as such are the causes of a chronic hypertrophy of the epithelium. The coccidium best studied is the *Coccidium oviforme*, causing in the liver of the rabbit a chronic disease of the epithelium of the bile ducts, by which the bile ducts become greatly distended, their epithelium much hypertrophied, and their coats thickened; in consequence of this, whitish-grey nodules, composed entirely of the hypertrophied folded and fringed wall of the bile ducts, appear in the liver.

The columnar epithelial cells lining those hypertrophied bile ducts which harbour the coccidia are, in fact, the soil at the expense of which the coccidia grow and ripen; these latter in their turn, and for their own purpose, causing a continuous multiplication of the epithelial cells. *Coccidium oviforme* occurs also in the epithelial cells lining the mucous membrane of the intestine in the rabbit. The coccidia are oval corpuscles about 83–87 μ in length, and 15–20 μ broad; each possesses a distinct capsule or cuticle, which at one (thinner) pole contains a minute opening or micropyle. The body of the parasite is a granular protoplasm, in its fully formed state completely filling the space within the capsule, and containing a clear oval nucleus. In this condition

¹ *Physiological and Pathological Researches*, collected papers of T. R. Lewis, 1888, pp. 604 and 680, Plate XLIII.

² *Journal of the Royal Microsc. Society*, November 10, 1886.

³ *Loc. cit.*

they are numerous found in the epithelium, and also free in the cavity of the intestine and the hypertrophied bile ducts respectively; but the majority of forms seen in the epithelium are almost spherical, less oval than the above, and possessing a thinner capsule; in some the capsule is hardly recognisable, they contain a more coarsely granular protoplasm, and within it a clear, spherical nucleus. These are found in great numbers in the epithelium, replacing at points almost completely the epithelial cells, some being distinctly seen within the body of the epithelial cells.

It is not at all easy to decide what is the exact relationship between these smaller, granular, indistinctly capsulated, spherical bodies and the larger, granular, oval, distinctly capsulated coccidia. According to Leuckart,¹ the former would represent young coccidia just germinated from the spores; but this can hardly be correct, considering that in all nodules, particularly the large ones, the small spherical, indistinctly capsulated coccidia abound, and, further, considering that spores are not formed in the coccidia within the animal body. It is therefore more probable that the small spherical coccidia are derived by division from the large oval forms, and in their turn, on their ripening, grow into these latter.

It can be shown that the coccidia first appear in the epithelium of the intestine, and thence find their way into the epithelium of the hepatic duct, and gradually into the bile ducts within the liver. Here their multiplication produces saccular, tubular, and cystic enlargements of the interlobular bile ducts, the wall of which becomes thickened by connective tissue, and folded in many ways. In this manner there are formed in the liver numerous whitish irregularly shaped, firm nodules and cysts, which, when cut into, show a cavity with the thick white wall folded inwards. Under the microscope the folds are made up of vascular connective tissue and covered on the internal or free surface with columnar epithelium, amongst the cells of which numerous coccidia in various stages of development are seen, some of them well buried amongst the epithelial cells, others adhering to the free surface, and others again free in the cavity of the cysts and nodules. As the disease progresses, and larger sections of the liver tissue constantly become involved, necrosis and degeneration by pressure of many liver lobules take place, leading ultimately to the death of the animal. When the coccidia pass out of the animal they undergo changes which lead to the division into two of the plasmatic contents within the capsule; then, again, of each of these into two, so that now each coccidium capsule includes four spherical granular corpuscles, each containing a spherical nucleus; then these corpuscles become oval and surrounded with a thin membrane (see Plate XLI., fig. 97), thus representing now the spores or psorospermia. When these find access, again, through water or food to the alimentary canal of the animal, the capsule probably becoming dissolved in the stomach, the spores are set free, and then commence germinating into granular, spherical, nucleated corpuscles, which, while enlarging, surround themselves with a cuticle or capsule with micropyle, and thus form the typical oval coccidium. The researches of Virchow, Kauffmann, Waldenburg, Lieberkühn, Stieda, and last, but not least, of Leuckart, have thrown a good deal of light on the life history of these parasites. Leuckart in his work '*Die Parasiten des Menschen*,' 2nd edition, I., 1, gives an exhaustive account of them. The figure 97, a-g, reproduced on Plate XLI. is copied from that work. In the human subject nodules of the liver have been observed which were also caused by coccidia, probably *Coccidium oviforme*. Gubler, Leuckart,

¹ *Die Parasiten des Menschen*, 2nd edition, I., 1, p. 264.

Dressler and Perls¹ have observed such cases. Amongst other animals, the *Coccidium oviforme* has been found in the intestines of the dog, cat, sheep, and guineapig. Leuckart states that the intestinal coccidium of the mouse and of poultry belongs to a different species. The writer has seen coccidia in the vessels of the medulla of the kidney both of the guineapig and of the mouse.

Miescher's Coccidia Tubes.—It is not at all rare to find in the muscles of the mouse, wild as well as tame, white streaks visible to the unaided eye as fine white lines between the muscle bundles of the pectoral muscles and of those of the anterior as well as posterior abdominal wall. On examining these lines under the microscope with a low power it is seen that they are composed of tubular granular opaque masses, placed either within the muscular fibres or, as is more common, in the interstices between the muscle fibres. These tubes vary greatly in length, some being several millimetres long, and when between muscular fibres often form continuous chains for considerable distances: the shorter tubes measure 0·7–1 mm. in length. The thickness of the tubes is a little more than twice that of the muscular fibres. They all are tapering towards their extremities. With a high power, a capsule surrounding the dark contents of the tubes can be distinguished. These contents are composed of pale corpuscles, crescentic, kidney-shaped, or, more correctly speaking, looking like thick commas, rounded and slightly attenuated at the ends; in some of them a vacuole can be seen at each end. These crescentic corpuscles measure about 0·01 mm., and are considered to be the spores. Although at first sight they appear densely packed within the tubes, on careful examination they are seen to be grouped into spherical masses, each surrounded by a thin capsule. On teasing out a part of the fresh muscular tissue, many of the tubes become broken, and innumerable isolated crescentic spores are hereby obtained. No movement is possessed by the tubes or the individual spores. That the presence of numerous tubes in the thoracic muscles must be a serious condition with respect to the respiratory action of the chest can hardly be doubted (Dammann, Leisering, v. Niederhausen, and others). The writer has several times had the opportunity of dissecting mice that had died spontaneously, and in which no disease of the viscera could be discovered; but the thoracic muscles and the muscles of the abdominal walls contained the white lines (Miescher's tubes) in enormous numbers. Leuckart does not feel satisfied that these parasites are real psorospERMIA. Fowls sometimes contain the same tubes, but they have not yet been discovered in the human subject.

L. Pfeiffer ('Die Protozoen als Krankheitserreger,' Jena, 1890) describes the presence of coccidia in the epithelium in variola, vaccinia, varicella, herpes zoster, and other vesicular eruptions. From his description and the illustrations given by him (figs. 28–34, pp. 88–97) he has no doubt that they occur in the substance of the epithelial cells; that here they (the coccidia) multiply by division, and form the spores in their interior. It can be easily shown that certain peculiar bodies do occur in the epithelial cells in these affections, which bodies are not the typical ordinary nuclei, but they can be brought out by various dyes, and thereby can be differentiated both from the cell-protoplasm as also from the ordinary cell nucleus. In sections through the vesicles of sheep-pox, as also of human small-pox, stained first with rubin and then with methyl blue, many of the epithelial cells in the region of the vesicle contain each an oval or spherical homogeneous body, which by its pink colour is well marked off both from the cell protoplasm and the swollen and hydropic cell nucleus, both these being stained blue; but it is extremely difficult—and it seems premature and improbable—to identify them as of the nature of extraneous parasites, viz. coccidia: on the contrary, these bodies look extremely like derivatives of the cell nucleus (*see below*).

It is different, however, in the chronic disease known as epithelioma contagiosum of fowls. This consists in the formation of firm tumours in the mucous membrane of the throat, and also in the skin. Sections through the tumour show the papillæ much enlarged and vascular; they and the corium are infiltrated with round cells, and the epithelium is much hypertrophied.

¹ See Leuckart, *loc. cit.*, p. 281.

Pfeiffer¹ describes the presence of oval coccidia in the epithelial cells of the tumour. The writer has had occasion to examine sections through the tumours of the skin of a fowl affected with this disease, and he can fully confirm Pfeiffer as to the presence of the oval parasites in the epithelial cells, in some parts every epithelial cell containing, and almost being replaced by, a parasite, the latter being easily brought out by staining the sections first in fuchsin and then in methyl blue.

CANCER

Assertions similar to these, i.e. as to the presence of coccidia, and which are of the utmost importance in etiological and clinical respects, have been also made with regard to cancer, viz. that this disease is due to the presence and growth of coccidia in the epithelium; just as in the above-named nodules in the liver of the rabbit and in the epithelioma contagiosum, the invasion of the epithelium or connective tissue cells—according to the nature of the cancer—by coccidia causes a hypertrophy of the tissue, causes, in fact, the tumour, i.e. epithelial carcinoma or sarcoma. This hypertrophy serves the purpose of supplying the parasite with the host necessary for its growth and multiplication. If this view be correct, cancer would have to be placed amongst the infectious or communicable diseases. That cancer is probably of this nature is indicated already to a certain extent by the following well-established facts:—(1) Heredity. That cancer is hereditary needs no further comment, and like other hereditary diseases is probably of the nature of infectious disorders. The theory that if a disease is inherited this is due to the transmission from parent to the offspring of the *materies morbi*, is the most ready explanation. (2) The well-known fact that cancer is transmitted in the affected individual from a primary focus to different organs, thus forming metastatic foci. (3) The positive experiments, made within recent years, of successful grafting of cancerous particles from a primary focus to distant parts of the skin of the same individual, by which in the latter new cancerous tissue became developed.

But against these facts must be placed the uniform negative results of producing by inoculation or grafting with fresh cancer material anything like cancer in animals otherwise otherwise subject to cancer (dogs). (See the carefully conducted numerous experiments by Ballance, Shattock, and many other experimenters.) Cancer in the mammary gland of bitches is not of rare occurrence; it completely coincides with epithelial cancer in the human subject, yet all inoculations and experiments of grafting of such cancer material on to healthy dogs have hitherto failed. But such negative results are only of relative value, since the conditions under which an individual becomes susceptible to cancer are not known, and therefore one of the essentials of a successful experiment is absent.

There exists at present a large amount of literature on the occurrence of coccidia in the epithelial cells constituting cancer; by various methods of staining, special bodies, oval and spherical, some containing what look like numbers of spores, have been described by various observers. They were noticed in Paget's 'Disease of the Nipple of the Breast,' and in other cancers. (Darrier, Albarran, Wickham, Thoma, Sjöbring. For the literature see Sjöbring in 'Fortschritt der Medizin,' VIII., No. 14, 1890). Russell, and particularly Podwyssozki, Jun., and Sawtschenko ('Centralbl. f. Bakter. und Parasit.,' XI., Nos. 16-18) give a very good account of these bodies, the latter also describing spores in them. According to these authors the presence of

¹ *Loc. cit.*, p. 81, fig. 24.

these assumed coccidia produces the epithelial multiplication for the object of supplying the parasite with a sufficient host.

There can be no question as to the facts described ; the only question is as to whether the interpretation given by these observers, in regard to these bodies being coccidia and spores, is the correct one. The interpretation as to these bodies being parasitic in nature is perhaps not quite unjustifiable ; but until their life history is better known, and until they have actually been seen in a living and growing state, i.e. in culture, and until the assumed spores have been seen to be capable of germination, it is advisable to be very cautious, without, however, denying the possibility that those bodies are really what they are said to be, viz. coccidia or something similar. In connection with this it might, however, be mentioned that bodies similar to these assumed coccidia are known to occur in the substance of epithelial and other cells under normal and various pathological conditions. Thus, Grützner noticed in certain phases of activity of certain glandular epithelial cells, the presence in the cell protoplasm, besides the typical nucleus, of a secondary nucleus-like body, probably corresponding to Balbiani's paranucleus. Other observers, again, have described in various pathological processes of epithelium in the protoplasm of the cells peculiar masses, spherical and oval, looking and staining differently from the main protoplasm. The caution enjoined by such experienced histologists as Klebs and Eberth¹ should be carefully considered by all less-experienced histologists before emphatically asserting (as is done by some) the parasitic nature of those bodies. What remarkable changes do occur in nuclei and cells, leading to the presence in the cell protoplasm of peculiar bodies, differentiated by complicated methods of staining, is well illustrated in a paper by Dr. Martin Heidenhain.² In Heidenhain's memoir in Plate X., bodies are shown in the cell protoplasm of leucocytes, brought out by special methods of staining, which are derived from the original cell nucleus ; and, further, in Plate XI., the presence of numbers of peculiar corpuscles, which bear a striking resemblance to what the above writers on cancer consider as spores in the epithelial cells of cancer, is differentiated in the cell protoplasm ; yet there can be no question that in Heidenhain's observations the bodies described and figured by him have nothing whatever to do with any intracellular parasites.

CHAPTER XXX

BLASTOMYCETES, OR TORULA

THESE organisms are of interest to the pathologist in two ways : (a) by their being constantly present in air, and consequently occurring on the skin and the surface of the mucous membranes in contact or in communication with the air ; and (b) by a species of torula being connected with a well-defined disease, to wit, *thrush* in infants.

Torula is capable of producing alcohol when growing in substances containing glucose. By torula is understood an oval microbe varying in length between 2 or 3 μ to 5 or 6 μ , consisting of a membrane and protoplasmic contents, including one or two vacuoles. Torulas multiply not by fission but by gemmation, as shown in Plate XII., fig. 98. They are

¹ *Fortschritte d. Med.*, VIII., No. 17.

² *Ueber Kern- und Zellprotoplasma*, Leipzig, 1892

capable of forming endocellular spores—ascospores (Rees). A large number of different species of torulas are known and have been carefully studied and cultivated by Hansen (*see* his various memoirs in the 'Berichte des Karlsberg Laboratorium'), and their ability to produce alcohol from glucose under various conditions of growth minutely studied by him.

Different species of torula are constantly to be found in the air, soil, drinking water, and in river and pond water; a plate of sterile gelatine, after the setting of the gelatine, exposed to air or infected with a drop of water, covered up again, and then incubated, not infrequently reveals the presence of colonies of torula, besides the usual colonies of moulds and bacteria. Amongst these torulas some species are characterised by their growth being coloured pink; at least two species of such pink torulas can be obtained from London air—one species which liquefies gelatine and one species which does not liquefy it. In the fluid of the mouth and pharynx, torulas are normally present; in the contents of the large intestine, they are normally present. In a case of diabetes from which Dr. Bedford Pierce procured urine drawn from the bladder, the freshly drawn acid urine was found uniformly turbid, owing to the presence of torulas; in fact, the urine contained a pure culture of torula, as was shown by plate and tube cultivations. This torula grew well in acid grape-sugar gelatine, and changed the sugar into alcohol; on ordinary alkaline nutrient gelatine (beef broth, peptone gelatine) it grew but slowly and not copiously, forming a whitish, grey, moist-looking growth. Pathological products of the skin and mucous membranes, i.e. those exposed to the air, contain torula; thus, for instance, vaccine lymph from the calf sometimes contains a few torula cells.

The torula species connected with alcoholic fermentation grows well on acid sugar gelatine, forming white round colonies in gelatine.

A definite species of torula, *Saccharomyces mycoderma*, is capable of forming acetic acid in wine, beer, and in fermenting cabbage (Sauerkraut). During its growth it forms a scum or pellicle on the surface, being dependent on the oxygen of the air. Its cells are oval, 6 μ long, 2–3 μ broad; when growing in fluids it forms spores, just like other torula species, the cells enlarging and containing, i.e. forming in its interior, the spores—ascospores (Rees).

When growing in fluids containing alcohol it oxidises the alcohol into acetic acid, and thus in its chemical action is analogous to the bacterium mentioned in a former chapter as *bacterium aceti*. Cienkowski first showed that in acid nutritive fluids the cells elongate, and by gemmation produce new cells, which elongate and remain joined to the mother cell, and by the repetition of this process form threads composed of oval or cylindrical cells, the older the more elongated. In this way septate threads are formed, which, just like a mycelium of a true fungus, form branches. A torula, morphologically identical with this, is the species known as *oidium albicans*, and as being the active cause of thrush (Plate XL., fig. 100). Grawitz,¹ who worked this out satisfactorily, has traced it through all phases of growth, and has also shown that, like other *saccharomyces*, it forms ascospores. It grows well on nutritive gelatine, and in streak cultivation forms a greyish-white fluffy growth, in which the formation of threads is actively going on.

¹ *Virchow's Archiv*, Band LXX.

CHAPTER XXXI

MOULDS, OR MYCELIAL FUNGI

THIS group comprises microbes which consist of cells multiplying by fission, and which by continued linear and lateral multiplication, and by elongation, form branched mycelial threads; each of these is composed of cylindrical cells. The ripe cells consist of a cellulose sheath, and within is a homogeneous substance, or rather the remains of a faintly granular protoplasm, filled with or replaced by clear vacuoles. The cells are separated from one another by a transverse septum of cellulose. The young and growing threads, both terminal and lateral, do not show septa, but have their sheaths filled with a uniform slightly granular protoplasm not segmented into separate cells; the ends of such threads are rounded.

In some species the terminal threads by a process of simple fission of their protoplasm produce oblong or spherical cells, which ultimately become altogether separated from one another and free; these cells are the conidia or spores. Such species are spoken of as oidium, of which there are known several, e.g. *oidium lactis*, *oidium* of *favus* (*Achorion Schönleini*), *oidium* of ringworm (*Trichophyton tonsurans*), and of pityriasis versicolor (*Microsporon furfur*). In these species no other mode of the formation of spores has yet been ascertained. The spores by germination elongate, and by continued growth and subsequent division and septation give rise to the branched mycelium composed of cylindrical cells.

But there exist fungi, like *Aspergillus*, *Mucor*, and *Penicillium*, which, while growing under suitable conditions, particularly when growing on a surface exposed to free air, show a more complex mode of spore formation; but which, when growing in a fluid medium and not exposed to free air, behave like an *oidium*, that is to say, they form spores by simple fission of the protoplasm of the terminal portions of the mycelial threads.

Oidium lactis forms on milk, bread, paste, potato, gelatine, &c., a whitish filamentous growth; its spores are spherical or oval, measuring 7–10 μ (Plate XL., fig. 99).

The *oidium* of *favus*, and that of herpes, of ringworm, and of pityriasis versicolor behave in morphological and cultural characters very much like the *oidium lactis*.¹ Just as in this, the spores are spherical or oval, 7–10 μ in length, and when germinating, elongate, and by repeated division form branched septate mycelial threads; the terminals of these form chains or clusters of spores. On agar and on gelatine the growth has a white colour, and appears fluffy (Plate XLI., fig. 101).

In ringworm, one of the loose hairs examined in liquor potassæ shows the spores in multitudes on the surface of the hair, and between the cells constituting the hair substance. A fresh hair of a ringworm patch is placed in melted nutrient agar; this is shaken and then poured out on a plate for plate cultivation. After several days' incubation at 37° C. numerous colonies of the *oidium* fungus pervade the agar. In order to avoid as much as possible accidental bacteria, it is best to make from the first tube, after inoculation and shaking, a second inoculation, that is to say, by inoculating with a platinum loop a second tube of agar, and use this for a plate cultivation. The number of *oidium* colonies will be found limited, and bacterial colonies probably altogether absent.

¹ Grawitz, *Virchow's Archiv*, Band LXX., p. 560.

Aspergillus.—The spores of species of *Aspergillus* abound in air, water, in the mucus of the nasal cavity, in the fluid of the mouth, and in most substances exposed to air and subjected to putrefaction.

During the growth of the mycelium some of its branches assume an upright position, are thicker and not at all, or only slightly septate, and at their free end form a flask-shaped enlargement, from which grow out radially thin cylindrical cells—*basidia*. These at their distal or free end produce a chain of spherical spores or conidia (Plate XL, fig. 104). This is a very common mould, and according to differences in the colouration of the mycelium and spores is subdivided into different species: *A. glaucus*, *candidus*, *flavescens*, *fumigatus*, *niger*, &c.

Besides this mode of spore formation (asexual), there is another (sexual), which according to De Bary consists in this: some terminal branch of the mycelium becomes twisted like a spiral; this is considered to be the female organ of fructification or *carpogonium*; from the same thread branches grow towards the carpogonium; one of these branches becomes fused with the terminal portion of the carpogonium called the ascogonium, while the others, the *pollinodia*, branch and surround the carpogonium like a capsule; the whole organ is now called a *perithecium*. Finally, the ascogonium by rapid division gives origin to a number of oval septate tubes, inside which by endogenous formation numerous spores make their appearance.

Grohe¹ was the first to show that the introduction of the spores of some species of *Aspergillus* into the vascular system of rabbits sometimes produces death, with symptoms of metastasis into the various organs, due to localised foci, where these spores grow into mycelial filaments. Lichtheim² showed that such mycoses in rabbits cannot be produced by the spores of *Aspergillus glaucus*, but easily by those of *Aspergillus flavescens* and *fumigatus*, in less degree by *Aspergillus niger*. Grawitz³ studied this process more minutely, and found that, whether the spores are injected into the vascular system or into the peritoneal cavity, there are established in the kidneys, liver, intestines, lungs, muscles, and occasionally in the spleen, long bones, lymphatic glands, nervous system, and skin, minute metastatic foci, due to the growth of the spore into mycelial filaments, with imperfect organs of fructification, but no spore formation. Grawitz thought that the spores of ordinary moulds (*Penicillium* and *Aspergillus*) are capable of assuming these pathogenic properties if cultivated at higher temperatures (39–40° C.) and in alkaline media. These fungi, as is well known, grow well at ordinary temperatures and in acid media, and are then innocuous when introduced into the animal body; but by gradual acclimatisation they can also be made to grow at higher temperatures and in alkaline media, when they assume pathogenic properties, becoming capable of resisting the action of living tissues and of growing in them. This view has been proved to be incorrect by Gaffky,⁴ Koch,⁵ and Leber.⁶ Those spores that do exert such pathogenic properties are not at all dependent on such acclimatisation, and are not ordinary moulds, but distinct species of *Aspergillus* (Lichtheim), which grow well at higher temperatures—such as 38–48° C.—and the spores of which under all conditions of growth are capable of producing in rabbits the mycosis in question.

Penicillium.—This fungus has also a wide distribution, but is not so common as *Aspergillus*. In *Penicillium* as in *Aspergillus*, hyphæ, which are

¹ *Berl. klin. Woch.*, 1871.

² *Ibid.*, IX. and X., 1882.

³ *Virchow's Archiv*, Band LXXXI., p. 355.

⁴ *Mittheil. a. d. k. Gesundheitsamte*, 1880.

⁵ *Berl. klin. Woch.*, 1881.

⁶ *Ibid.*, 1882.

not septate, grow out from the mycelium; from the end of each of these hyphæ arise like the fingers of the hand a number of short, branched, cylindrical cells, each of which gives origin to chains of spherical spores (see Plate XLI., fig. 102). Of this no pathogenic species are known.

Mucor occurs on many substances subject to putrefaction, but is less common than either of the two preceding; it is characterised by this, that from the mycelium, non-septate hyphæ grow out, at the end of which a spherical enlargement appears, *sporangium*, in which by endogenous formation a large number of spherical spores are developed; the wall of the sporangium giving way, the spores become free (see Plate XLI., fig. 108).

Lichtheim¹ describes two species, which possess pathogenic action, *Mucor rhizopodiformis* and *Mucor corymbifer* (see Plate XLI., fig. 108). The mycelium of the former is at first white, then changes to grey; that of the latter is always whitish-grey. Both species were found growing on wheat bread kept at higher temperatures (87° C.). Spores of either, injected into the vascular system of rabbits, produce death in forty-eight to seventy-two hours. On *post-mortem* examination the Peyer's patches of the ileum are found intensely congested, swollen, and even ulcerated; in these, as also in the swollen and congested mesenteric glands and in the kidneys, mycelial growths are present. Both these species grow luxuriantly at the body temperature.

An important case of general '*mycosis mucorina*' in man, ending in death, has been described by Dr. Paltauf.² From the alimentary canal of the patient an invasion of the internal organs by the mycelium and spores of a species of *Mucor* occurred, leading to the formation of metastatic inflammatory foci in the Peyer's glands, lungs, pharynx, larynx, cerebrum, and cerebellum. In all these organs were found smaller and larger foci of inflammation, caused by the presence of non-septate branched mycelial threads and sporangia, showing that the fungus belonged to the group of *Mucor*.

Saprolegnia (Plate XLI., fig. 107)—colourless tubular threads, forming gelatinous masses on living and dead animal and vegetable matter in fresh water. The cylindrical or flask-shaped ends of the threads, *zoosporangia*, form in their interior numbers of spherical or oval spores, *zoospores*, possessed of locomotion (one flagellum at each pole), and which finally escape from the threads. These zoospores after some time become resting spores, surround themselves with a membrane, and finally germinate into a cylindrical mass which becomes transformed in the mycelium. Besides this asexual, there is a second or sexual mode of fructification, consisting in this: At the end of a mycelial thread a cell grows up into a spherical large ball, the *oogonium*. From the same thread, thin threads, *antheridia*, grow towards the oogonium, into the protoplasm of which they merge. This latter then differentiates into a number of spherical masses, the *oospores*, which become invested with a membrane; these become free and then germinate and grow into a mycelium. *Saprolegnia* grows on the skin of living fish, and causes here severe illness, often terminating in death. Thus the salmon disease, as Professor Huxley has shown,³ is caused by this parasite. The zoospores of this salmon saprolegnia are, however, as Huxley has shown, as a rule, non-motile. The hyphæ of the fungus traverse the epidermis in the diseased patches of the salmon, and they bore through the superficial layer of the derma, a stem part being situated in the epidermis, and a root

¹ *Zeitschrift f. klin. Med.*, Band VII., Heft 2.

² *Virchow's Archiv*, Band CII. and CIII., p. 543.

³ *Proceedings of the Royal Society*, 1882, No. 219.

part in the derma ; each of these elongates and branches out. 'The free ends of the stem hyphæ rise above the surface of the epidermis, and become converted into zoosporangia, more or fewer of the spores of which attach themselves to the surrounding epidermis and repeat the process of penetration.' In *Saprolegnia* associated with the salmon disease, Professor Huxley observed only the asexual mode of fructification.

PLATES I-XVII inclusive—figures 1 to 118 (except figures 45, 46, 69, and 86)—are from micro-photograms by A. PRINGLE and E. C. BOUSFIELD. Except when otherwise stated, the magnifying power is 1,000.

PLATE I

FIG.

1. Cover-glass specimen from a culture of *staphylococcus albus liquescens*. A. P.
2. From a culture of a sarcina-like staphylococcus A. P.
3. From a culture of *sarcina lutea* E. C. B.
4. From a specimen of *micrococcus tetragonus* A. P.
5. *Micrococcus scarlatinae*, from a culture on gelatine . . . A. P.
6. From a cultivation of bronchial sputum of influenza, showing chains of short rods, many of these resembling a diplococcus . . . A. P.
7. From a culture of a short oval bacillus from veal pie that had caused choleraic diarrhoea. The bacilli are marked as short ovals, each with polar granules A. P.

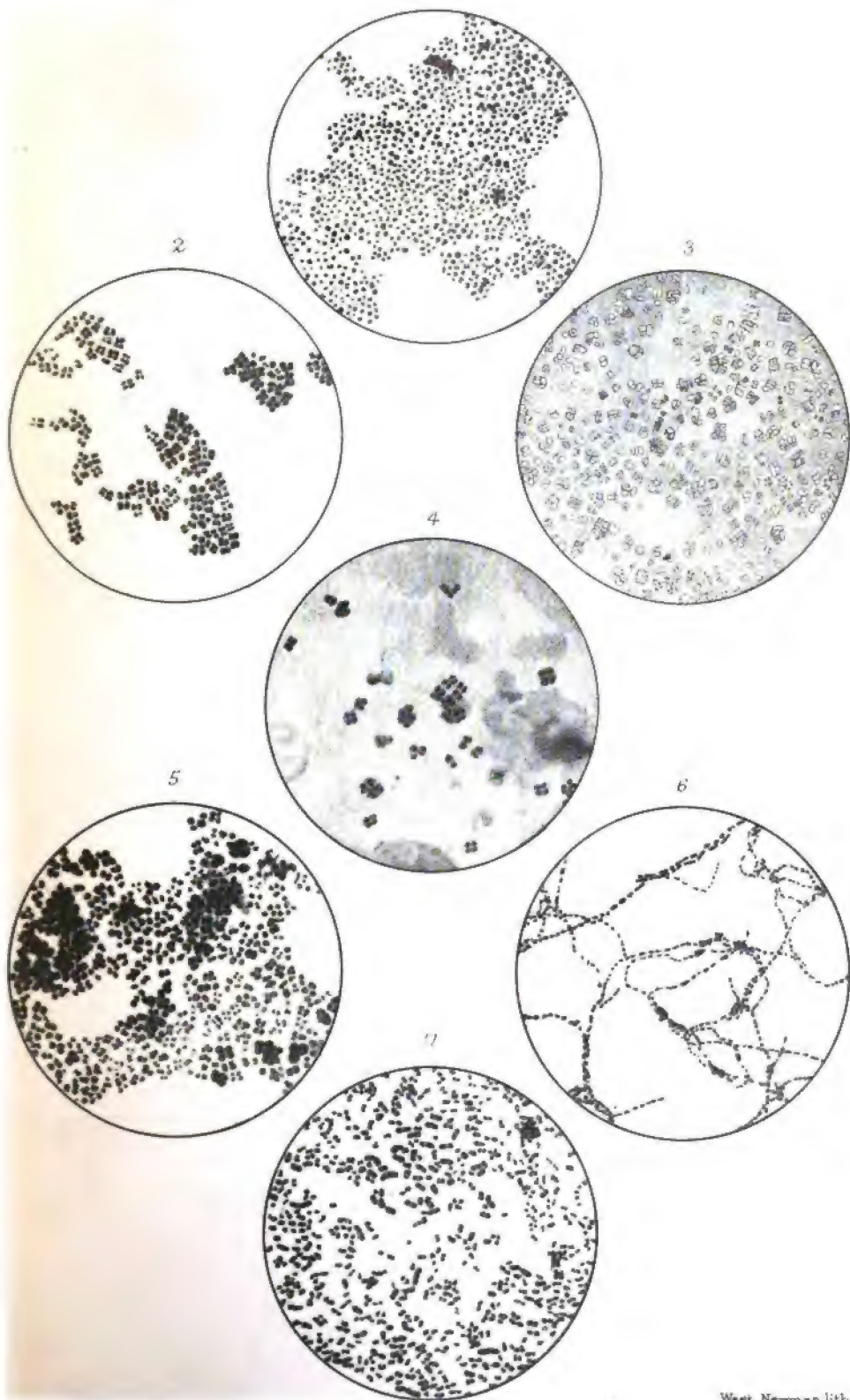


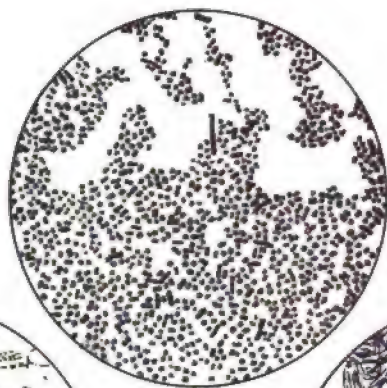


PLATE II

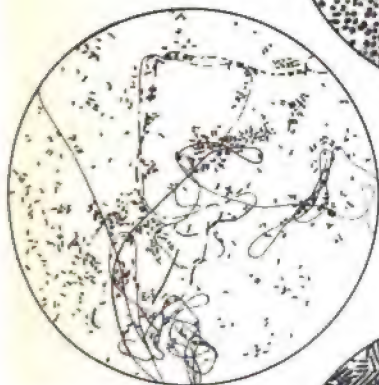
FIG.

8. From a cultivation of bacillus of grouse disease obtained from an infected mouse. Amongst the short oval forms there are some which are more distinctly cylindrical bacilli A. P.
9. From a cultivation of a proteus-like bacillus obtained from pork which had caused choleraic diarrhoea in Carlisle A. P.
(All forms between cocci and long threads are present. $\times 650$.)
10. From very young colonies on gelatine of proteus vulgaris, as seen under a low power. The 'swarmers' passing out from the colonies are well seen A. P.
- 11 and 12. From the same specimen as fig. 10 magnified 1,000. The thread-like character of the bacilli is well shown A. P.
13. From a plate culture of bacillus mesentericus on gelatine. The figure represents the margin of a young colony A. P.
14. Bacillus filamentosus from a gelatine culture of sewage. The cylindrical nature of the bacilli constituting the chains and threads is well shown.
A. P.

8



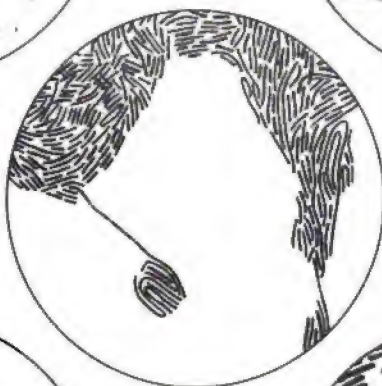
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PLATE III

FIG.

15. From a gelatine culture of bacillus anthracis two days old ; some of the threads show the constituent bacilli as spherical or oval torula-like forms A. P.

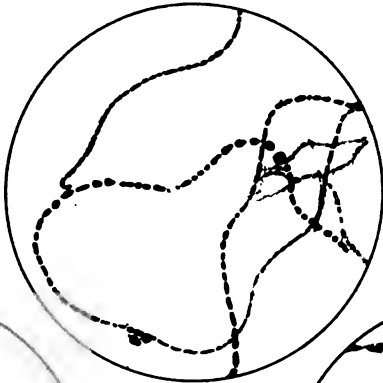
- 16 and 17. From a recent gelatine plate cultivation of bacillus anthracis. The bacillary threads are composed of spindle-shaped elements, many of them containing small vacuoles A. P.

18. From a culture on gelatine of a non-liquefying, extremely minute bacillus that had been found in dust of a room. Each bacillus contains in its middle an oval spore A. P.

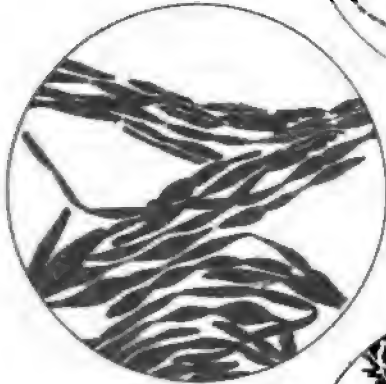
19. From a culture of bacillus filamentosus, the threads containing oval spores A. P.

20. From a gelatine culture of the typhoid bacillus, each bacillus being possessed of numerous flagella E. C. B.

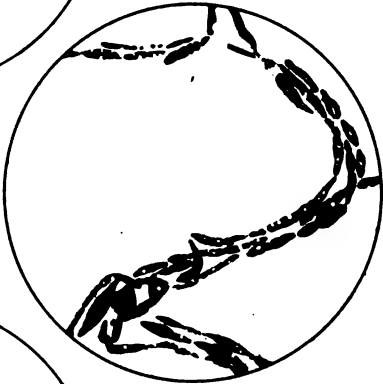
21. Spirilla, each with two flagella at one end A. P.



16



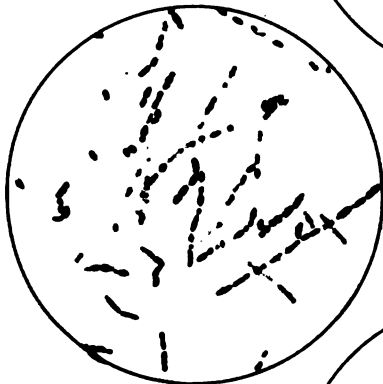
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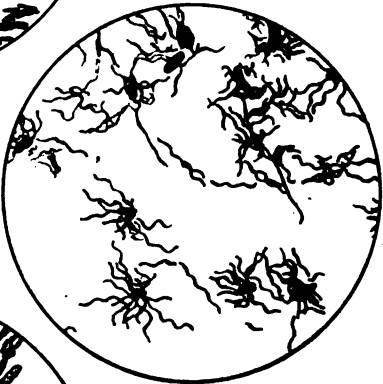
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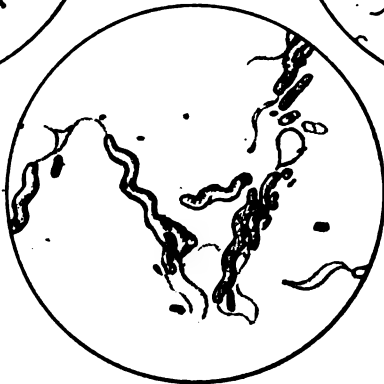
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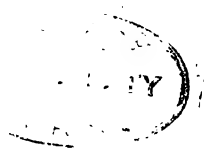
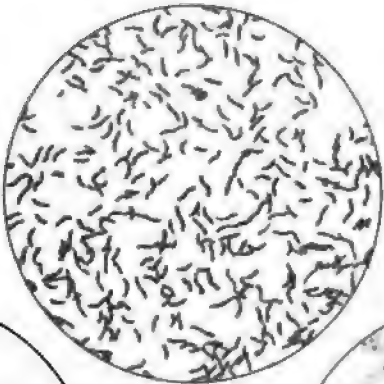


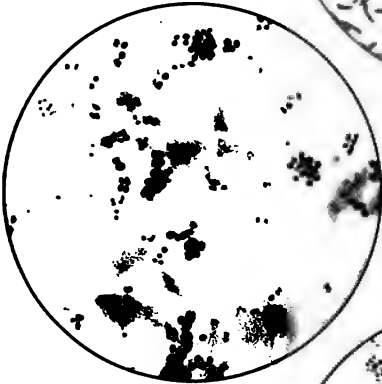
PLATE IV

FIG.

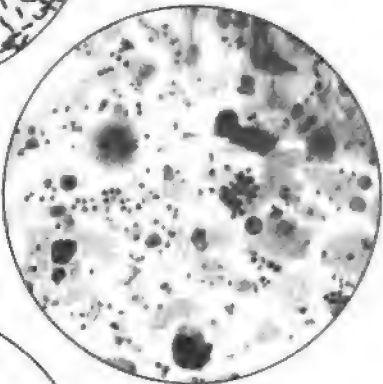
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|---|----------|
| 22. From a culture in gelatine of the comma bacillus of Finkler | . A. P. |
| 23. Purulent matter of abscess of rabbit, numerous cocci . . . | . A. P. |
| 24. Purulent matter of abscess of the human mastitis . . . | . A. P. |
| 25. Streptococcus scarlatinae from an agar culture . . . | . A. P. |
| 26. Streptococcus scarlatinae from culture | . A. P. |
| 27. Streptococcus of foot-and-mouth disease, culture . . . | . A. P. |
| 28. Bacillus pyocyaneus, culture | E. C. B. |



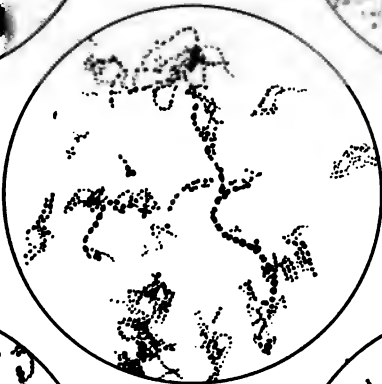
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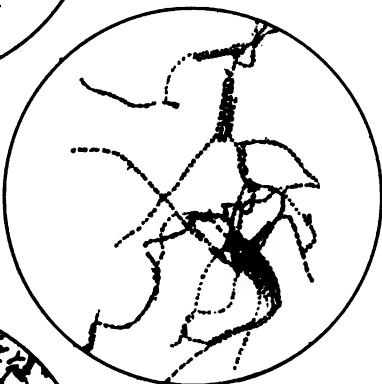
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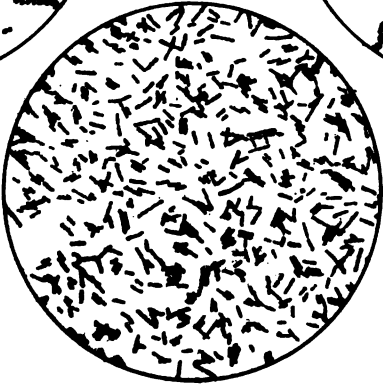
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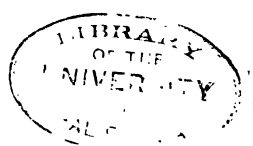


PLATE V

Fig.

29. Section through human variola, showing the loculi forming the vesicle.
 Low power A. P.

80. Section through the inoculated pock of sheep, showing the loculi forming
 the vesicle. Low power A. P.

81. Section through the natural eruption of foot-and-mouth disease in the
 sheep, showing the nature and formation of the vesicle in the
 epidermis. Low power E. C. B.

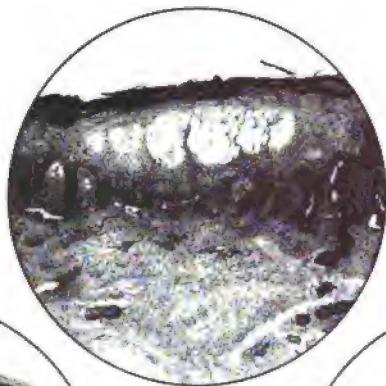
82. Cover-glass specimen of the lung juice in croupous (Middlesbrough)
 pneumonia. Amongst the red blood discs, there are numerous short
 bacilli, single and as diplo bacilli A. P.

84. Section through the lung in the stage of red hepatisation in croupous
 (Middlesbrough) pneumonia; the alveoli of the lung are filled with
 fibrin and red blood discs. Low power A. P.

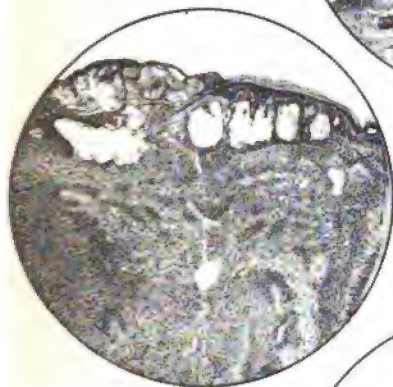
88. From the same lung, more highly magnified. $\times 650$ A. P.

85. Cultivation of the bacillus pneumoniæ (Middlesbrough). A. P.

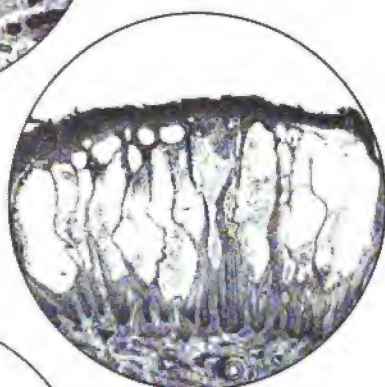
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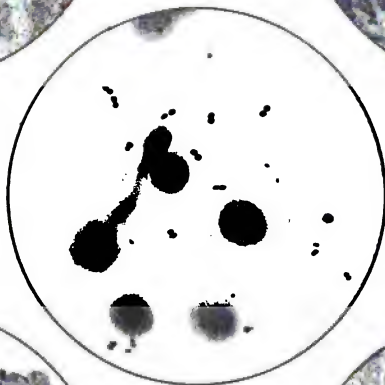
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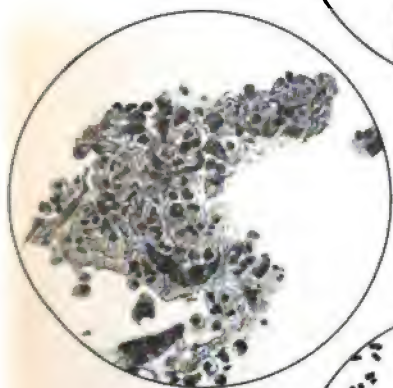
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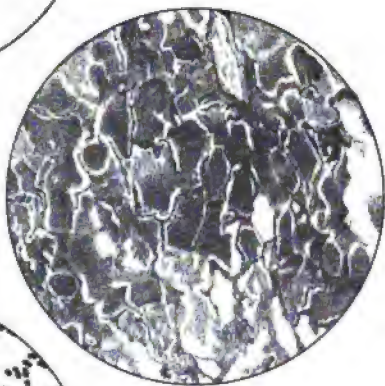
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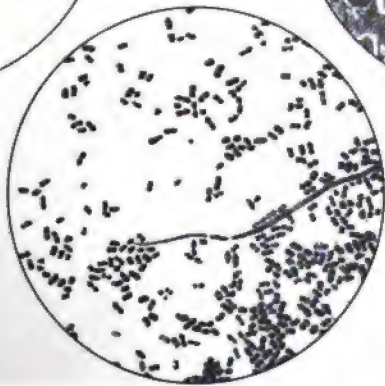




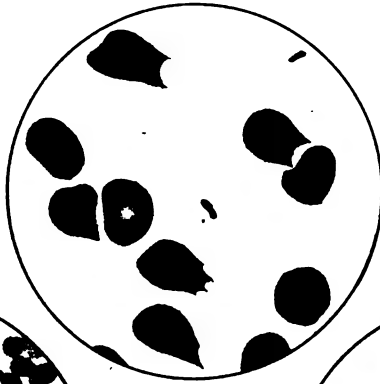
PLATE VI

Fig.

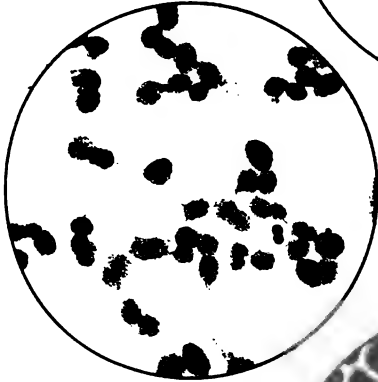
86. Blood of mouse infected with the bacillus pneumoniæ . . . A. P.
87. Bacillus (capsulated) of Friedländer in pneumonic sputum . . A. P.
88. Streptococcus pyogenes in chronic abscess. Amongst the pus cells, chains
of the streptococcus are shown . . . E. C. B.
89. Blood of mouse dead of Koch's mouse septicæmia . . . E. C. B.
40. Œdema fluid of a guineapig dead of malignant œdema, after inoculation
with garden earth . . . E. C. B.
41. Œdema fluid of rabbit dead after inoculation with the aërobic bacillus of
malignant œdema . . . E. C. B.
42. Culture in broth of the aërobic œdema bacillus . . . E. C. B.

Vol. II.

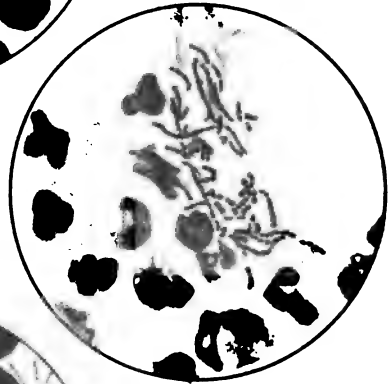
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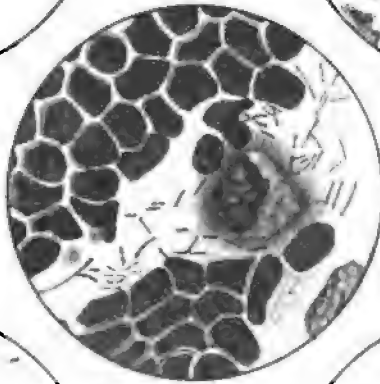
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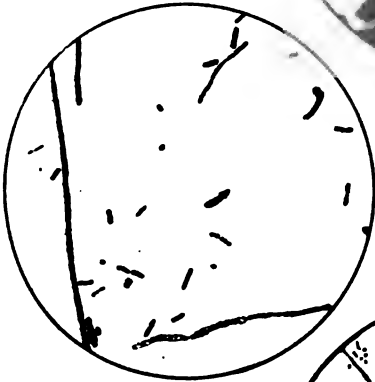
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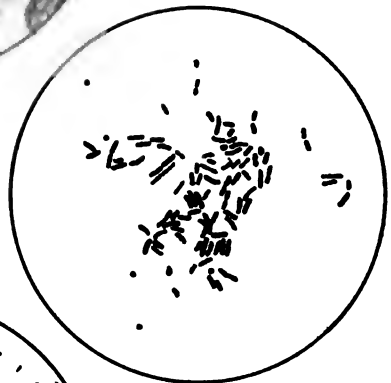
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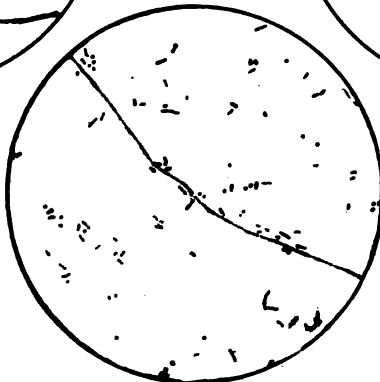
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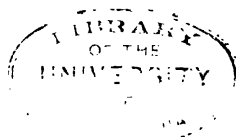


PLATE VII

FIG.

43. Section through the subcutaneous tissue of a guineapig dead after inoculation with the aerobic oedema bacillus E. C. B.

44. Oedema fluid of a guineapig dead after inoculation with (Koch's) anaerobic oedema bacillus E. C. B.

45. Copied from Fränkel and Pfeiffer's Atlas, Plate XXIII, fig. 45. Cover-glass specimen of tissue juice of a guineapig dead after infection with garden earth. Magnifying power, 1,000.

46. Copied from Fränkel and Pfeiffer's Atlas, Plate XXIV, fig. 48. Spore formation in the bacilli of a culture in agar. Magnifying power, 1,000.

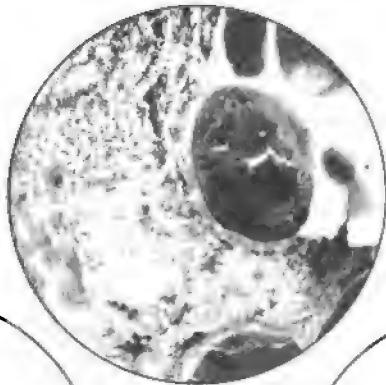
47. Blood of grouse dead of the grouse disease, showing the bacilli amongst the blood corpuscles, of which only the nuclei are shown . . . A. P.

48. Bacillus of grouse disease in the blood of ammer, dead after inoculation with culture A. P.

49. Blood of guineapig infected with culture of the bacillus of grouse disease. The bacilli are very numerous present, either free in the plasma or enclosed in white blood cells, the latter swollen up and disintegrating A. P.

Vol. II.

43



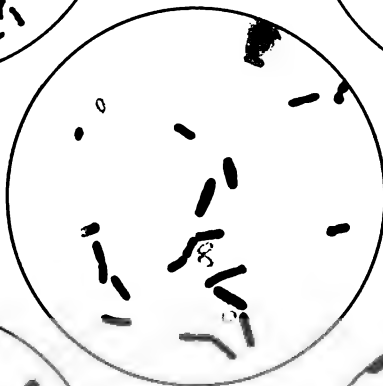
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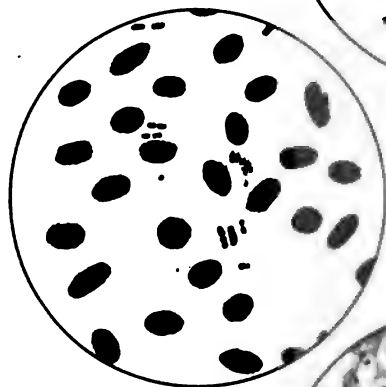
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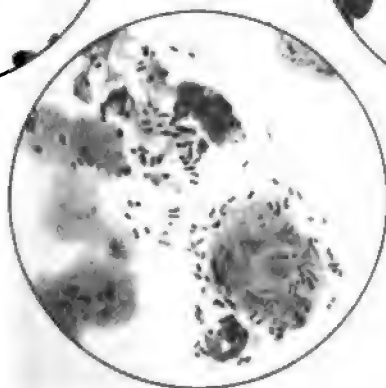
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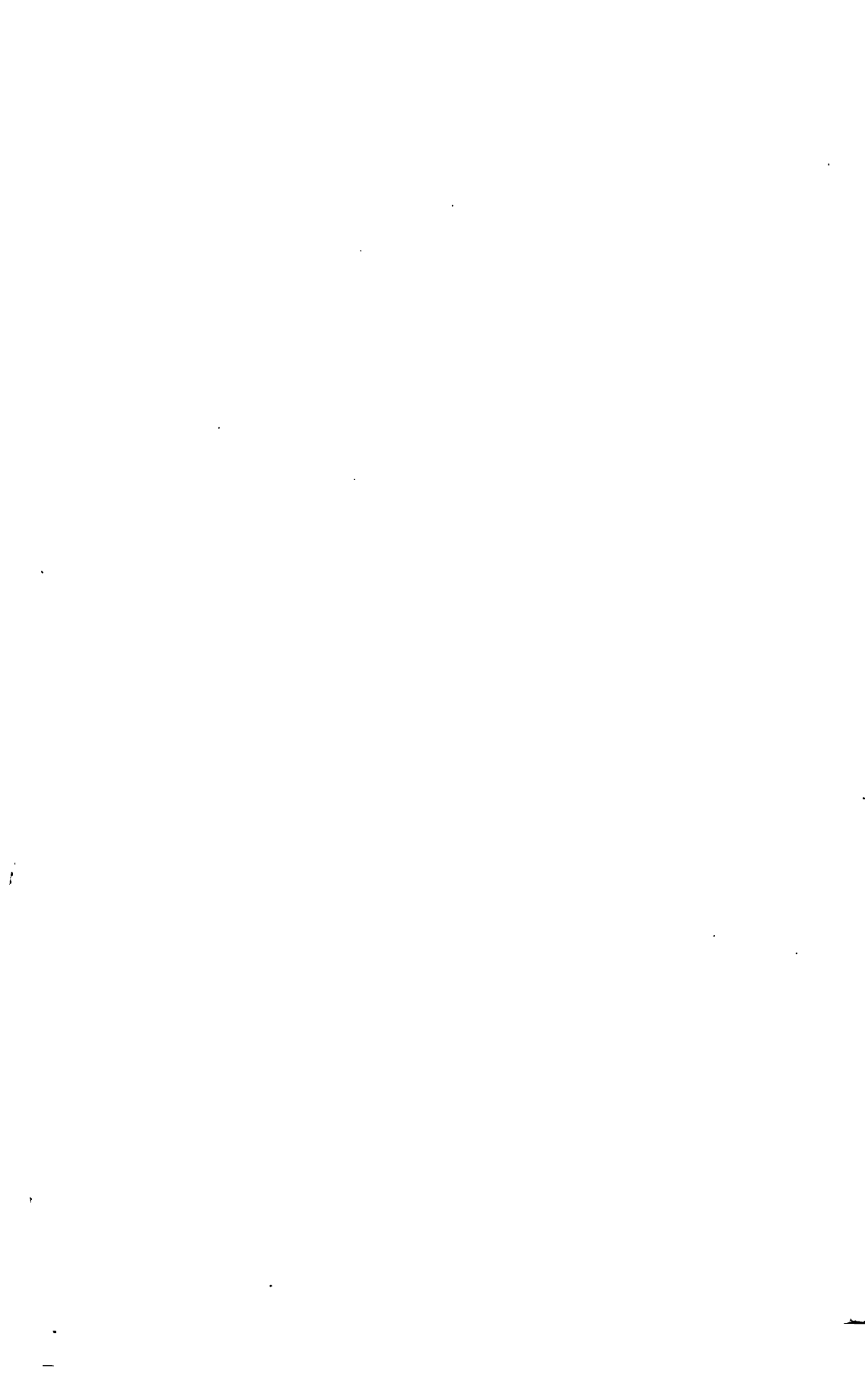


PLATE VIII

FIG.

50. Blood of fowl dead of fowl enteritis: very few bacilli amongst the red
blood discs A. P.

51. Intestinal mucus of fowl dead of fowl enteritis. The bacilli are present
in almost pure culture A. P.

52. From a culture of the bacillus of fowl enteritis A. P.

53. Blood of fowl dead of fowl cholera. The minute oval bacilli are very
abundant amongst the blood discs. The bacilli show the characteristic
staining of polar granules A. P.

54. Blood of rabbit dead of fowl cholera A. P.

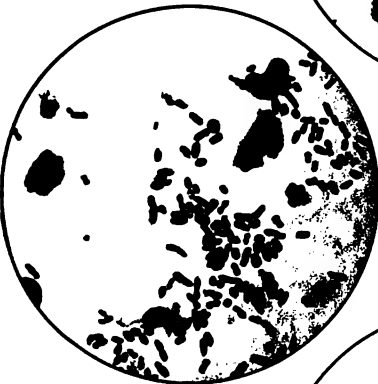
55. Cultivation of the bacillus of fowl cholera A. P.

56. Bacillus of swine fever, from the spleen of a mouse dead after inocula-
tion A. P.

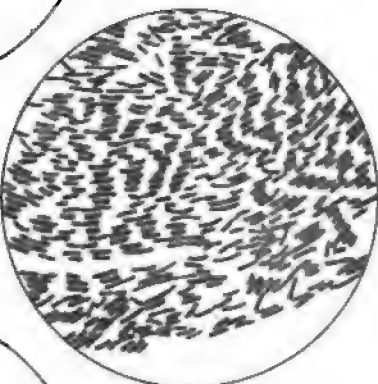
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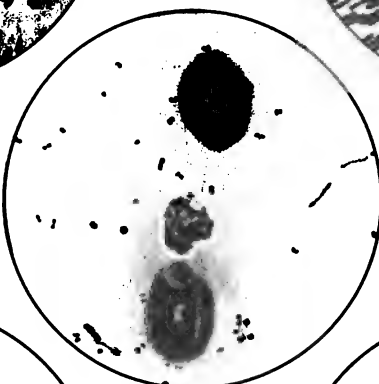
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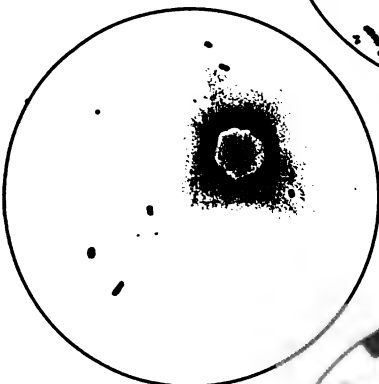
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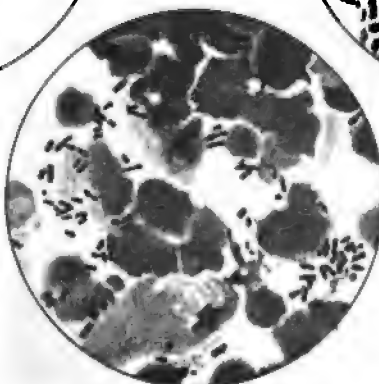
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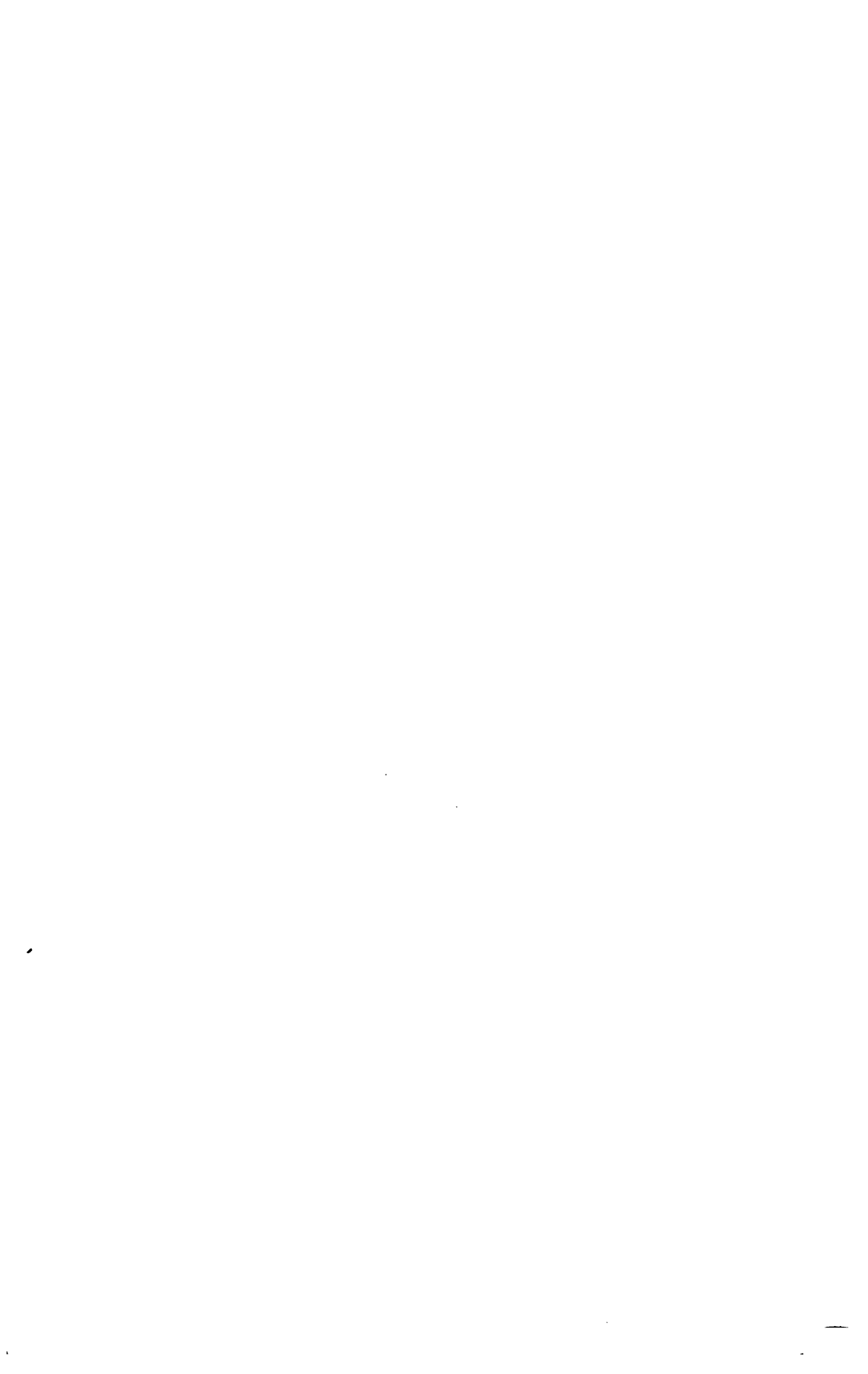
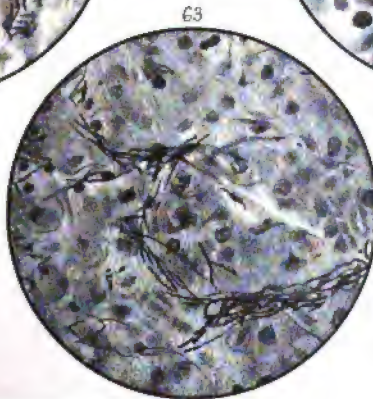
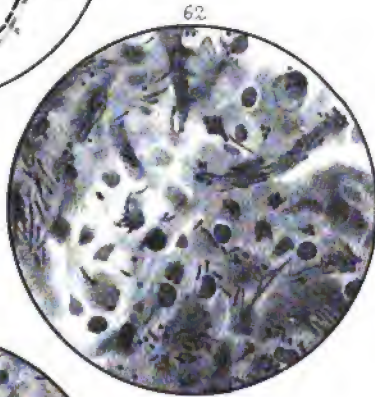
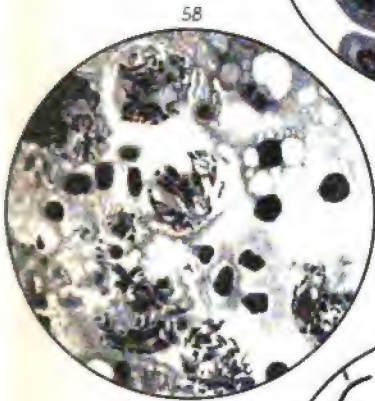
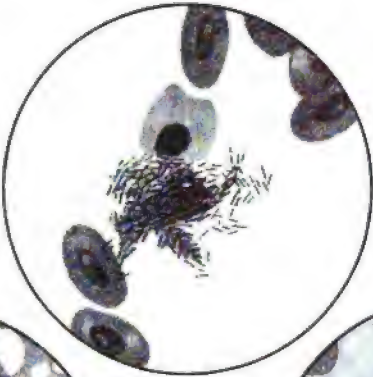


PLATE IX

FIG.

57. Blood of pigeon dead after inoculation with culture of the bacillus of swine erysipelas E. C. B.
58. Section through the liver of pigeon dead of swine erysipelas. In the capillary blood-vessels leucocytes are seen, the interior of which is filled with the bacilli E. C. B.
59. Blood of guineapig dead of virulent anthrax A. P.
60. Bacillus anthracis from the subcutaneous tissue at the seat of inoculation of rabbit. Many chains of the bacilli show the protoplasm degenerated, and only the sheath as a pale, faintly stained thread.
61. Section through the spleen of a guineapig dead of anthrax. × 650. E. C. B.
62. Part of glomerulus of a Malpighian corpuscle in the kidney of rabbit dead of virulent anthrax. Some of the capillary blood-vessels are filled with the bacilli anthracis. × 650 A. P.
63. Section through the liver of a guineapig dead of anthrax. The capillary blood-vessels contain abundantly the bacilli anthracis. × 650 A. P.



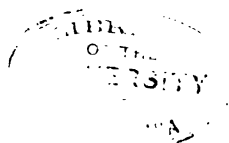
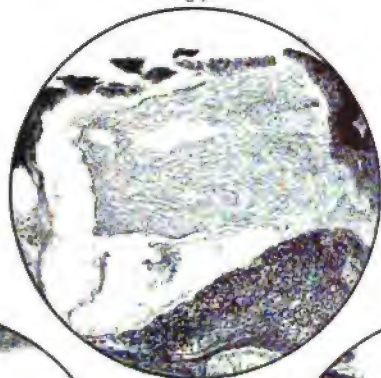


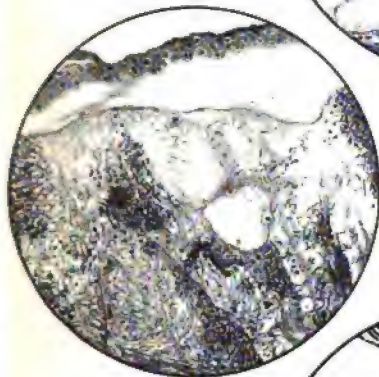
PLATE X

FIG.

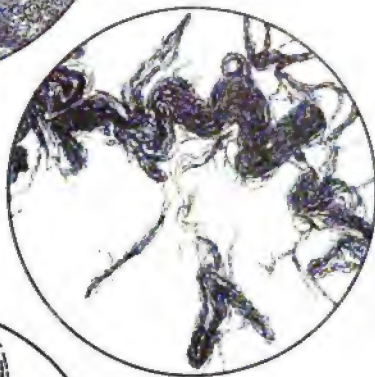
64. From a section through a human malignant pustule. A vesicle is shown between the epidermis and the corium; the vesicle is filled with fibrin; the corium is full of the bacilli anthracis. Low power. E. C. B.
65. From a similar preparation as the preceding figure. The vessels of the corium are filled with the bacilli anthracis. Low power . E. C. B.
66. Impression of a colony of bacilli anthracis on gelatine. The colony is composed of threads of the bacilli. Low power . . . A. P.
67. From a similar preparation as fig. 66, more highly magnified, showing the threads composed of cylindrical bacilli. M. P. 650 . . A. P.
68. From an impression preparation of the margin of a colony of bacillus anthracis growing in a gelatine plate. The bacilli were derived from the blood of frog infected with anthrax during narcosis; the characteristic threads are composed of cylindrical bacilli . E. C. B.
69. Threads of bacilli anthracis with spores in them in the fresh condition. The spores are the oval bright corpuscles contained in the pale threads. Copied from Koch.
70. Threads of anthrax bacilli from a gelatine culture. Many bacilli constituting the threads are swollen up into spherical, oval, or spindle-shaped elements A. P.



65



66



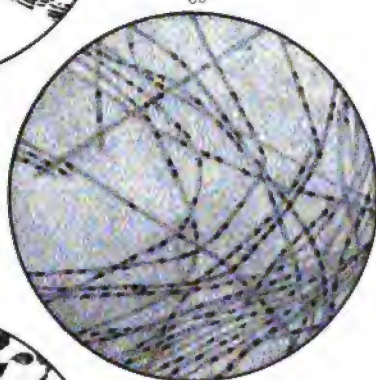
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68



69



70





PLATE XI

FIG.

71. Blood of a guineapig dead after inoculation with symptomatic charbon. Long bacilli (chains) are seen amongst the blood discs . . . A. P.
72. From the false membrane of human diphtheria of the fauces. Numerous diphtheria bacilli are seen amongst the tissue elements . . . A. P.
73. From a similar preparation. Amongst the tissue cells are seen the diphtheria bacilli in almost pure culture A. P.
74. From a colony of an agar culture of the bacillus diphtheriæ, many bacilli single or in chains, some club-shaped A. P.
75. From an agar streak culture of the bacillus diphtheriæ . . . A. P.
76. From a colony on gelatine of the bacillus diphtheriæ. The bacilli are, in many instances, conical A. P.
77. From an agar culture of the bacillus diphtheriæ derived from the necrotic tumour at the seat of inoculation of a cow inoculated with the human bacillus diphtheriæ. The bacilli are slightly smaller than those derived directly from the human A. P.

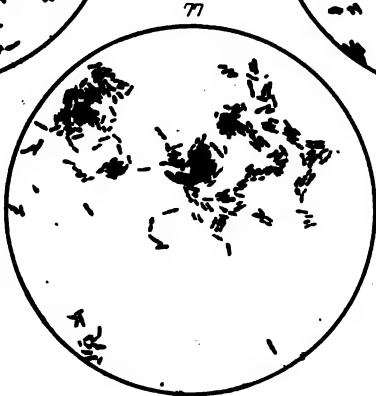
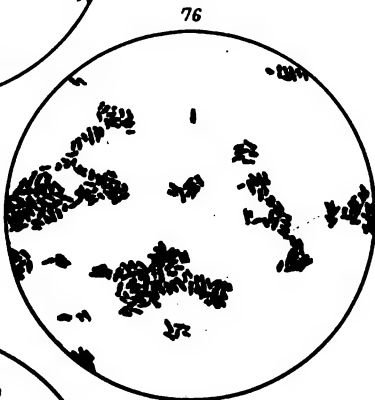
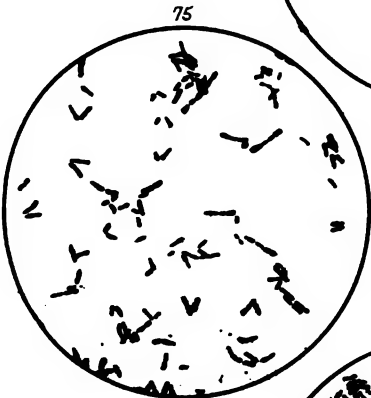
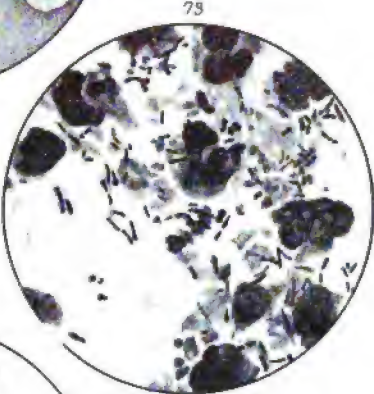
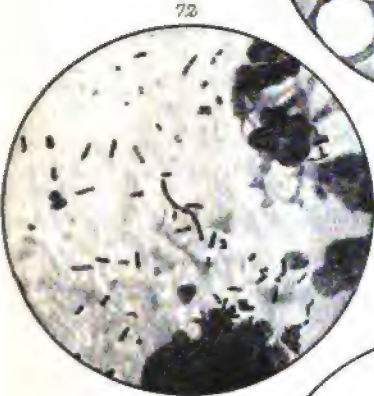




PLATE XII

FIG.

78. From a culture of the bacillus diphtheria derived from the corneal ulcer of cat inoculated on the cornea with diphtheritic membrane. The bacilli are distinctly smaller than those of the cow or human . A. P.

79. From a gelatine culture of the milk of a cow infected by subcutaneous inoculation with the human bacillus diphtheriæ. The characteristic club-shaped bacilli are numerous A. P.

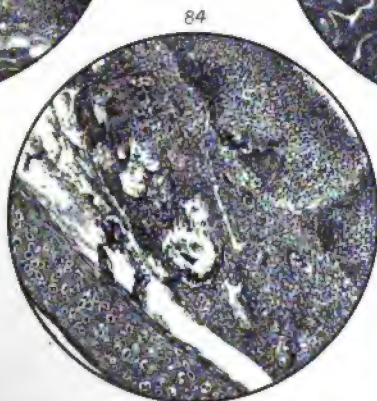
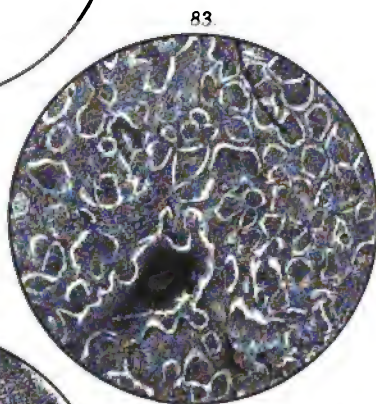
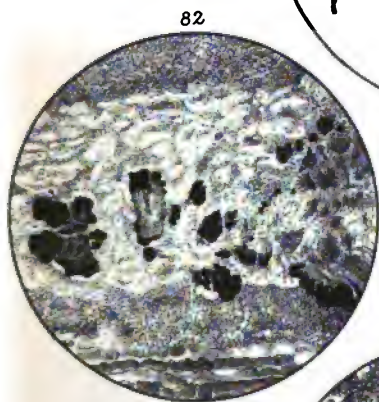
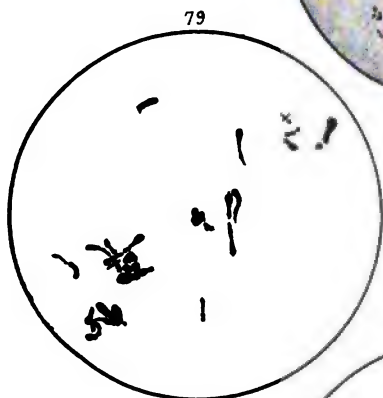
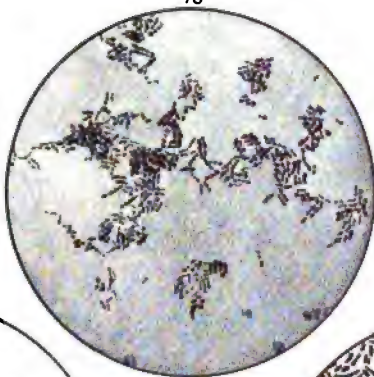
80. From a culture of the pseudo-diphtheria bacillus of a case of human diphtheria A. P.

81. From a culture of a different kind of pseudo-diphtheria bacillus obtained from the milk of a cow. All bacilli are club-shaped or dumb-bell-shaped E. C. B.

82. From a section through the necrotic part of the local tumour produced in the cow by subcutaneous inoculation of the human diphtheria bacillus. The dark masses are masses of the diphtheria bacillus located in a reticulated necrotic tissue. Low power . . . A. P.

83. From a section through a pneumonic patch of the lung of cat dead of natural diphtheria. The infundibula and air cells are filled with necrotic masses in which nuclei of leucocytes are still to be recognised. Low power A. P.

84. From a section through the lower part of the larynx of a cat dead of diphtheria, contracted naturally by ingestion of the milk of an infected cow: on the left is the cartilage, on the right the necrotic mucosa which formed a complete grey false membrane in the larynx and upper part of the trachea. Low power A. P.



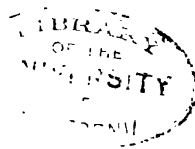
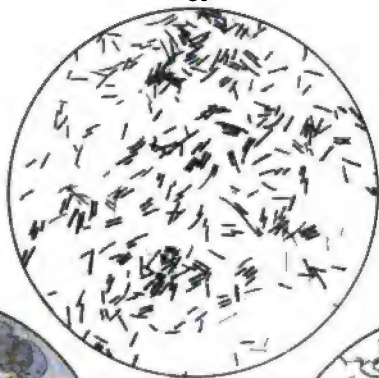


PLATE XIII

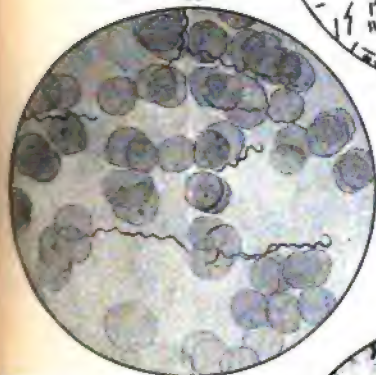
FIG.

85. Cultivation on gelatine of the typhoid bacillus A. P.
86. Blood in relapsing fever, showing the spirilla of Obermeyer, copied from Koch.
87. From mucus flakes of the ileum of a case of cholera asiatica, showing the comma bacilli of Koch in almost pure culture. Magnifying power, 1,000 A. P.
88. Mucus flakes of a case of Asiatic cholera. The comma bacilli in this case are several times the size of those shown in previous figure. One spirillum is here shown. Magnifying power, 1,000 A. P.
89. Culture in gelatine of the choleraic comma bacilli A. P.
90. Culture on agar of the choleraic comma bacilli; spirilla are shown . A. P.
91. Cultivation on gelatine of choleraic comma bacilli after several sub-cultures. The bacilli are very polymorphous, some quite spherical . . . A. P.

85



86



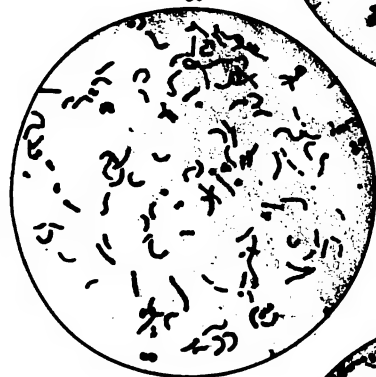
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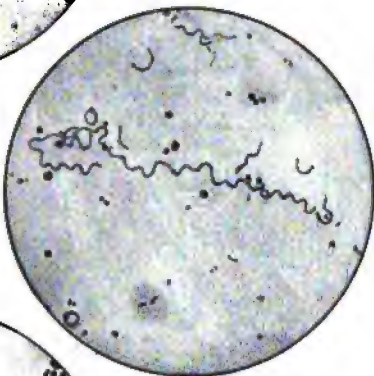
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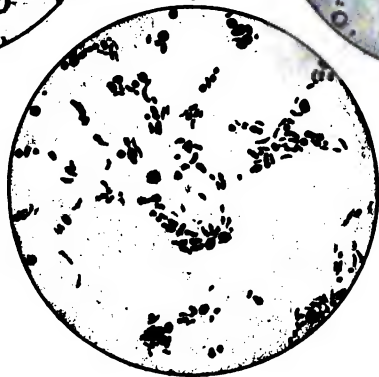




PLATE XIV

FIG.

92. From a similar preparation as in fig. 91, but after more prolonged sub-cultures. The organisms are scarcely to be recognised as being related to the comma bacilli; some are much swollen up and full of vacuoles A. P.

93. Mucus flakes of an ape dead with symptoms of diarrhoea; comma bacilli in almost pure culture A. P.

94. Mucus from a loop of the intestine of an ape, after ligaturing the loop and injecting into its cavity sulphate of soda solution. Numerous minute comma bacilli and spirilla are seen A. P.

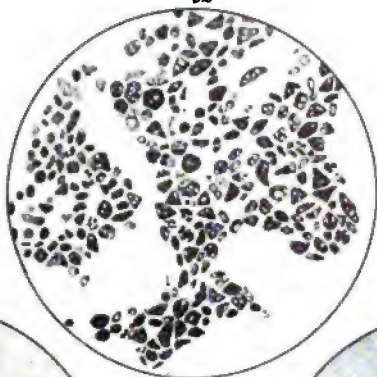
95. Blood of a case of influenza. Numerous minute bacilli seen amongst the blood discs E. C. B.

96. Specimen of pus of gonorrhoea. Two pus corpuscles are shown which contain in their interior numerous gonococci E. C. B.

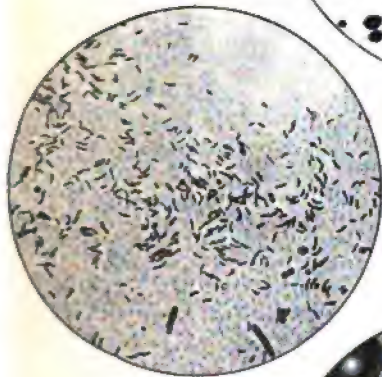
97. Pus of catarrhal ophthalmia or Egyptian ophthalmia (Koch). Numerous minute bacilli are seen free and within the substance of the pus corpuscles E. C. B.

98. From the same specimen as fig. 97. A large epithelial scale is shown, the surface of which is covered with the small bacilli E. C. B.

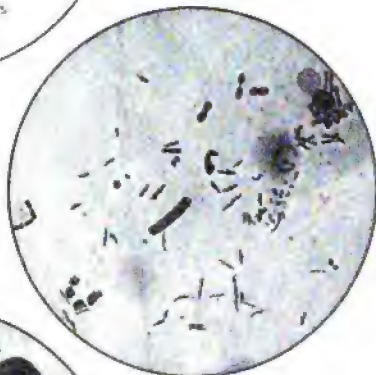
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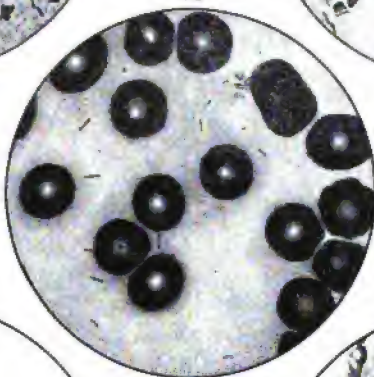
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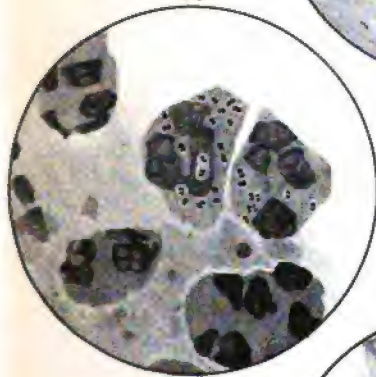
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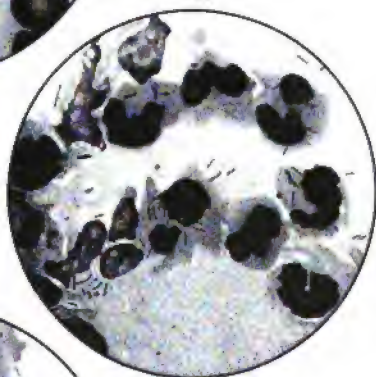
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98

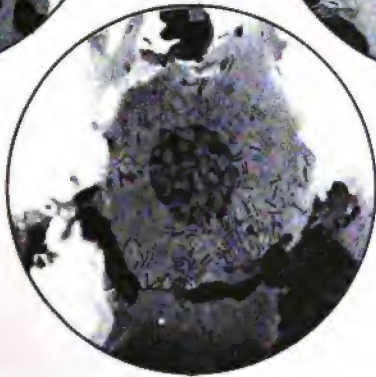
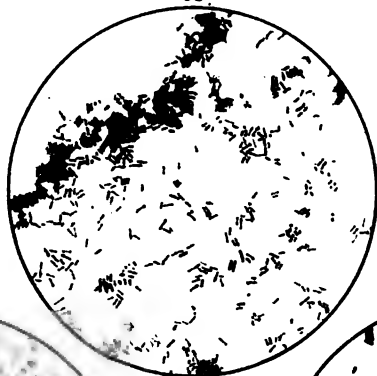


PLATE XV

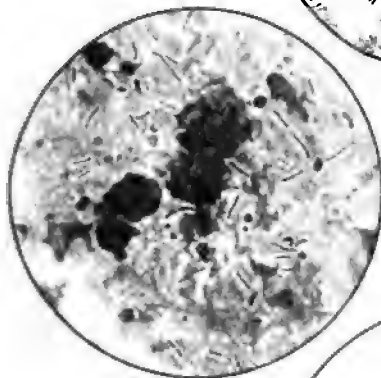
FIG.

99. Culture of minute bacilli, isolated from a beef pie that had caused choleraic diarrhoea at Portsmouth E. C. B.
100. Cover-glass specimen of lung juice of a nodule of the lung in glanders of the horse. The bacilli of glanders are shown very numerous.
A. P.
101. Cultivation on agar of the glanders bacillus A. P.
102. Cultivation of the tetanus bacillus. Some of the bacilli show the spore formation at one pole. These bacilli look like drumsticks . . A. P.
108. Cover-glass specimen of pulmonary sputum in tuberculosis. The tubercle bacilli are very numerous present, few nuclei of pus cells . A. P.
104. From a culture on glycerine agar of the tubercle bacilli . . A. P.
105. Purulent matter of a human cerebral abscess, due to tubercle. Two leucocytes are shown, containing the tubercle bacilli . . A. P.

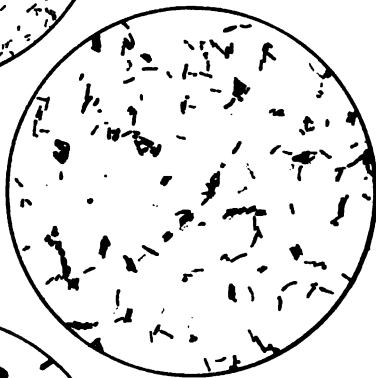
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100



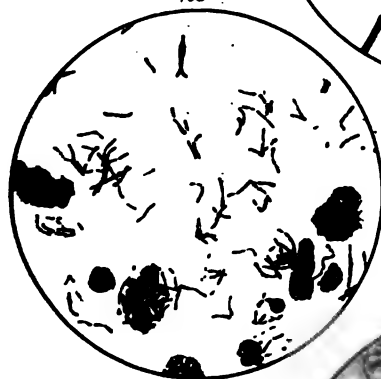
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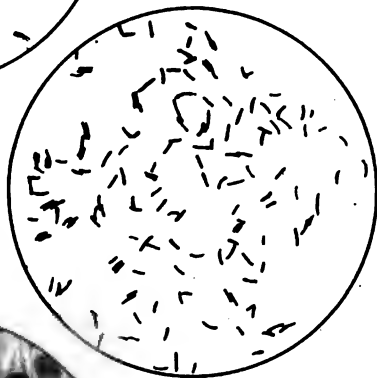
102



103



104



105





PLATE XVI

FIG.

106. Section through a tubercle of the human lung. Low power . A. P.

107. The centre of a caseous tubercle in the human lung (same specimen as shown in previous figure), numerous tubercle bacilli in the central part. Magnifying power, 650 A. P.

108. From a section through a pulmonary tubercle in the cow ; a giant cell full of tubercle bacilli, also other isolated tubercle bacilli . . A. P.

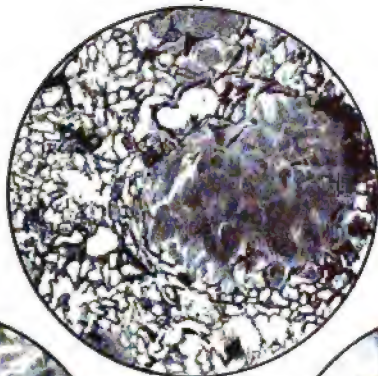
109. A grey tubercle in the lung of a rabbit developed in a few weeks after injection of culture of tubercle bacilli into the vein of the ear. The centre of the tubercle is surrounded by leucocytes containing the tubercle bacilli A. P.

110. From a tubercle in the liver of a rabbit infected by intravenous injection of culture of tubercle bacilli. Two giant cells full of tubercle bacilli are shown E. C. B.

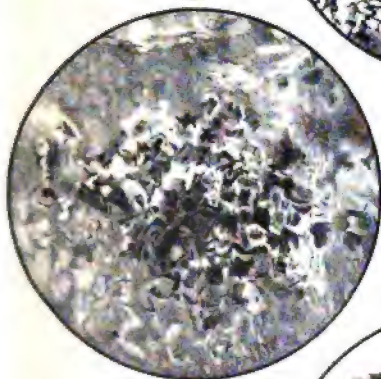
111. From a section through caseous tubercle of the spleen of a fowl. Groups of tubercle bacilli in the necrotic tissue . . . A. P.

112. One large giant cell of a tubercle in the lung of a cow. Numerous tubercle bacilli are shown in the substance of the giant cell . A. P.

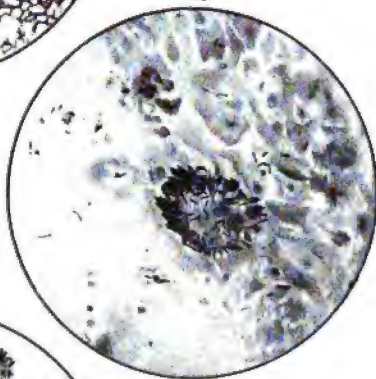
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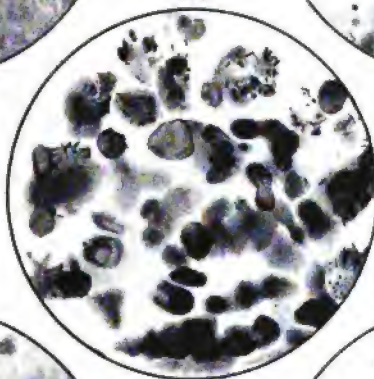
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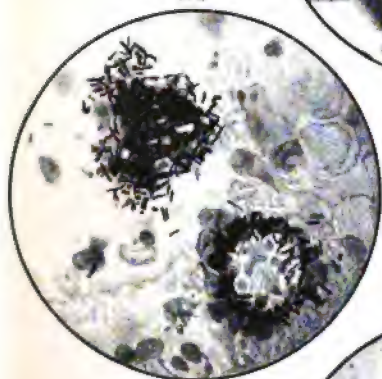
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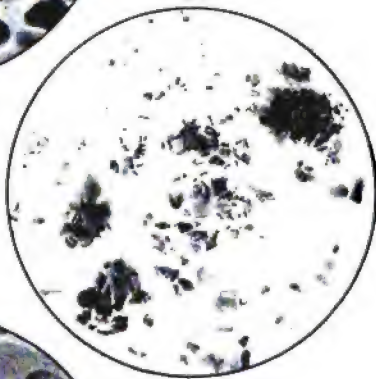
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110



111



112

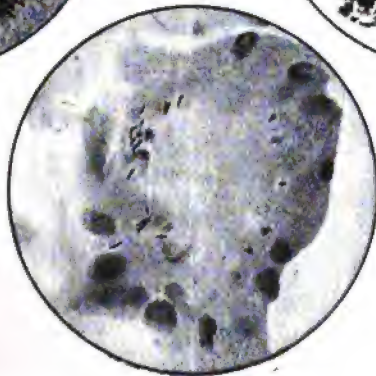
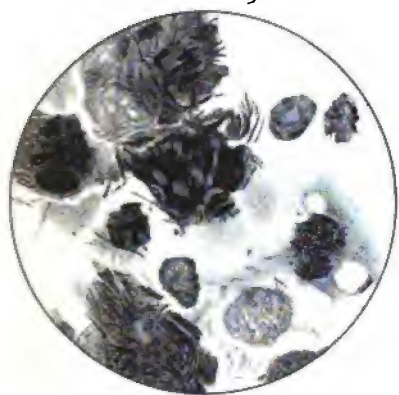




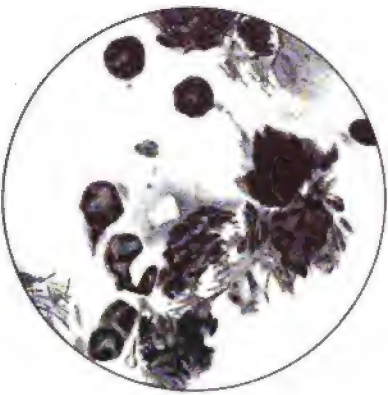
PLATE XVII

FIG.

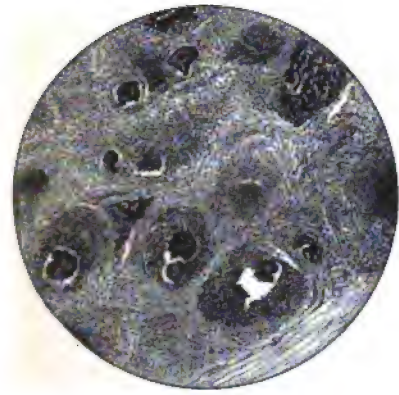
118. Large cells of a leprous nodule, containing an abundance of the lepra bacilli, free and particularly filling the substance of the cells . A. P.
114. A similar preparation as in previous figure A. P.
115. Section through the tongue of ox. Numerous round-cell nodules, the centre of which is occupied by a mass of actinomyces. Low power. A. P.
116. From a similar preparation, more highly magnified, showing the actinomyces mass. The 'rays' are distinct A. P.
117. A similar preparation, highly magnified; the 'clubs' of the rays are well shown A. P.
118. Part of the mycelium of actinomyces, from a culture on gelatine of the actinomyces. Branched threads are shown, which threads are either homogeneous, or they show granules, or they are made up of rods and cylinders E. C. B.



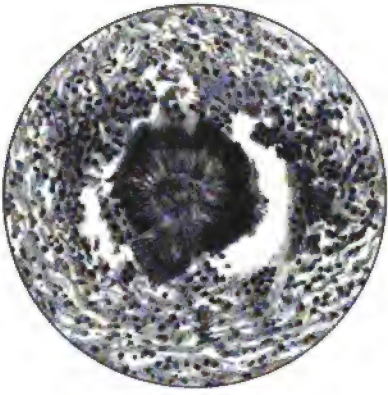
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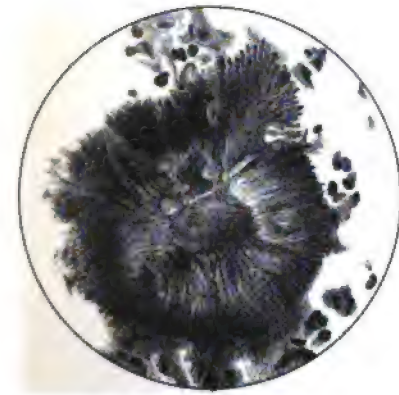
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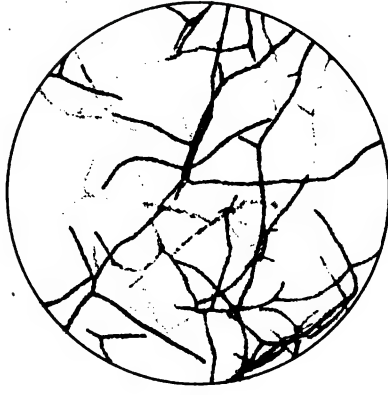
115



116



117



118



PLATES XVIII-XXV inclusive (figures 119 to 152) are from drawings made by Mr. M. H. LAPIDGE.

PLATE XVIII

FIG.

119. *Bacillus filamentosus* with spores. The bacillary threads are stained in methyl blue, the spores in fuchsin. Magnifying power, 750.
120. *Bacillus anthracis* from a potato culture. The bacilli are stained in methyl blue, the spores in fuchsin. Magnifying power, 350.
121. Pus from gonorrhoeal discharge, showing pus cells, some containing in their interior the gonococci stained with gentian violet. Magnifying power, 700.
122. Two pus cells of gonorrhoeal discharge, containing numerously the gonococci as diplococci. Same specimen as fig. 121. Magnifying power, 1,400.
123. Purulent conjunctival discharge of true Egyptian ophthalmia of Koch. Pus cells indicated by their nuclei, containing at *a* fine bacilli; at *b* masses of free bacilli. Methyl blue staining. Magnifying power, about 1,000.
124. Section through the valve in ulcerative endocarditis. The section was stained in methyl blue. The blue masses are masses of streptococci. Magnifying power, 100.
125. Section through the valve in another case of ulcerative endocarditis, stained with methyl blue and eosin. The blue masses are masses of staphylococcus aureus; *a* is the free edge of the villous valve. Magnifying power, 100.

120



121



122



119



b

123



124

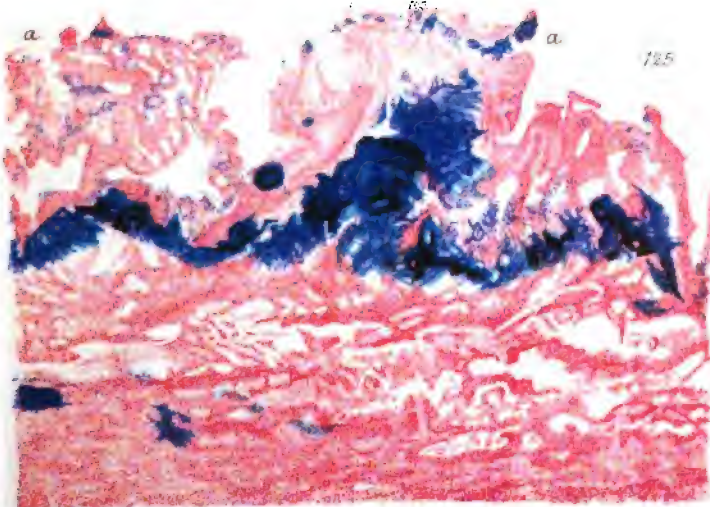


PLATE XIX¹

Figs.

126. From a section through the medulla of the kidney of a rabbit after injection into the ear vein of a culture of the staphylococcus aureus of the above case of ulcerative endocarditis, stained in methyl blue and eosin. The blue masses are masses of staphylococcus aureus filling blood-vessels; the red ground are uriniferous tubules. Magnifying power, 100.

Figs. 124, 125, and 126 are from preparations by Dr. F. H. Andrewes.

138. From a section through the mucous membrane of the pharynx of a child dead of acute diphtheria, stained in gentian violet: (a) surface of the false membrane filled with the diphtheria bacilli; (b) the mucous membrane filled with extravasated blood. Magnifying power, about 800.

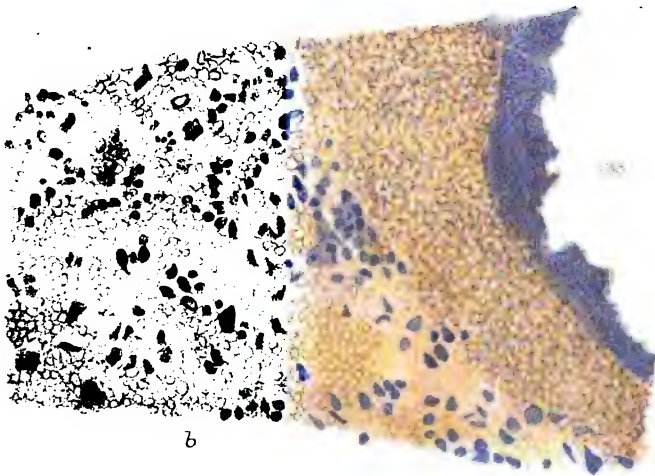
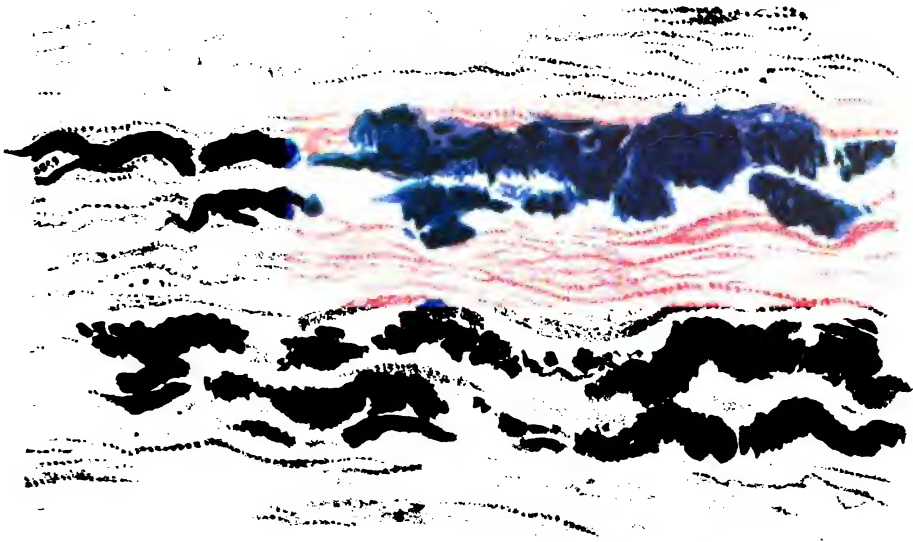
137. The kidney of a cat that died after ten to twelve days' illness of diphtheria naturally acquired. The enlargement and fatty white cortex are conspicuous. Natural size.

138. The kidney of a cat that died after five to six days' illness, resulting from inoculation of the diphtheria bacilli. Natural size.

138, 137, and 138 are copied from Klein's 'Etiology of Diphtheria' in the Reports of the Medical Officer of the Local Government Board for 1889.

¹ The making up of this and some of the subsequent plates necessitated a slight derangement in the numbers of the figures.

126



a

b

137



138



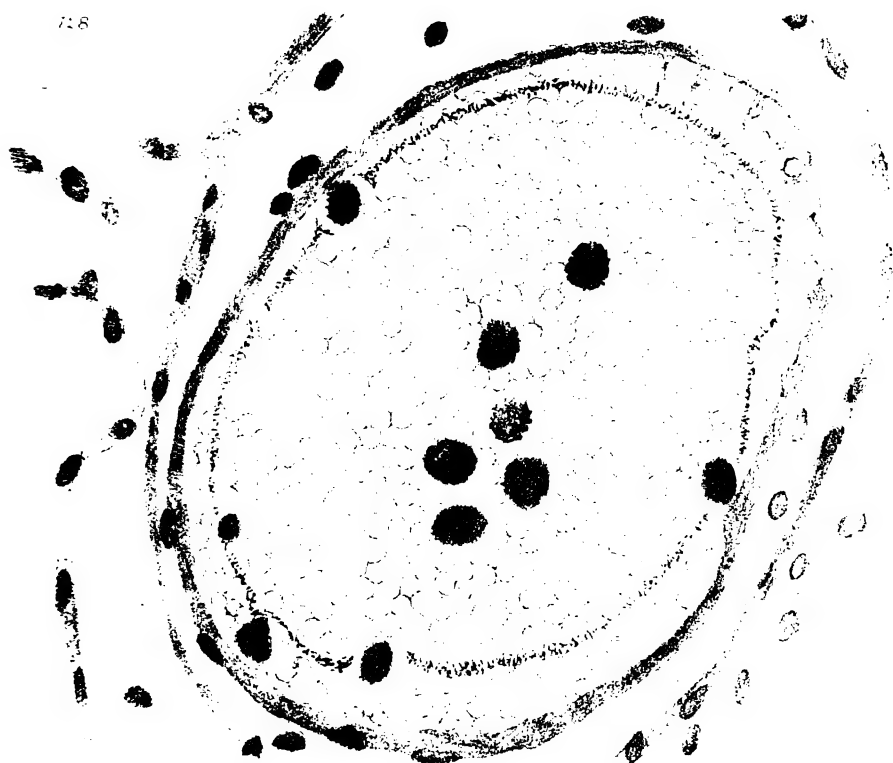


PLATE XX

FIG.

128. From a section through the lung of a mouse dead of Koch's acute mouse septicæmia. Stained with gentian violet. A large blood-vessel is here represented, distended by and filled with blood, the white blood cells containing in their cell substance numerous the fine bacilli. M. P. 700.
181. Section through the cortex of the kidney of a rabbit dead of virulent anthrax, stained with Spiller's purple: (a) Malpighian corpuscles, the capillaries of which are distended by and filled with the bacilli anthracis; (b) capillary blood-vessels filled with the bacilli. M. P. about 800.

728



731

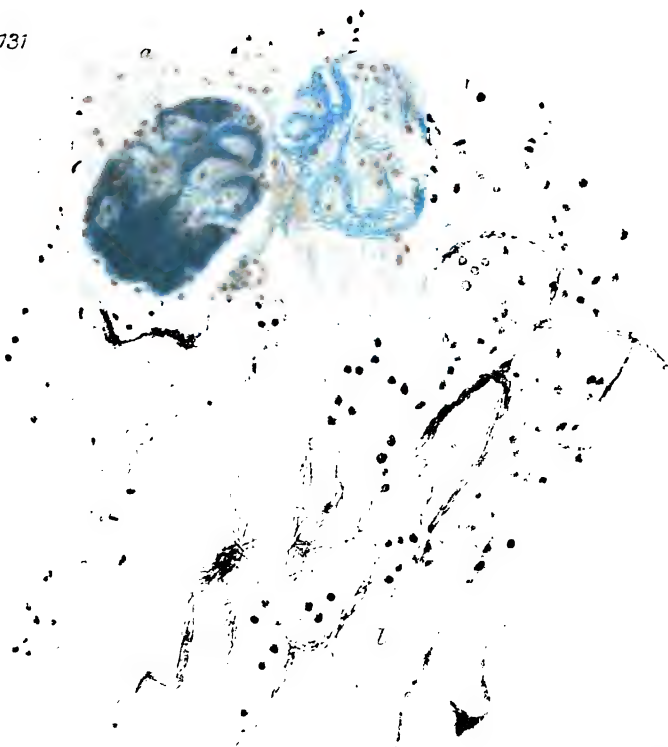
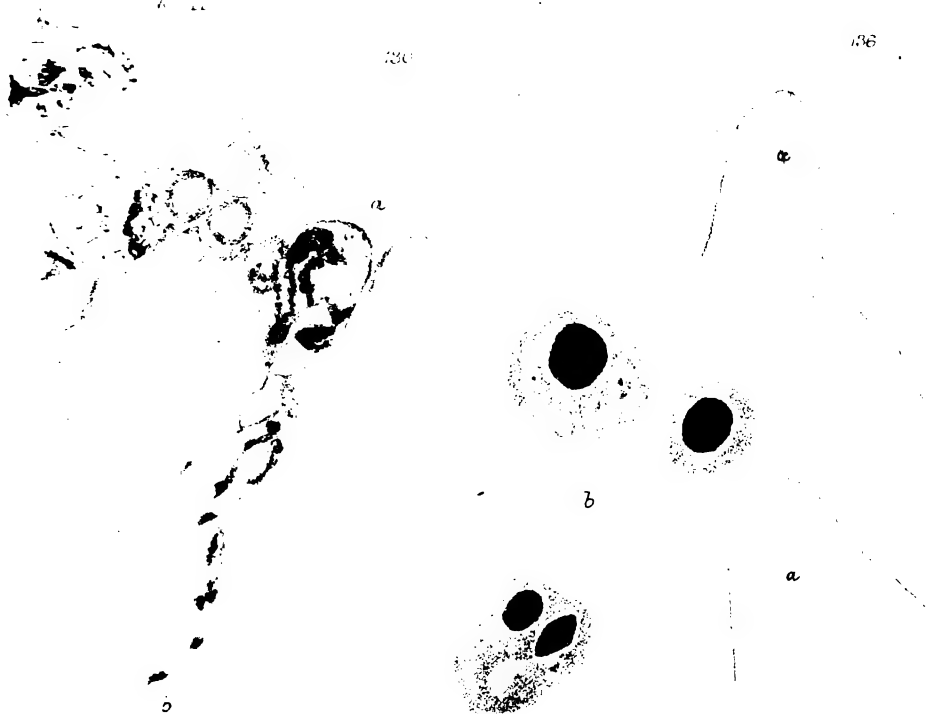




PLATE XXI

FIG.

180. From a section through the kidney of a mouse dead after inoculation with culture of the bacillus of swine fever, stained with methyl blue :
(a) A Malpighian corpuscle, its capillary blood-vessels filled with the bacilli ; (b) capillary blood-vessels filled with the bacilli. M. P. 175.
186. The eruption on the sixth day on the udder of a cow subcutaneously inoculated into the shoulder with a culture of the bacillus diphtheriæ :
(a) The teats ; (b) the eruption : some are still in the stage of vesicles, others are already covered with black crusts. Natural size.
129. From a section through the lung tissue of a mouse dead of Koch's acute mouse septicæmia. In the tissue separating the lung alveoli leucocytes are seen, whose protoplasm includes numerous the characteristic minute bacilli, stained with gentian violet. M. P. about 1000.
127. Section through a necrotic patch in the liver of a mouse dead after septicæmia : (a) Capillary blood-vessels distended, and containing besides blood-corpuscles masses of minute bacilli stained blue ; (b) the strands of necrotic liver cells. The section had been stained in methyl blue and eosin. M. P. about 800.



129

9

127



a

West, Newman chr. lith.

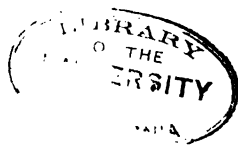
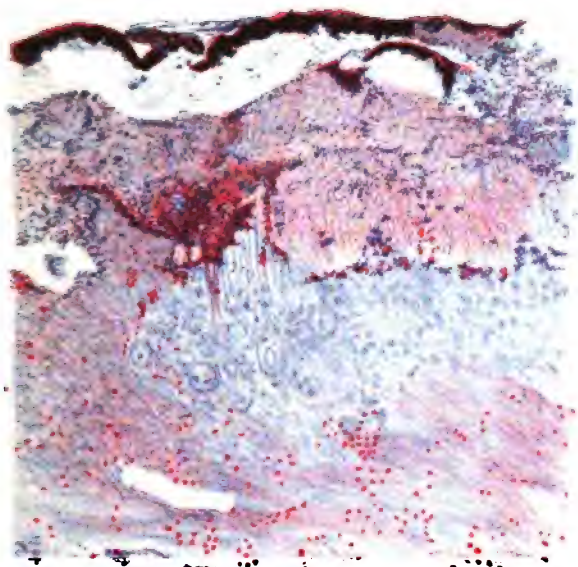


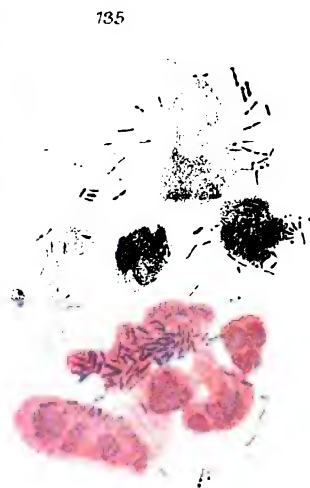
PLATE XXII

FIG.

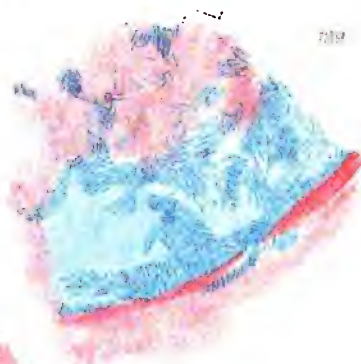
182. From a section through the mucous membrane of the larynx of a child dead of diphtheritic croup. The mucous membrane is transformed into a typical diphtheritic membrane, in the superficial layers of which are large numbers of the diphtheria bacilli, stained blue. On the free surface of the membrane they form an almost continuous mass. The section was stained in rubin and then in methyl blue. M. P. 100.
185. Cover-glass specimen of the superficial parts of the diphtheritic membrane, showing cells and numerous diphtheria bacilli, stained blue, free and enclosed within the cells. The preparation was stained in rubin and then in methyl blue. M. P. about 700.
184. Section through the diphtheritic membrane from the tonsil of a child. The section shows in the superficial layers of the membrane (lower part of figure) almost continuous loculi, large and small, filled with clumps of the diphtheria bacilli, stained blue; in the middle portions are also numerous smaller masses of the bacilli. The section had been stained with rubin and then with methyl blue. M. P. about 100.
189. From a section through a necrotic nodule of the lung in a horse dead of glanders. Numerous bacilli of glanders, stained blue, are seen. The section had been stained in rubin and then in methyl blue. M. P. about 1,000.



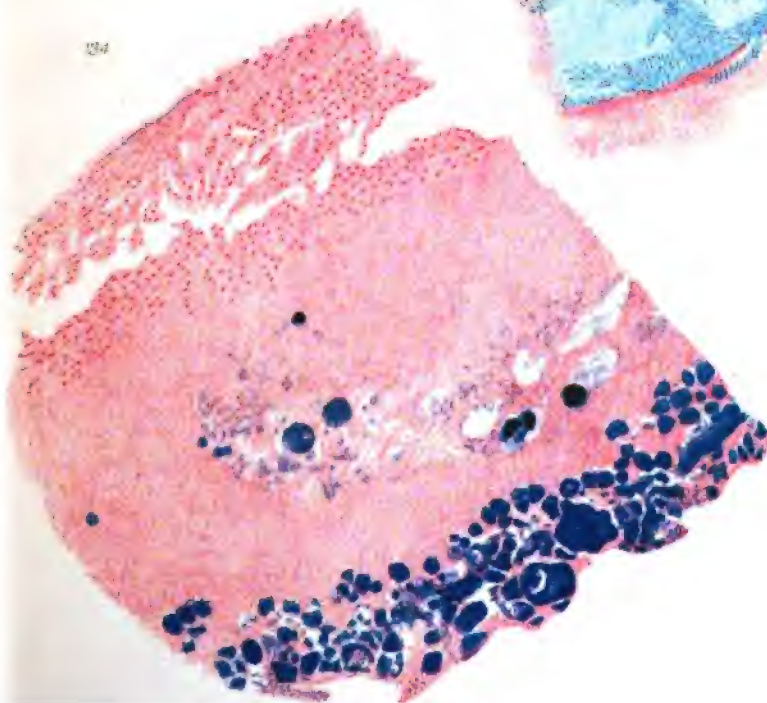
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735



734



732

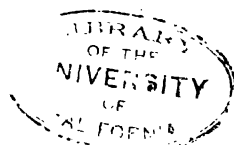


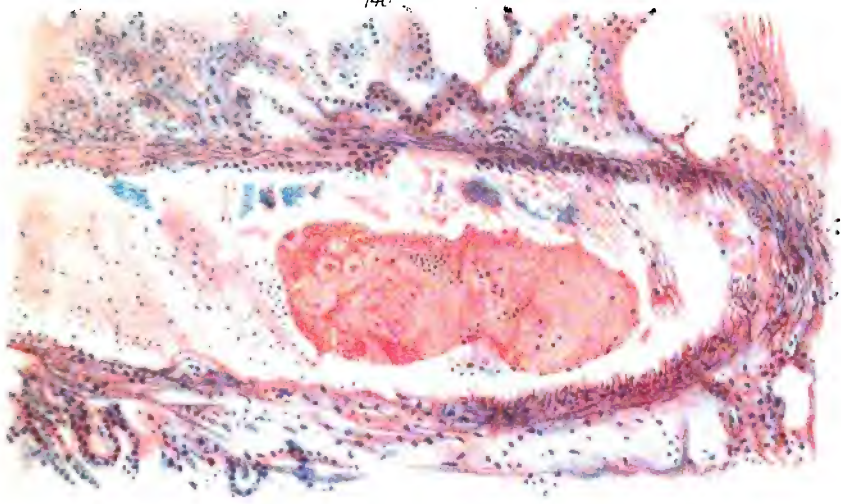
PLATE XXIII

FIG.

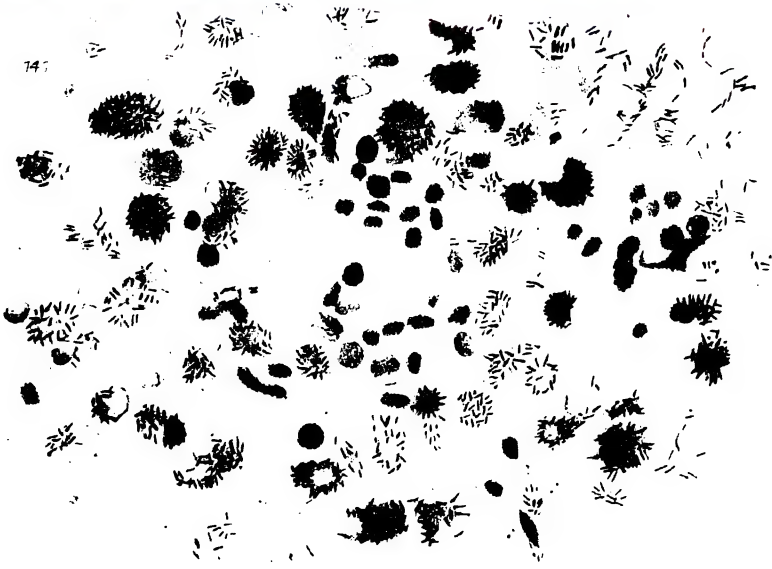
140. From a section through the glanders lung of horse. The section represents a large lymphatic vessel containing a coagulum. In the upper part of the contents of the vessel are several blue clumps of the glanders bacilli; stained with rubin and then methyl blue. M. P. 100.
141. Section through a young tubercle in the lung of rabbit; numbers of cells are seen filled with the tubercle bacilli—carbol fuchsin, nitric acid, and then methyl blue. M. P. about 1,000.
142. From a section through a tubercle in the spleen of a fowl artificially infected with human tubercle. The central part is a caseous mass containing abundantly the tubercle bacilli—carbol fuchsin, nitric acid, and then methyl blue. M. P. 800.
143. From a similar tubercle as in fig. 142, but more highly magnified. M. P. 1,000.

140

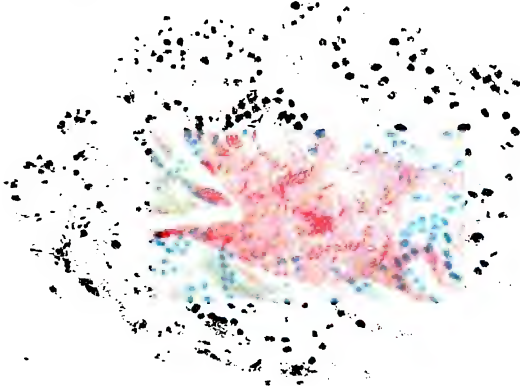
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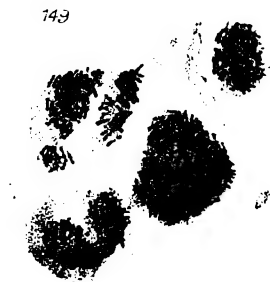
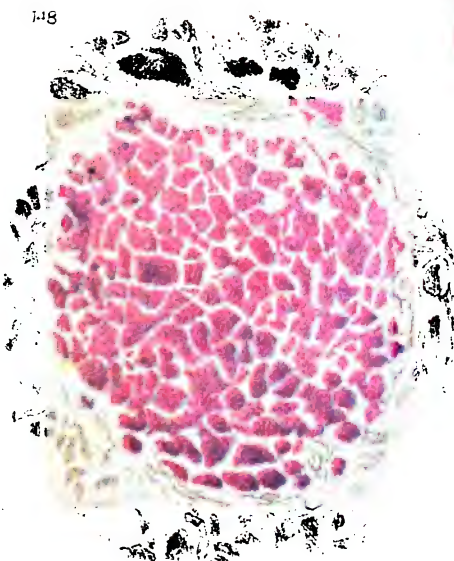
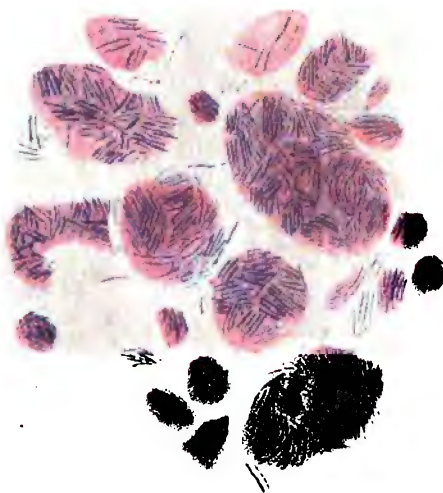
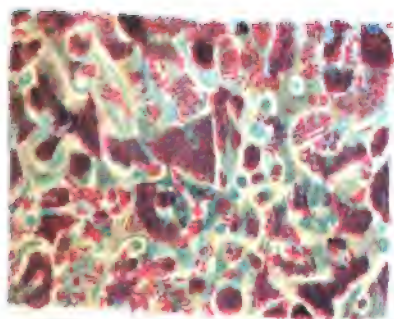
143



PLATE XXIV

FIG.

144. A giant cell in a tubercle in the lung of a cow—carbol fuchsin, nitric acid, and then methyl blue. The tubercle bacilli form a characteristic peripheral annular zone. Magnifying power, 1,000.
145. Section through a cutaneous leprosy nodule—carbol fuchsin, nitric acid, and then methyl blue. (a) The stratum corneum of the epidermis; (b) the stratum Malpighii; (c) the superficial layer of the cutis; (d) the upper part of the leprosy nodule. In this, numbers of lepra cells filled with the lepra bacilli. Also in the layers above there are a few lepra bacilli seen. Magnifying power, 800.
146. Section showing the middle part of a cutaneous leprosy nodule, same as in fig. 145. The tissue of the nodule contains cells of different sizes, the substance of all of them filled with the lepra bacilli. Magnifying power, 800.
147. Cover-glass specimen of the purulent secretion of an ulcerated leprosy nodule, showing the leprosy cells filled with bundles of the lepra bacilli. Magnifying power, 1,000.
148. From a section through the liver of a Rhea. The liver was crowded with white nodules. Such a nodule is here shown as composed of densely aggregated cells, each filled with extremely minute bacilli, stained red with fuchsin. Fuchsin and nitric acid. Magnifying power, 800.
149. Several of the cells of fig. 148 shown, more highly magnified. Magnifying power, 1,000.



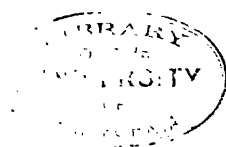
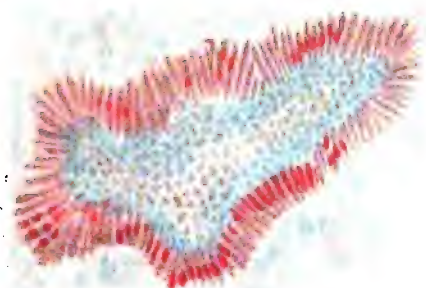


PLATE XXV

FIG.

150. From a section through an actinomyces nodule in the tongue of ox. The centre shows the actinomyces with its radiating clubs. Rubin and then methyl blue. Magnifying power, 500.
151. From the same specimen, showing in surface view part of an actinomyces clump. The red dots are the 'clubs' seen in bird's-eye view. Magnifying power, 500.
152. A Malpighian corpuscle of the kidney of a person dead after meat poisoning at Welbeck. Some of the capillaries of the glomerulus are plugged with masses of bacilli, the remainder of the glomerulus swollen, and showing hyaline degeneration. Gentian violet staining. Magnifying power, 500.

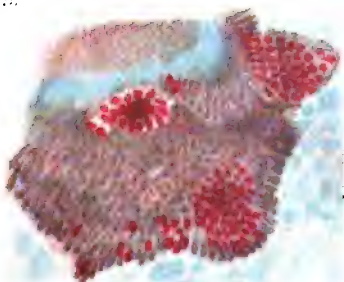
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126





PLATES XXVI-XXX inclusive. Figs. 1 to 46 are from photograms made by Mr. E. C. BOUSFIELD, except figs. 35, 37, and 38. All of natural size.

PLATE XXVI

FIG.

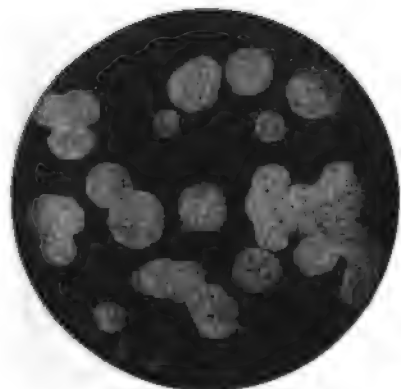
1. Plate cultivation of air of Wandsworth Common.
2. Plate cultivation of air of Oxford Street. Both plates are on gelatine, which after it was set was exposed for three minutes to the air, the difference between the time of exposure at Wandsworth Common and in Oxford Street being about one hour. Date, March 1891. The difference in the number of colonies, that is, in the number of micro-organisms, in the two localities is striking.
3. Plate cultivation in gelatine of Finkler's comma bacilli; some of the colonies isolated, others confluent, all liquefying the gelatine.
4. Plate cultivation in gelatine of the aerobic bacillus of malignant oedema. The small dots are colonies in the depth, the large patches are the colonies growing on the surface.
5. Plate cultivation in gelatine of a quarter of a cubic centimetre of the ordinary standpipe water of the West Middlesex Water Company; three colonies per $\frac{1}{4}$ c.c., that is, 12 per 1 c.c.
6. Plate cultivation in same manner of water of Grand Junction Water Company, about eight colonies per $\frac{1}{4}$ c.c.



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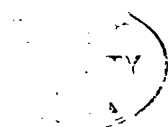
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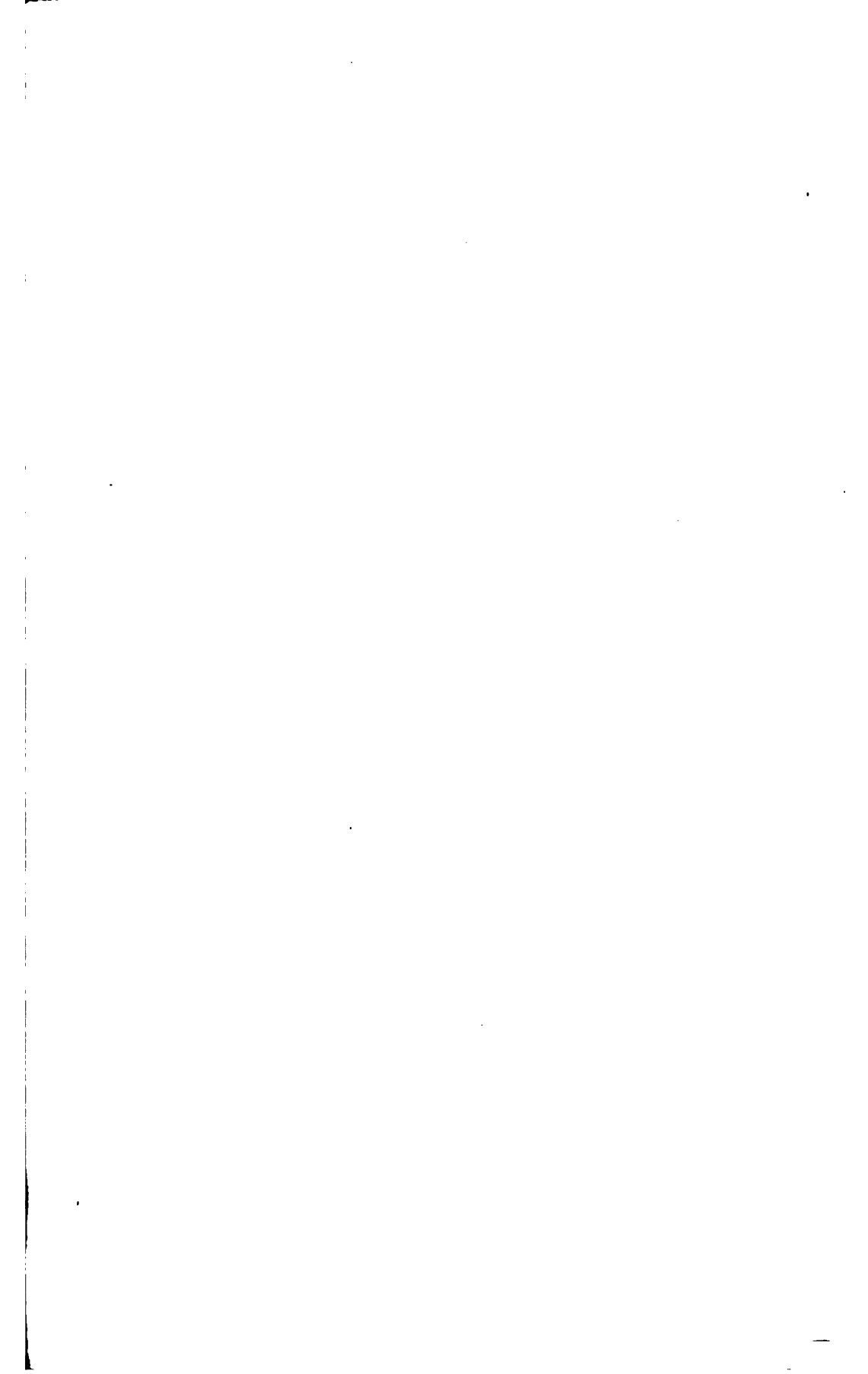


PLATE XXVII

Fig.

7. Streak culture on the surface of slanting gelatine of fowl cholera.
8. Ditto of fowl enteritis.
9. Ditto of the typhoid bacillus.
10. Ditto of the bacillus of Middlesbrough pneumonia.
11. Ditto of the bacillus of grouse disease.
(All these five cultures are of the same age.)
12. Streak culture, on the surface of slanting gelatine, of the streptococcus of the Edinburgh disease.
13. Ditto of streptococcus scarlatinæ.
14. Ditto of streptococcus pyogenes.
15. Ditto of streptococcus erysipelas. This last photo does not correctly represent the actual appearances, being too much of a homogeneous character.
(All these four streptococcus cultures of same age.)
16. Streak culture on gelatine of the pie bacillus of Portsmouth pie poisoning, two days old.
17. Ditto six days old.
18. Broth culture of staphylococcus aureus, the broth uniformly turbid after twenty-four hours at 37° C.
19. Broth culture of the bacillus of Middlesbrough pneumonia, after twenty-four hours.
20. Stab culture in gelatine of bacillus fluorescens liquefascens. The upper part of the gelatine liquefied by the growth.



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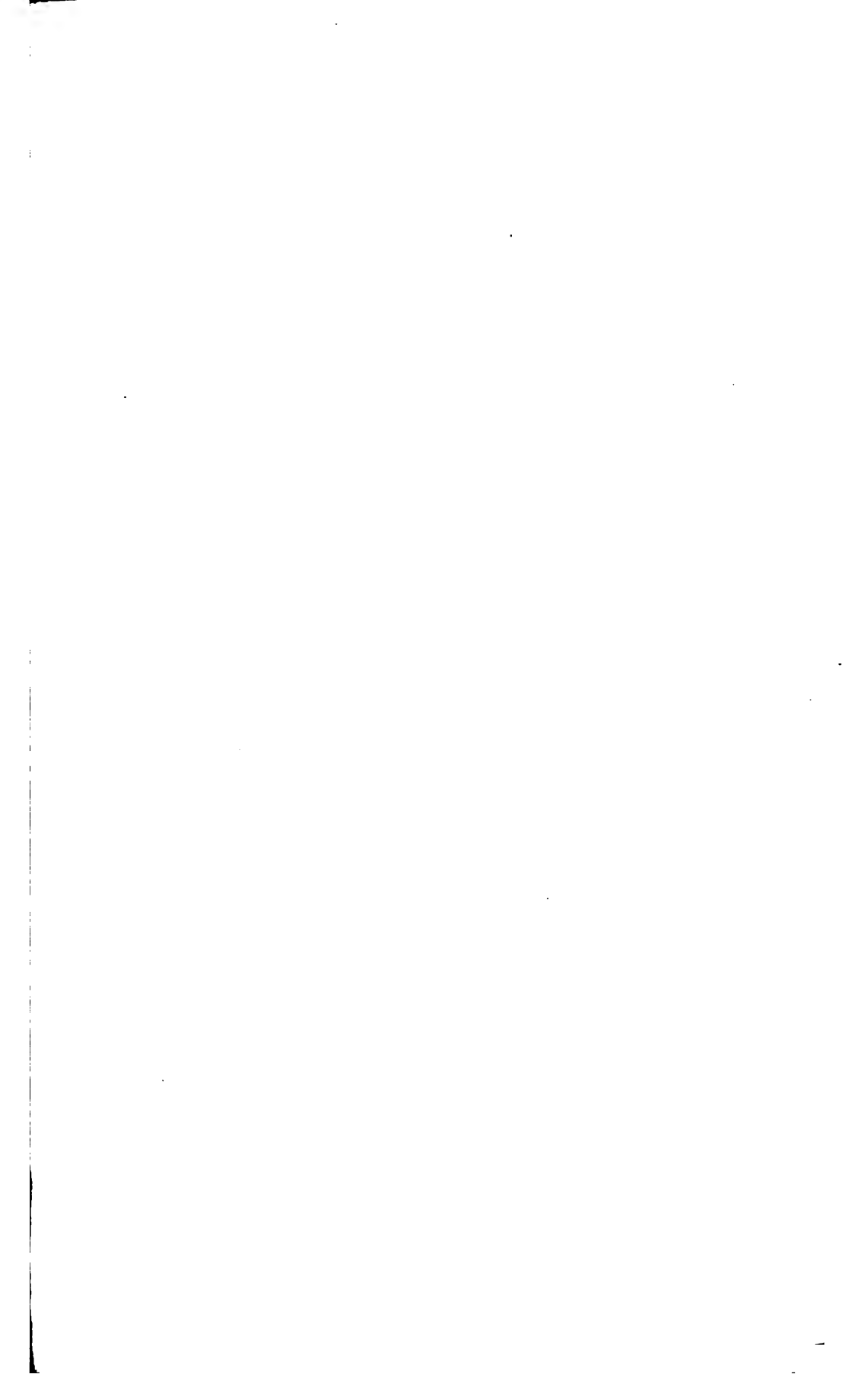


PLATE XXVIII

FIG.

21. Stab culture in gelatine of the bacillus of swine erysipelas.
22. Stab culture in gelatine of bacillus anthracis. The upper part of the growth has commenced to liquefy the gelatine.
23. Stab culture in gelatine of the aërobic bacillus of malignant oedema. A number of flat gas bubbles are attached to the streak growth.
24. Shake culture in gelatine of the aërobic bacillus of malignant oedema. Numerous gas bubbles in connection with the dot-like colonies.
- 25 and 26. Shake cultures in gelatine of the aërobic bacillus of malignant oedema from the blood of rabbit and mouse respectively, dead after inoculation.
27. Streak cultures on the slanting surface of gelatine of the bacillus diphtheriæ of the human subject.
28. The same bacillus forming isolated colonies on the slanting surface of gelatine—tube plate cultivation.
29. The same bacillus forming colonies on the surface of gelatine, after two weeks' incubation.
30. A pseudo-diphtheria bacillus, derived from cow's milk, forming colonies on the slanting surface of agar.
31. Streak culture on agar of the same pseudo-diphtheria bacillus as in fig. 30.
32. Stab culture in gelatine of the bacillus of Portsmouth pie, the same as in 16 and 17 of Plate XXVII.



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PLATE XXIX

FIG.

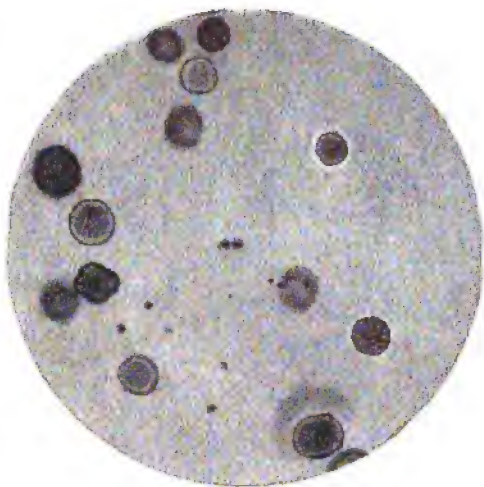
- 88.** Stab culture in gelatine of the bacillus diphtheriæ derived from the (human) diphtheritic membrane. The culture is several weeks old.
- 84.** Similar culture of the bacillus diphtheriæ derived from the diphtheritic necrotic tumour of the cow, artificially produced by subcutaneous inoculation.
- 85.** Plate cultivation in gelatine of Koch's cholera spirilla, seventy-two hours old. Magnified fifty times. Copied from Plagge.
- 86.** Cultivation of Koch's anaërobic bacillus of malignant œdema in grape sugar gelatine. The growth (in the depth) has liquefied the gelatine. Numerous gas bubbles are noticed.
- 87.** Cultivation of the tetanus bacillus in the depth of grape sugar gelatine, six days old. Copied from Fränkel and Pfeiffer's Atlas, Plate XXVII, fig. 55.
- 88.** Cultivation of the bacillus of symptomatic charbon in the depth of grape sugar gelatine, four days old. Copied from Fränkel and Pfeiffer's Atlas, Plate XXVIII, fig. 57.



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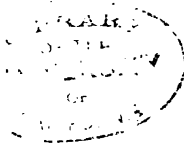
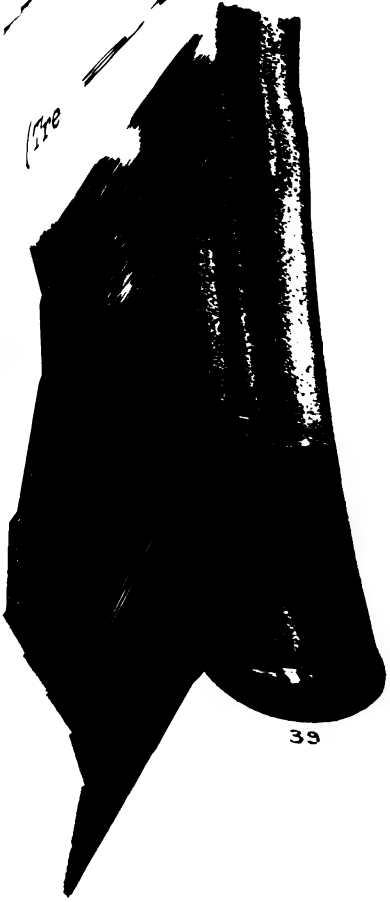


PLATE XXX

FIGS.

39-46. Cultivations (stab) in gelatine of different varieties of choleraic comma bacilli. All the cultures have been incubated for nine days at 20° C. The amount of growth and the amount of liquefaction by them of the gelatine at the upper part of the stab differ in the different species.



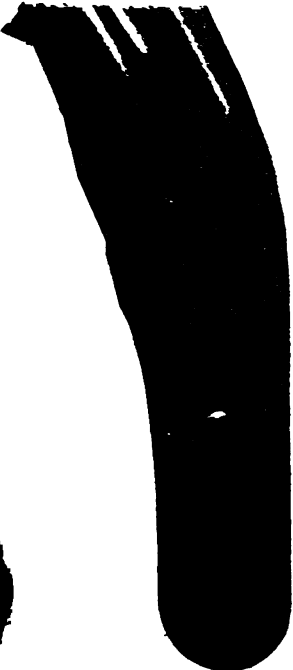
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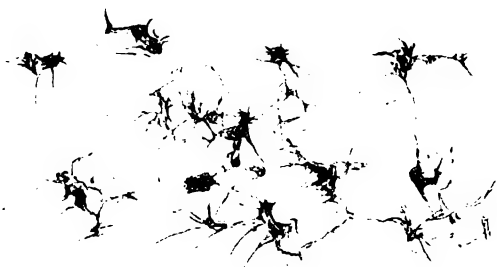
PLATES XXXI-XXXIX inclusive are from drawings by Mr. M. H. LAPIDGE.

PLATE XXXI

FIG.

47. Plate cultivation in gelatine of bacillus anthracis, after three days' incubation at 20° C. The colonies are seen in transmitted light as angular masses, numerous bundles of threads passing between them. Magnifying power, 10.
49. Plate cultivation of the bacillus of swine fever, seen in transmitted light, after twelve days' incubation at 20° C. Each colony is marked as a circular or slightly irregular patch with clear centre. Magnifying power, 10.
50. Colonies of the glanders bacilli on the surface of agar, seen in transmitted light after nine days' incubation at 37° C. Each colony is a flat disc, dark granular in the centre. Magnifying power, 10.
51. Colonies of the bacillus of Middlesbrough pneumonia, on the surface of gelatine, seen in transmitted light, after forty-eight hours' incubation at 20° C. Magnifying power, 10.

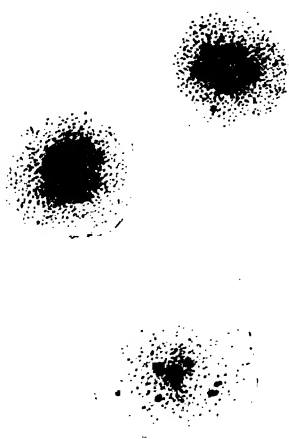
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PLATE XXXII

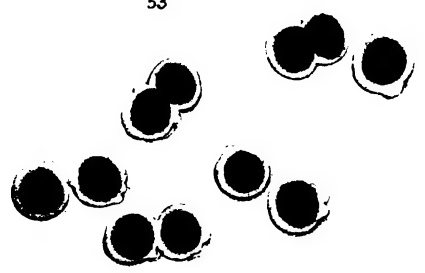
FIG.

52. Colonies of the bacillus diphtheriæ on gelatine, seen in transmitted light, after ten days' incubation at 20° C. Magnifying power, 10.
53. Same after three weeks' incubation. Magnifying power, 10.
48. Colonies of the bacillus of fowl enteritis on gelatine, seen in transmitted light, after several days' incubation at 20° C. Magnifying power, 8.
56. Colonies of the bacillus diphtheriæ on the slanting surface of agar, seen in transmitted light, incubated two weeks at 36–37° C. Natural size. The lithographic representation is not quite correct, the centre of the colonies being less knob-like, as here represented, and more yellow brown.
61. Streak culture on gelatine of micrococcus aurantiacus, after a week's incubation at 20° C. Natural size.

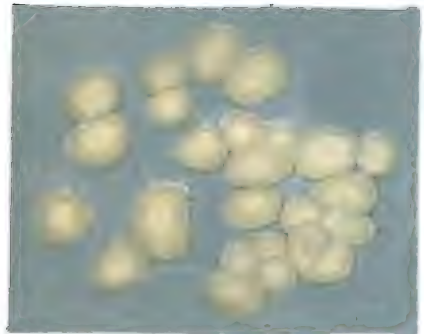
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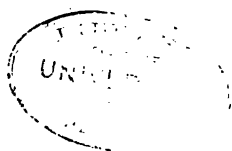


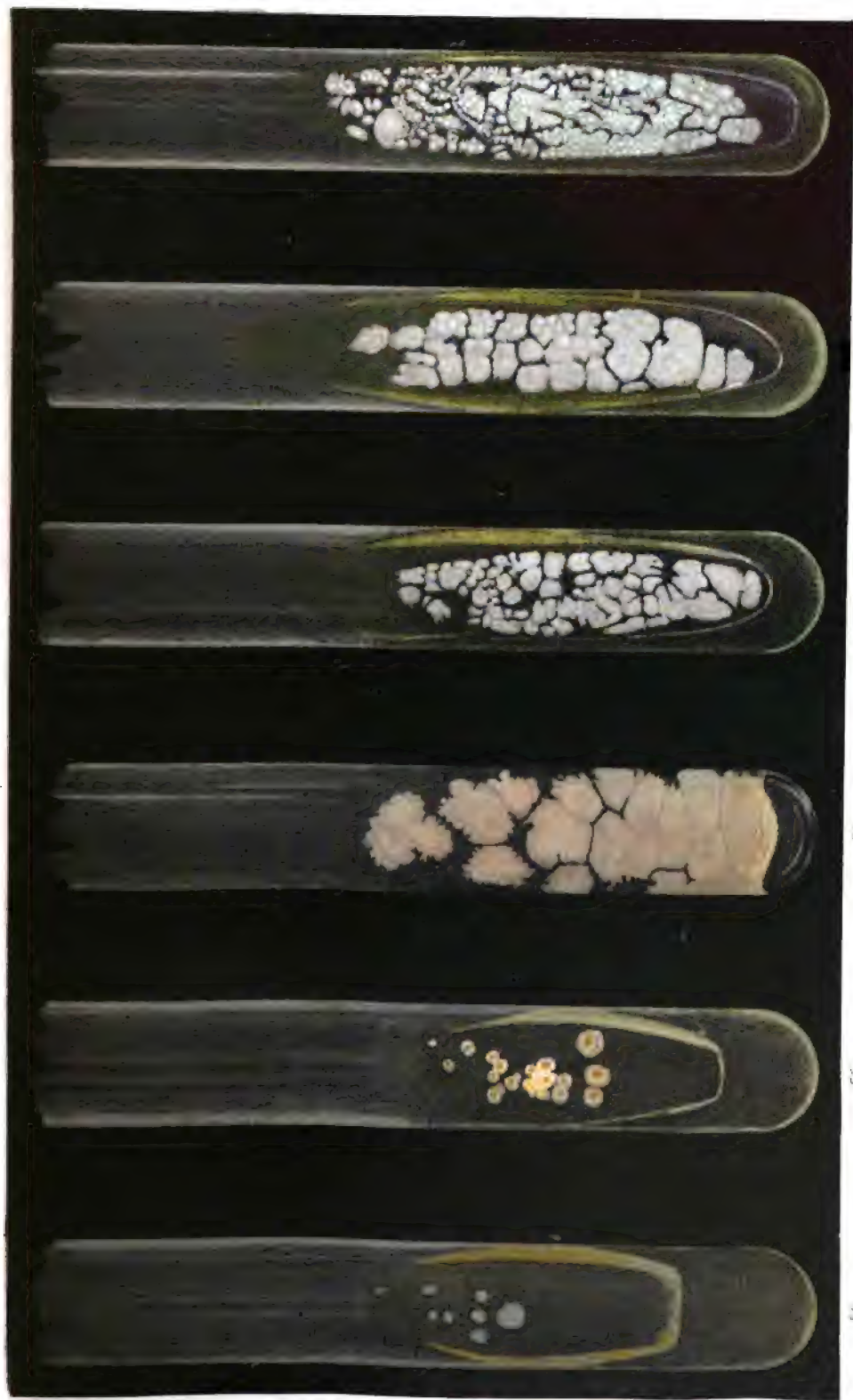


PLATE XXXIII

TUBE CULTIVATIONS, NATURAL SIZE, SEEN IN REFLECTED LIGHT

FIG.

54. Colonies of the glanders bacillus on the surface of agar, after nine days' incubation, at 87° C.
55. Colonies of the diphtheria bacillus on agar, after two weeks' incubation.
57. Colonies of the Middlesbrough pneumonia bacillus on gelatine, one week's incubation.
58. Colonies of the bacillus of fowl enteritis on gelatine, one week's incubation.
59. Colonies of the typhoid bacillus of Eberth-Gaffky on gelatine, after one week's incubation.
60. Colonies of the bacillus of grouse disease on gelatine, after one week's incubation.



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PLATE XXXIV

TUBE CULTIVATIONS, NATURAL SIZE, SEEN IN REFLECTED LIGHT

FIG.

62. Streak culture on gelatine of the bacillus of pork-pie poisoning.
63. Streak culture on agar of the staphylococcus aureus (37° C.).
64. Streak culture of bacillus pyocyaneus on agar (37° C.).
65. Streak culture on gelatine of the bacillus of Middlesbrough pneumonia.
66. Streak culture on gelatine of the bacillus diphtheriæ.
67. Streak culture on agar of the bacillus prodigiosus (20° C.).





PLATE XXXV

TUBE CULTIVATIONS, NATURAL SIZE, SEEN IN REFLECTED LIGHT

FIG.

68. Surface cultivation on glycerine agar of the tubercle bacillus after five months (37° C.).
69. Similar cultivation after nine months (37° C.).
70. Stab culture in gelatine of the bacillus of Middlesbrough pneumonia.
71. Stab culture in gelatine of the staphylococcus albus liquescens, after three days' incubation, at 20° C. ; liquefaction of the gelatine very marked.
72. Stab culture in gelatine of Finkler's comma bacillus after about three days.
73. Stab culture in gelatine of the bacillus pyocyaneus after three days.



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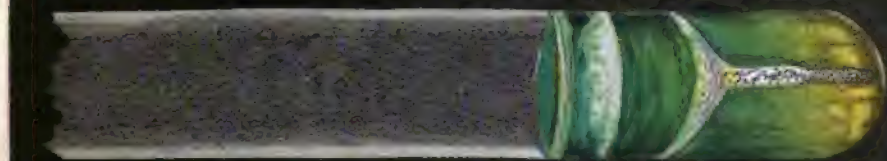


PLATE XXXVI

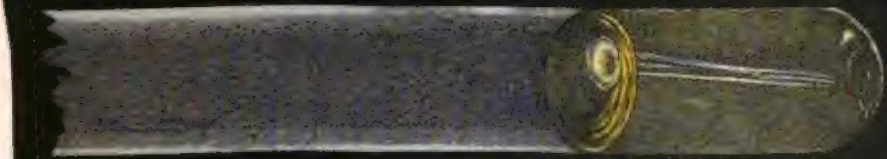
TUBE CULTIVATIONS, NATURAL SIZE, SEEN IN REFLECTED LIGHT

FIG.

74. Stab culture in gelatine of the *bacillus fluorescens liquescens*, after seven days.
75. Stab culture in broth gelatine of the glanders bacillus, after seventeen days' incubation, at 22° C.
76. Same after twenty-three days. Slight liquefaction has commenced at the upper end of the stab.
77. Stab culture in gelatine of the bacillus of swine erysipelas, after fourteen days.
78. Stab culture in gelatine of the *bacillus anthracis*, after seven days. Liquefaction has commenced at the upper end of the stab.
79. Same after fourteen days. Liquefaction on the surface is more pronounced.



74



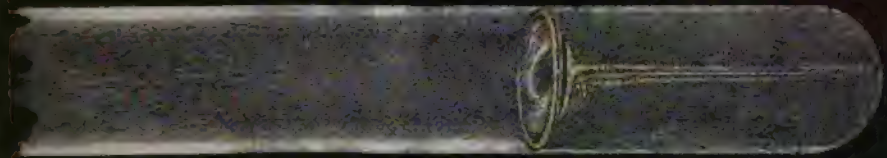
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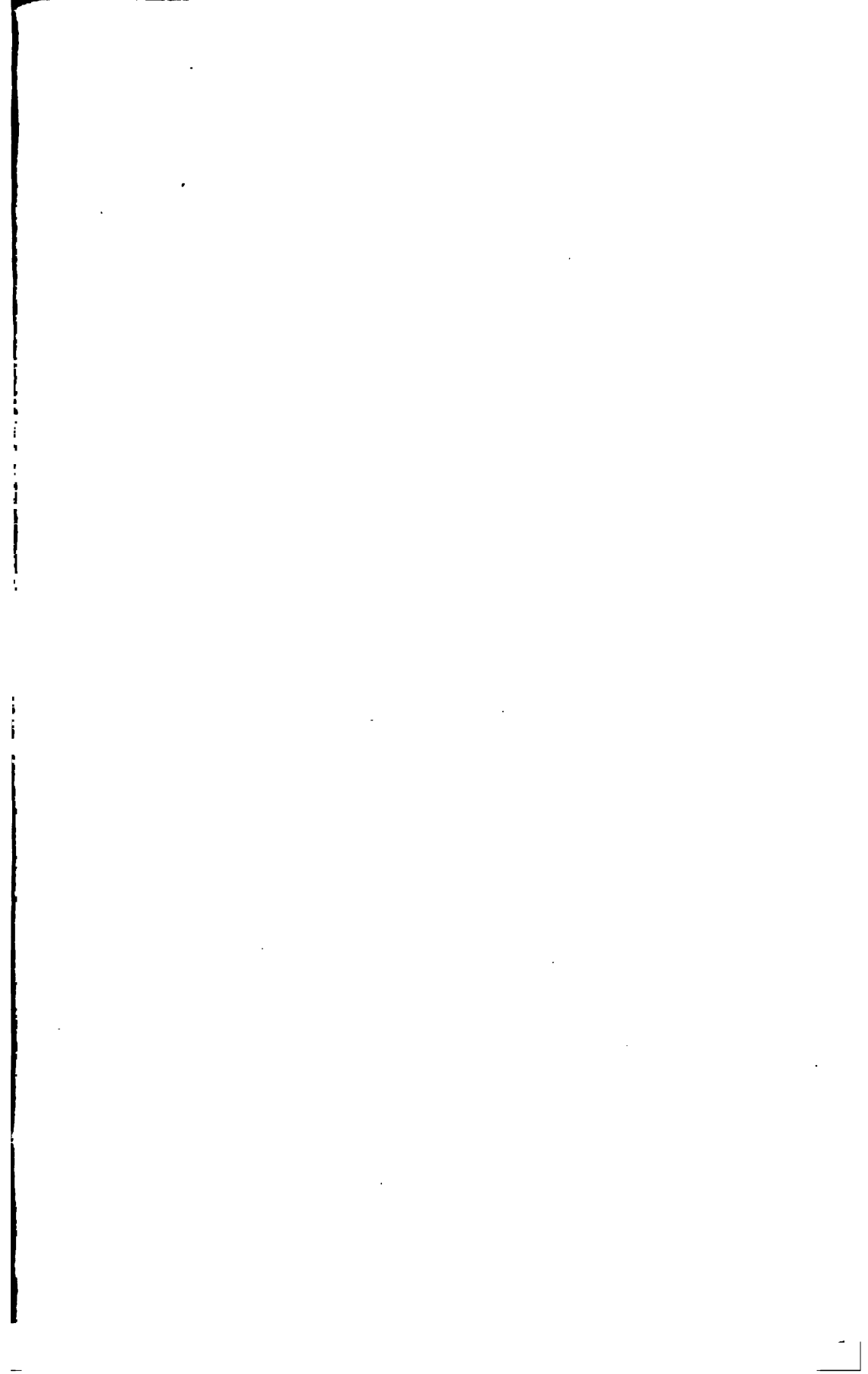


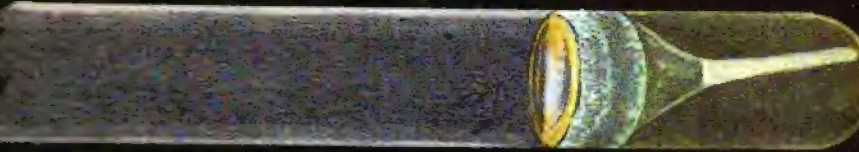
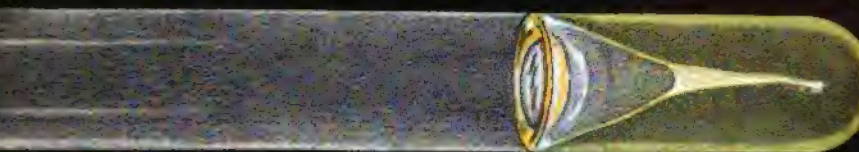
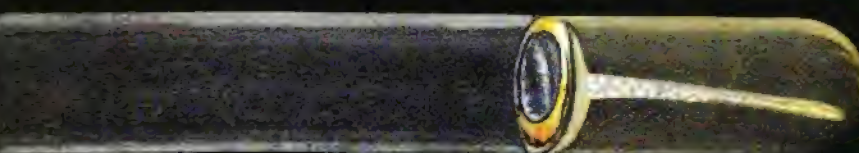
PLATE XXXVII

TUBE CULTIVATIONS, NATURAL SIZE, SEEN IN REFLECTED LIGHT

FIG.

80. Stab culture in gelatine of bacillus anthracis after seven days. Liquefaction at the surface has commenced.

81, 82, 83, 84, and 85 are stab cultures in gelatine of different varieties of cholera spirilla, all after eight days' incubation, at 20° C. They all show the funnel-shaped depression on the surface, and in all liquefaction of the gelatine is proceeding; but the amount of growth and the rapidity of liquefaction are different in the different varieties.



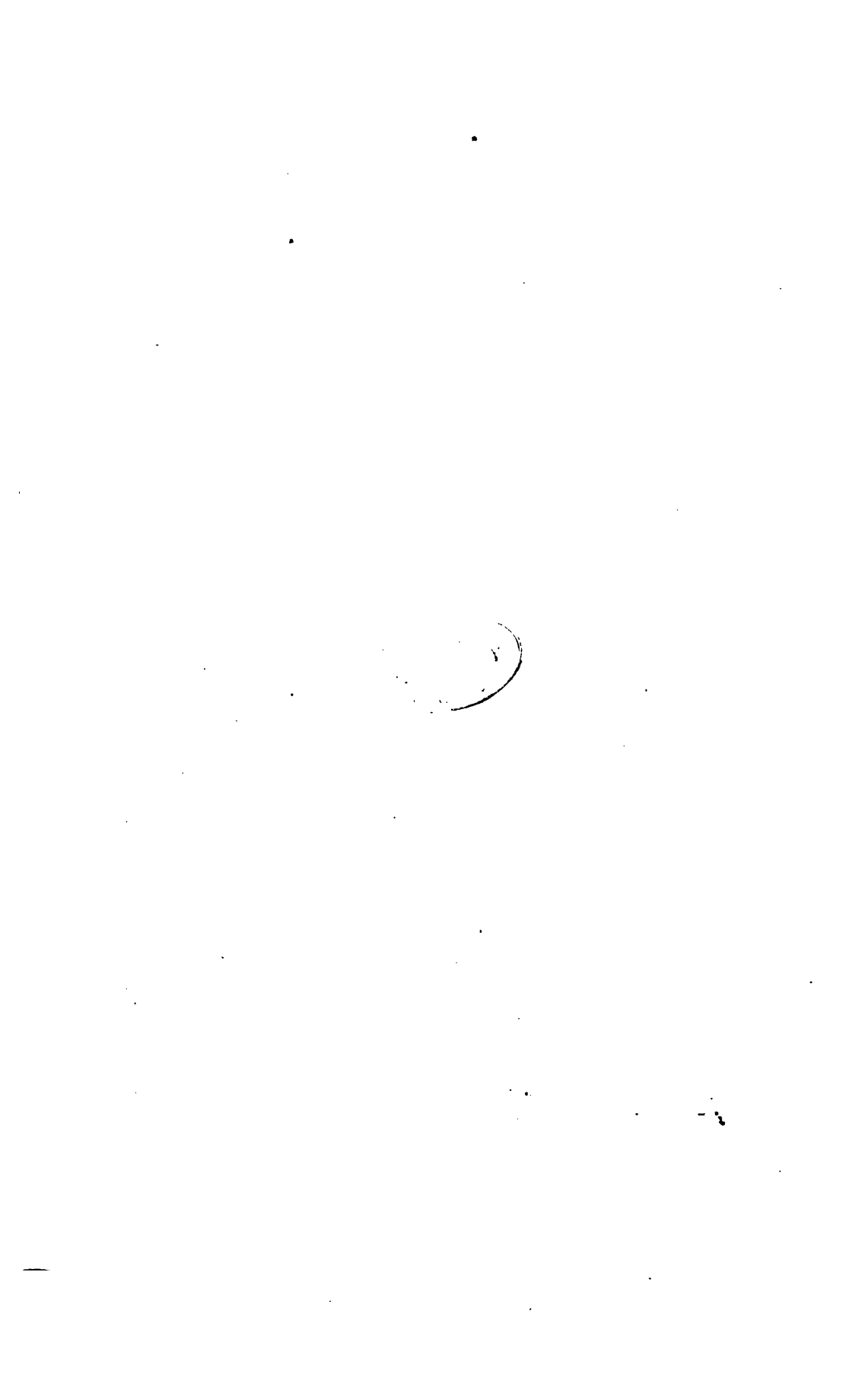
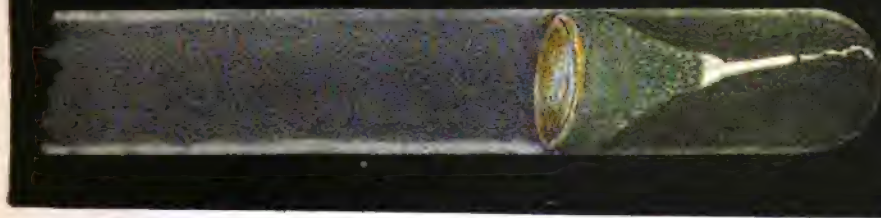


PLATE XXXVIII

TUBE CULTIVATIONS, NATURAL SIZE, SEEN IN REFLECTED LIGHT

FIG.

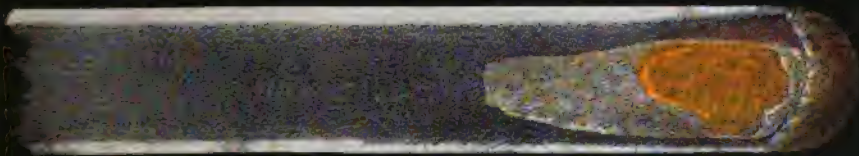
86. A further variety of the cholera spirilla, belonging to the series shown in Plate XXXVII. This last one corresponds to the one described by Koch.
87. Culture on potato of the *sarcina lutea*.
88. Culture on potato of a bacillus of chronic necrotic deposit in the membrane of the fauces of fowl.
89. Culture on potato of *bacillus prodigiosus*.
90. Culture on potato of bacillus of glanders, after fourteen days' incubation.



86



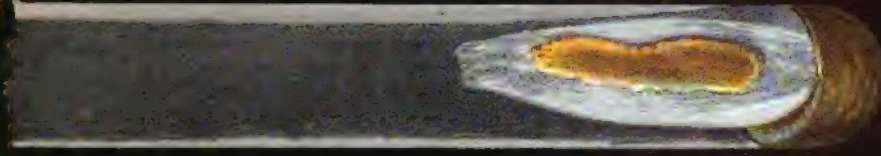
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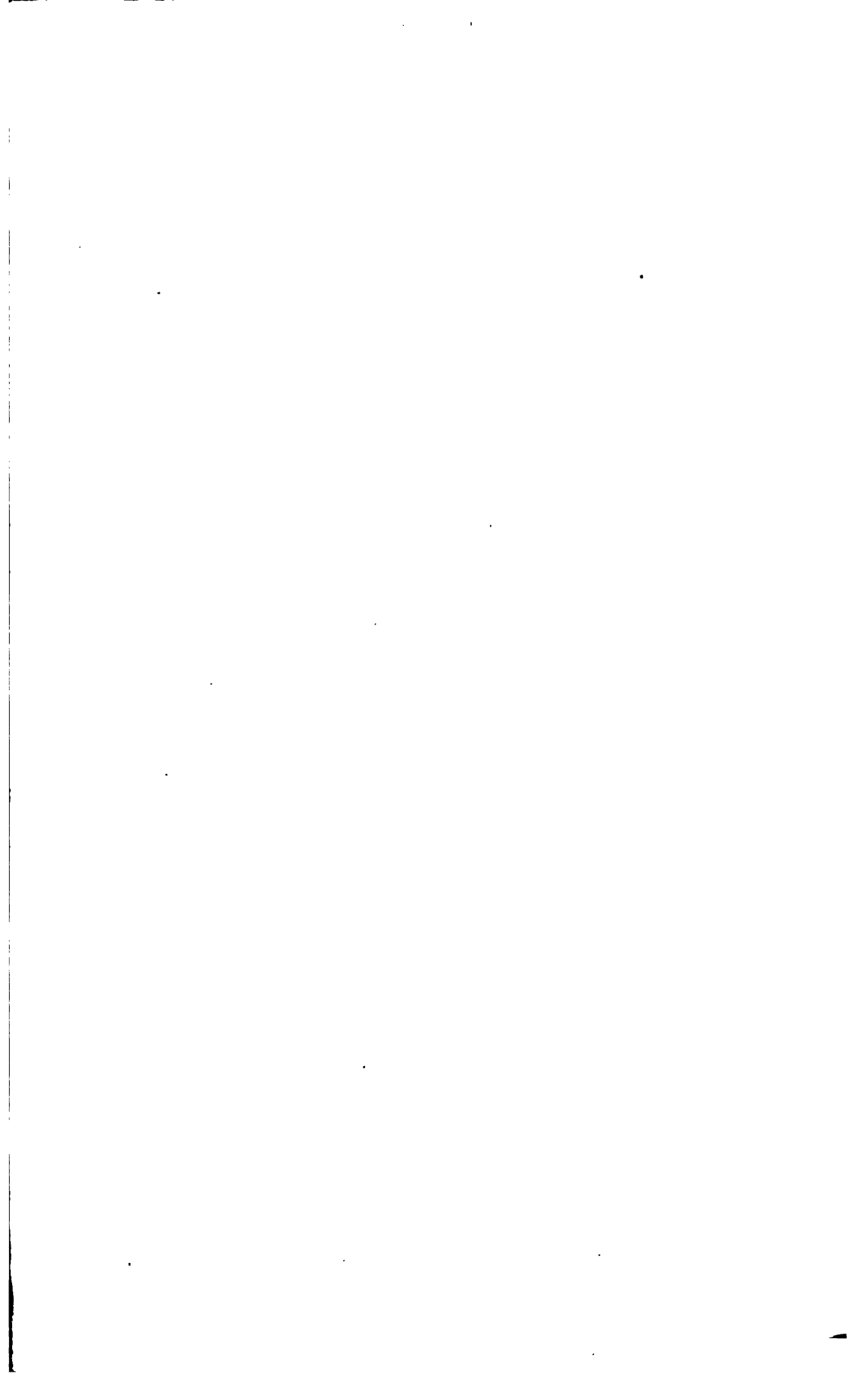


PLATE XXXIX

POTATO CULTURES IN TUBES, NATURAL SIZE, SEEN IN REFLECTED LIGHT

FIG.

91. Cultivation of bacillus of glanders, after twenty-six days. This is a companion cultivation to that shown in fig. 90 of Plate XXXVIII, after fourteen days' incubation.

92, 93, 94, and 95. Cultivations on potato of different varieties of cholera spirilla.

92 corresponds to the variety shown in gelatine culture 86.

93 corresponds to the variety shown in gelatine culture 81.

94 corresponds to the variety shown in gelatine culture 83.

95 corresponds to the variety shown in gelatine culture 85.





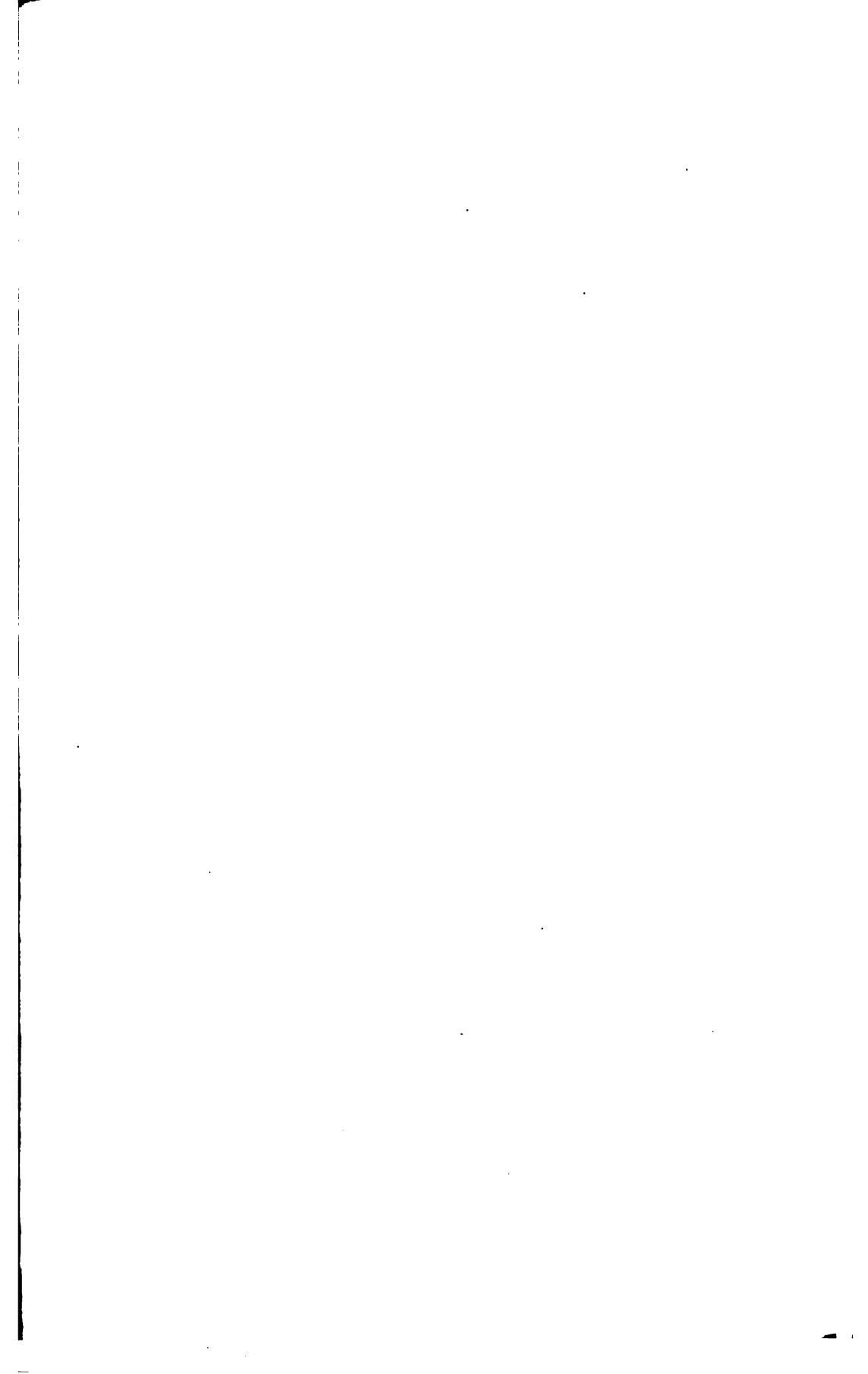


PLATE XL

FIG.

99. *Oidium lactis*, mycelium, and spores. Copied from Klein's 'Micro-organisms and Disease,' fig. 114.
100. *Saccharomyces mycoderma* (after Gravitz) from an artificial cultivation in dilute nutritive material: (*d*) branched mycelium; (*f*) α , torula stage; β , mycelial stage. Copied from Klein's 'Micro-organisms and Disease.'
106. From a section through the kidney of a rabbit dead thirty-six hours after the injection of *aspergillus* spores into the jugular vein (after Gravitz). T, Tyrosin crystals; F, fat droplets. In the upper part of the figure is a metastatic focus, composed of spores and mycelium, numerous fat droplets amongst them. In the lower part the uriniferous tubules and two Malpighian corpuscles are shown. Copied from Klein's 'Micro-organisms and Disease,' fig. 118.
104. *Aspergillus glaucus* (after De Bary). A, hypha. At the end, at *c*, it bears *st*, the basidia; *as*, ascogonium.
105. E, perithecium; *as*, ascogonium; *w*, cells of the pollinodia. Copied from Klein's 'Micro-organisms and Disease,' figs. 116 and 117.
107. *Saprolegnia* of salmon disease. The figure represents a sporangium filled with zoospores; in connection with it are several young mycelial threads, marked by their coarsely granular dark protoplasm. Copied from Klein's 'Micro-organisms and Disease,' fig. 119.

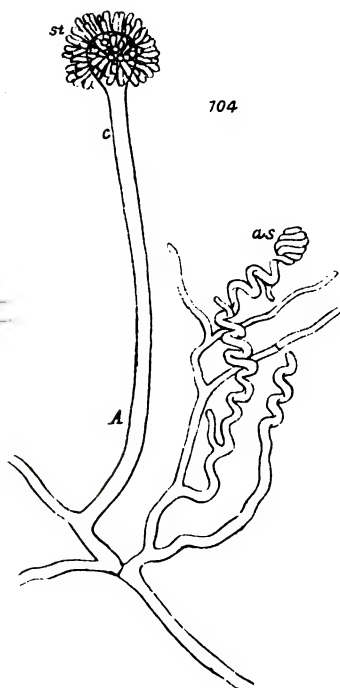
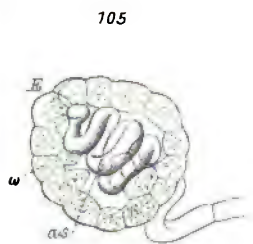
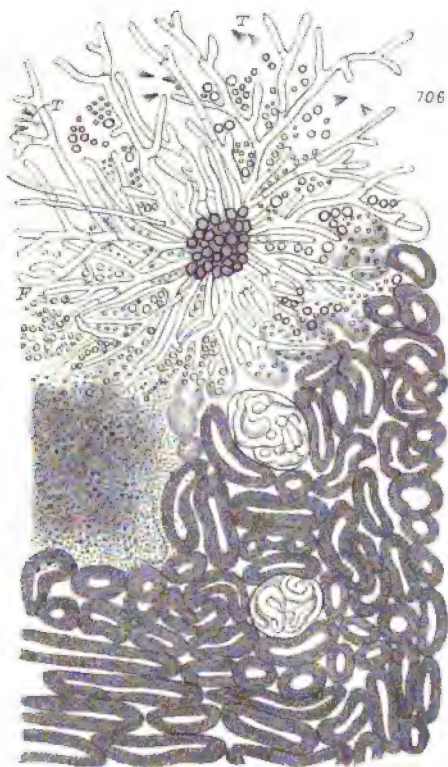
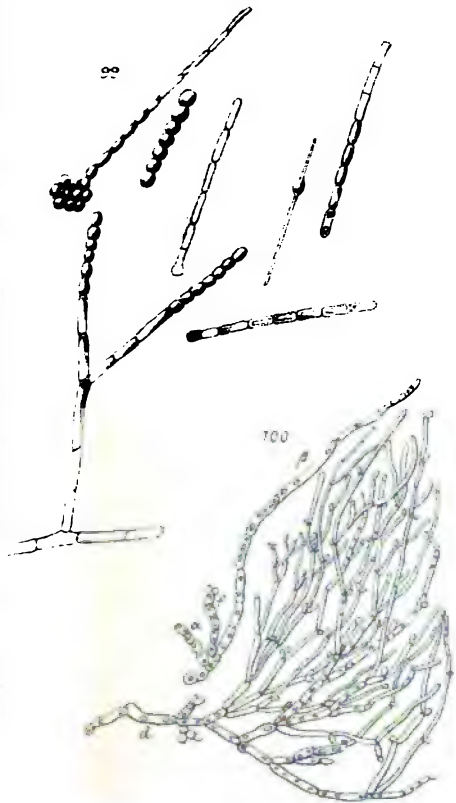




PLATE XLI

Fig.

101. Herpes fungus, mycelium, and spores, after Gravit, copied from Flüge's 'Mikroorganismen,' fig. 17.
108. Mucor corymbifer, hyphæ and sporangia, after bursting of the membrane of the sporangia. After Lichtheim, copied from Flüge's 'Mikroorganismen,' fig. 21 *b*.
98. Torula or saccharomyces. In the lower part of the figure ascospores and four isolated spores (after Rees) are shown. Copied from Klein's 'Micro-organisms and Disease,' fig. 112.
102. Penicillium glaucum : (*m*) mycelium ; from it rise non-septate hyphæ bearing at their ends the spores. Copied from Flüge's 'Mikroorganismen,' fig. 22.
96. Amœba coli in mucus of intestine. Numerous blood-corpuscles and degenerated epithelial cells are also seen. After Löscher, copied from Leuckart's 'Die Parasiten des Menschen,' I., 1, p. 235, fig. 94.
97. Coccidium oviforme from the liver of rabbit seen 550 times magnified. *c* to *g*, progressive changes during the formation of spores, observed only outside the animal body. Copied from Leuckart's 'Die Parasiten des Menschen,' I., 1, p. 256, fig. 106.

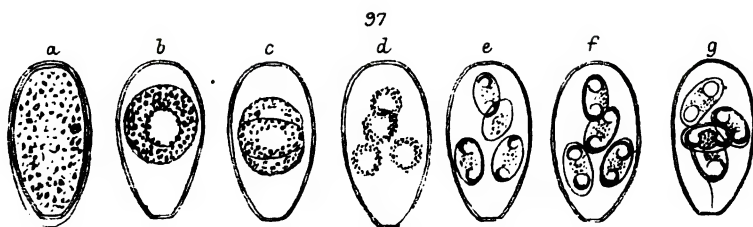
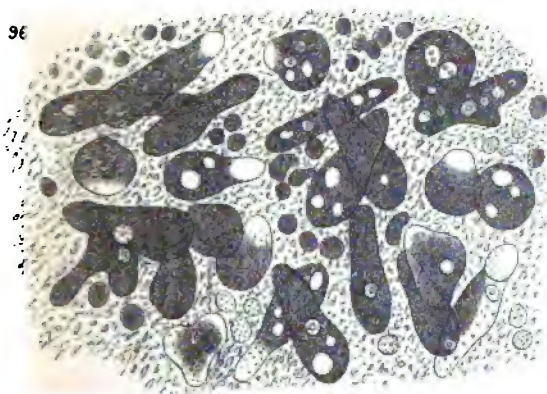
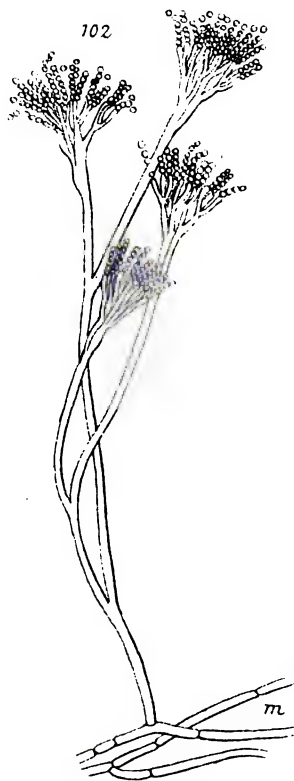
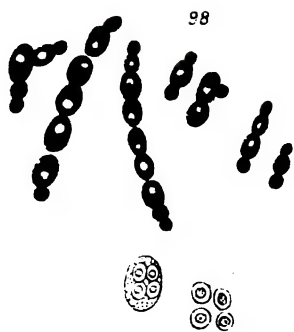
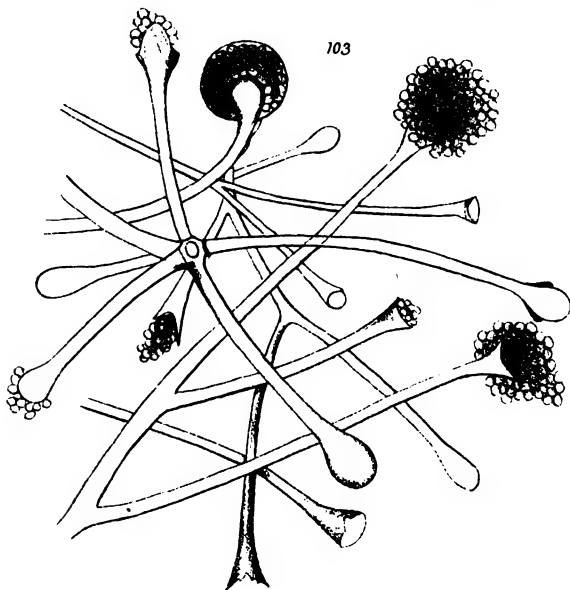
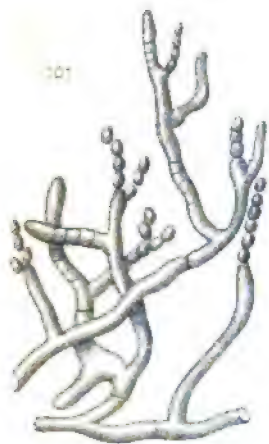




PLATE XLII

This plate is copied from Golgi, Studien über Malaria, 'Fortschritte der Medicin,' Bd. IV, Tafel III.

Figs.

108-119 show the malaria plasmodium as a pale amœboid body inside a red blood disc. It gradually increases in size at the expense of the substance of the red blood disc: in 116-119 it has become conspicuously enlarged, and contains masses of pigment granules derived from the hæmoglobin.

120-125 show the various progressive stages in the process of the cleavage of the plasmodium and the gradual shifting of the pigment granules.

126-131 show the completion of the process of cleavage and the final isolation of the spores; the dark granules are the pigment granules.

132-138 are oval parasites—Laveran's corpuscles, from atypical cases of malaria.

108



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110



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114



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120



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122



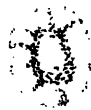
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THE NATURAL HISTORY OF INFECTIOUS DISEASES

BY

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THE NATURAL HISTORY OF INFECTIOUS DISEASES¹

In this article it is proposed to deal, mainly from the point of view of natural history, with some of the more important of that class of diseases which may at present be regarded as coming within the domain of public health.

The following is a list of the diseases about to be considered, taken in the order in which they are given by the Registrar-General :—

Measles	Mumps	Malarial Disease
Rötheln	Diphtheria	Erysipelas
Scarlet Fever	Cerebro-spinal Fever	Puerperal Fever
Typhus	Simple Continued Fever	Tuberculosis
Relapsing Fever	Enteric Fever	Croup
Influenza	Cholera	Pneumonia
Whooping Cough	Diarrhoea	Yellow Fever ²

As regards history, it is perhaps sufficient here to say that diseases akin to those under consideration have existed from very early times. Some of the actual diseases in question are indeed clearly referred to by the earliest medical writers. Historical records also support the view that at different periods of the world's history the diseases most conspicuously affecting man have varied in type, as well as in geographical distribution from time to time.

Whatever their variations and whatever their distributions in space, it will be generally admitted that these diseases are in all cases due to the effects upon the body of poisons originally introduced from without. The precise nature of these poisons can of course only be finally ascertained by experiment, though in this matter the laboratory investigator must work in concert with the epidemiologist, each supplying the other with indications for further observation or experiment.

But it having now been demonstrated that the poisons responsible for the causation of some of the diseases in question are bound up with the life processes of microphytic organisms, not only is the view originally based upon clinical and epidemiological considerations—that the diseases of this class are due to processes allied to fermentation—completely established as regards some of them, but very strong suggestion arises that it is true also of the whole class; indeed, so strong is the probability of such being the case that we are justified in adopting this so-called germ theory of these diseases as an hypothesis deserving to be put to the test. This course has

¹ The use of the term 'infectious' as a class name for these diseases is no doubt open to criticism, since, in the sense in which it is popularly understood, it can hardly be said to be applicable to all the maladies enumerated above. In the present state of knowledge, however, it is difficult, probably impossible, to find a class name which is at the same time applicable to all the diseases in question, and free from the objection of connoting etiological views at present unproved as regards the diseases as a class.

² Yellow Fever is, of course, not included in the mortality statistics of the Registrar-General.

been very generally entered upon with results which, so far, appear entirely to justify the procedure; for not only has the germ theory been found to lend itself in a remarkable degree to the explanation of the observed phenomena of disease of this sort, but the labours of the bacteriologist are day by day extending the area of demonstrated association between microphytic life processes and particular human and animal diseases. It is true that closer study of these microphytic life processes has led to some modification of the germ theory as originally formulated, in the sense of showing that, as regards an increasing number of diseases, the pathogenic influence of the microphyte upon the body is indirect, resulting rather from the action of certain chemical poisons formed during the cycle of microphytic life than from mere multiplication within the body of the microphytes themselves, as had been formerly supposed; and still further modifications of the germ theory will no doubt follow with increasing knowledge. Nevertheless it may fairly be said that the whole progress of research has tended to confirm the position of the microphyte itself as in one or other way the cause of the diseases of this class.

It is necessary, however, to state that the expression 'the cause' is here used in the popular sense only, for the *real cause* of any effect is generally, if not always, the resultant of a combination of causes. That in the present instance this point is one of practical importance, must be evident; and a moment's consideration will satisfy anyone that in the production of disease many highly important factors are operative in addition to the microphyte itself; for, although the microphyte may be truly described as an essential element in the cause, it is none the less true that for the effective operation of the microphyte certain conditions of environment are also requisite. Such conditions may be those which are external to the sufferer—e.g. climatic, topographical, or domestic; or they may be intrinsic conditions—viz., peculiarities, *quod* susceptibility, of species, sex, age, heredity, and tissue condition of the individual.

Any attempt to unravel the problems of disease origin and propagation must take account of these several conditions, and it is to be feared that in the outburst of zeal to which the modern study of bacteriology has given rise, this consideration has been to some extent overlooked; a fact which perhaps accounts for the nature of some of the criticisms to which 'the microbe' and its supporters have been subjected by clinical physicians and students of practical medicine.

But to return to our hypothesis. The occurrence of an attack of one of these diseases presupposes the operation of microphytic life, or the products of microphytic life, upon the person attacked, and the question arises, whence came the microphyte? Was it the progeny of a similarly endowed parent, or did it arise independently of such parent?

These questions at once open up two conflicting views which have been extensively held as regards the origin of the more infectious of the diseases under consideration. On the one hand, the frequency with which such diseases have been observed to spread by infection from case to case has led to a widespread belief in infection as the only mode of their origin and dissemination. And this view—which implies that the microphyte is always derived, directly or indirectly, from a pre-existing case of disease—is still the orthodox one as regards certain well-known diseases. On the other hand, owing to the fact that in a certain proportion of instances it has been thought impossible to trace fresh attacks of a given disease to previous cases, and owing also to certain *a priori* considerations, the old theory of spontaneous generation has at different times been revived for explanation of the pro-

blem of disease origin; and it has been held by some that the microphytic causes of disease—although often, and as regards some diseases usually, descended from parent microphytes of like kind—are yet at times capable of arising independently of such parents.

As the views which we entertain with regard to these and allied questions lying at the root of the problems of disease origin and dissemination must profoundly influence our general attitude of mind towards the diseases under consideration, it is important that the subject should be considered somewhat closely. It is proposed, therefore, to inquire how far either of the two views above referred to is in accord with the facts and indications of modern science.

It will be convenient first to consider the spontaneous generation theory, or, as it is now more correctly called, the *de novo* theory.

The suggestion that one or other of these diseases has arisen *de novo* might be taken to contain the assumption that the micro-organism, either known or inferred to be the cause of such disease, has been originated afresh, presumably out of non-living matter. It becomes necessary, therefore, to ascertain whether there is any foundation for such a belief as this, and for this purpose there is occasion to refer to the general doctrine of abiogenesis.

That the origin of beings whose parental descent was not obvious should have been ascribed to spontaneous generation is not surprising, and accordingly we find that, 'the checks which experience alone can furnish being absent, the spontaneous generation of creatures quite as high as the frog in the scale of being was assumed for ages to be a fact. Here, as elsewhere, the dominant mind of Aristotle stamped its notions on the world at large. For nearly twenty centuries after him men found no difficulty in believing in cases of spontaneous generation, which would now be rejected as monstrous by the most fanatical supporter of the doctrine.'¹

One by one, however, these beliefs have given way before better knowledge, and, as Sir Joseph Lister remarks, 'the doctrine of spontaneous or equivocal generation has been chased successively to lower and lower stations in the world of organised beings as our means of investigation have improved.'² These results of progressive investigation naturally incline us to regard all phases of the abiogenesis theory with grave suspicion, and to anticipate that future additions to our knowledge will still further support the view expressed in the formula *omne vivum ex vivo*. But the modern doctrine of evolution has been considered by some to put a different aspect upon the matter. It has been argued that though according to that doctrine in its wider sense the various forms of life existing upon the globe owe their existence to unbroken parental descent from the *lowest* forms of life, these lowest forms of life must themselves have arisen at some time or other, and of necessity, from non-living matter. And if once, why not again? It has been further pointed out that 'those who strictly adhere to the Evolution Hypothesis could never believe in the origination of any but the "lowest and simplest" organic forms by a process of Archebiosis. So that the gradual driving of the question back as one possibly applicable to such organisms only, is just what the Evolutionist would have expected. . . .'³ As a purely abstract criticism upon the historical argument this at first sight appears to have weight, but when it is applied to the concrete aspect of the question it loses point. For although it may of course be true that all existing forms of life are descended from the *lowest* forms of living matter,

¹ "Spontaneous Generation." Tyndall's *Fragments of Science*, ii. 292.

² *Introductory Address* (University of Edinburgh), 1869, p. 12.

³ *Evolution and the Origin of Life*, p. 29. By H. Charlton Bastian, M.D., F.R.S.

and that such again were originally evolved from non-living matter, what are the grounds for assuming that the particular forms of life, such as the bacteria, with regard to which it has been endeavoured to establish a *de novo* origin, do, as a matter of fact, constitute the lowest forms of living matter? On the contrary, may not the decided variations in function which they exhibit fairly be regarded as indicating a considerable degree of complexity of structure, notwithstanding that such complexity may not be recognisable by any means as yet at our disposal?

Furthermore, it would appear that the abstract discussion of this matter upon evolution principles by no means necessarily favours the doctrine of abiogenesis. Not only are there no sufficient grounds for supposing that we have yet found the lowest possible form of life—'the first organism' which has been assumed by some to be a necessity—but, as Mr. Herbert Spencer remarks, 'the conception of a "first organism" in anything like the current sense of the words is wholly at variance with the conception of evolution. . . . The affirmation of universal evolution is in itself the negation of an "absolute commencement" of anything. Construed in terms of evolution, every kind of being is conceived as a product of modifications wrought by insensible gradations on a pre-existing kind of being; and this holds as fully of the supposed "commencement of organic life" as of all subsequent developments of organic life. It is no more needful to suppose an "absolute commencement of organic life" or a "first organism" than it is needful to suppose an absolute commencement of social life and a first social organism. The assumption of such a necessity in this last case made by early speculators with their theories of "social contracts" and the like, is disproved by facts; and the facts, so far as they are ascertained, disprove the assumption of such a necessity in the first case.'¹

It thus appears that the doctrine of evolution, according at least to one of its most distinguished exponents, gives no *a priori* support to the notion of the *de novo* origin of any organisms properly so called, much less of organisms such as those upon which certain diseases are known to depend—i.e. organisms capable of being identified and of propagating their kind. Of course it might be urged, as indeed it has been urged, that time like all else is relative; that an interval which to us is but a short period represents perhaps æons of time in the life history of lower organisms, and that therefore the evolution of living organisms out of non-living organic matter may be accomplished in a few hours or a few days.

However this may be, there appears sufficient experimental evidence to show that, whether or not organisms of the class we are discussing may be mentally conceivable as arising *de novo* in a short space of time, they do not as a matter of fact now so arise. And this brings us to the gist of the matter. The whole question is one which must be decided by a balance of evidence accumulated by long-continued observation and experiment. Judged by this test we have in the experiments of Pasteur, Tyndall, and others, and in the successful application of the teaching of such experiments to the practical arts, an overwhelming mass of testimony against spontaneous generation. It has been shown that, if properly sterilised and protected against accidental germ contamination, putrescible substances may be kept without alteration for indefinite periods of time; though putrefaction may be at once set up in such substances by the simple device of admitting to them a germ-charged atmosphere. All along the line, in fact, experimentation has gone to show that there is no life apart from antecedent life. Whatever, there-

¹ *Principles of Biology*, vol. i., Appendix.

fore, may have been the origin of life, the conclusion seems forced upon us alike by observation and experiment, that in the present day there is no life in the sense of anything which could with any propriety be described as a living being or organism which has not descended from antecedent life.

How far this conclusion negatives the possibility of a *de novo* origin of disease as disease will be presently seen. For it has to be noted that epidemic diseases, as we observe them, are simply the manifestations exhibited by man or other animal of the operation of certain poisons which we assume to be bound up with micro-organisms. And conditions may easily be imagined which would give rise to an apparent or even a real *de novo* origin of disease independently of any *de novo* origin of bacteria.

To summarise the point reached: We have inferred, provisionally, from what we already know of certain of these diseases, as well as from the general direction of bacteriological research, that all the diseases under consideration are due to microphytic life processes; and we have further seen no reason to doubt that the microphytes upon which these diseases depend are invariably descended from parent microphytes, and that, therefore, nothing of the nature of a true *de novo* origin occurs so far as the microphytes themselves are concerned.

Having now sufficiently cleared the ground by a review of the theory of *de novo* origin of the germs of disease, we may go on to consider the thesis which, in the absence of 'spontaneous generation' of disease germs, has been regarded by some as the only alternative—namely, the unbroken continuity of disease descent from antecedent cases. This doctrine, signifying, as it seems to do, belief that every single case of each of the diseases in question is the progeny, so to speak, of an antecedent case of the same disease, is one of little elasticity. In so far, indeed, as certain of these diseases affect the lower animals as well as man, the antecedent case of a given sample of such disease need not be a human case; but as regards others, which are held to exclusively affect man, the antecedent case must be sought in the human subject alone. But we now see difficulties in the way of this thesis, and hence arises occasion for reconsideration of the subject from the point of view which regards the microphytes upon which these diseases depend as invariably descended from parent microphytes.

Although it is no doubt true that some of the disease microphytes now in question, though capable (whether as bacteria or as spores) of existing in a latent condition outside the body for considerable periods of time, may yet be incapable of passing through their life cycle, including of course proliferation, except in human living tissue—so that diseases due to such microbes usually arise (in the present) by direct or indirect infection from a previous human case of the same disease—we nevertheless know that there are microphytes, causative of disease, which are capable of thriving and multiplying either upon human or other animal tissues; and that there are others again which have their habitat largely outside the living body, either human or animal, and are capable of thriving and multiplying upon non-living organic matter.

Accordingly, to affirm the continuity of life, from generation to generation, of the microphytic causes of disease, is by no means the same thing as to allege that human diseases descend in a continuous series from human case to human case. For instance, with respect to a particular species of pathogenic micro-organism capable of preying either upon human and other animal hosts, and of thus giving rise to a disease intercommunicable between man and other animals, it is obvious that although the micro-organisms them-

selves would, according to the view here adopted, descend in a continuous series from antecedent parent organisms, yet the cases of human disease dependent upon such organisms would not necessarily follow in a continuous series from human case to human case.

But we may go farther than this. It is evident that a given attack of disease might be due to the action of microbes which, although not developed *de novo*, had yet descended from a particular strain of microbes that had not, for a number of generations, found a habitat either in human or other animal bodies, though their remote ancestors might have done so or might not. In such an instance the disease would practically have for man a new beginning so far as this particular occurrence of it is concerned. The question therefore whether diseases do or do not descend in a continuous series from antecedent cases is one which must be worked out separately, as regards each disease, by a study both of the epidemiological behaviour of different diseases and the life history of the particular microphytes upon which such diseases depend.

So far, however, the matter has been discussed as though pathogenic property remained a constant quantity for each species of micro-organism, and throughout the life cycle of each individual micro-organism. But we are by no means assuming this either as regards the individual or the species, and many facts pointing to an opposite conclusion might be cited. It is notorious, for instance, that very considerable variations both of severity and type are observed between different epidemics of the same disease, and even between different stages of one and the same epidemic. Such variations, too, at times occur under circumstances which seem to render it improbable that they are due to corresponding variations in the conditions of the communities affected, and we thus seem led to the view that differences of severity and type are likely to be due to variations from time to time occurring in the pathogenic property of the same species of micro-organism.

And besides the variations observed in epidemics as a whole there are commonly considerable differences both of severity and type between different individual cases of a given disease during a particular epidemic; and although such individual differences are no doubt more largely due than in the former case to personal differences of the people attacked, and probably also to differences in the dose of the virus received, yet they may be in part due to differences of pathogenic property of one and the same species of microbe.

Now differences in the pathogenic property of micro-organisms of the same species may from time to time doubtless result from a variety of causes, among which temperature, moisture, light, and other factors, probably have important influence. But there would appear to be other causes, and it is to these that it seems desirable here to refer. It is well known that the pathogenic property of certain microbes varies largely according to the soil in which they are sown—accordingly, for instance, as they are cultivated in the body of one or another kind of animal. This would suggest that outside as well as within the laboratory the pathogenic property of micro-organisms that prey alike upon man and other animals may be modified from time to time by the 'soil' of the particular class of animal they chance to invade. And, further, as different species of animals seem to differ in their influence upon the pathogenic property of some micro-organisms, so possibly may different individuals of the same species differ; and certain individuals, having become infected, may be thought of as handing on the virus to others, either in a relatively attenuated or more potent form than that in which they themselves received it. Considerations of this kind may perhaps supply the key to the explanation of some of the differences ob-

served in the severity and type of different epidemics and different cases of the same epidemic. And, indeed, it is easily conceivable that the influence of the hosts upon the pathogenic function of micro-organisms may be capable of originating actually new varieties of disease.

It may be asked whether the pathogenic property of some micro-organisms may not be a function which has become superadded, so to speak, to micro-organisms originally saprophytic, as a result of their having gradually acquired ability to thrive upon animal hosts. Or, indeed, whether all pathogenic parasites may not have descended from saprophytes by a process of adaptation to environment.¹ It is so far consistent with this view that there is no sharp line of demarcation between the saprophytes and the parasites, and we should anticipate that in the struggle for existence such microphytes as were capable of adapting themselves to new environments would, in surviving, undergo variation. There would seem no reason therefore why the pathogenic function should not have been in the first instance acquired, and subsequently from time to time modified in this way. If this should be so, the evolutionary changes of micro-organisms might afford very considerable possibilities as regards disease variation, and even disease origin, altogether apart from any 'spontaneous generation' of disease germs. For, although disease germs of the more highly specialised sorts must doubtless be expected to exhibit a relatively high degree of constancy as regards pathogenic property, others, less specialised, would probably in this respect be subject to considerable variation, either in the direction of increasing capacity for production of pathogenic material, or of reversion toward innocence of function in this respect.

In view of the criticism that evolutionary changes would be expected to occupy longer periods of time than seems to be here contemplated, it has to be borne in mind that the evolution now suggested is one of function rather than a decided morphological evolution. And although, as will be pointed out presently, many micro-organisms may be remarkably stable, both pathogenically and morphologically, it has been shown under artificial conditions in the laboratory that, as a matter of fact, the functions of others may undergo considerable modification in comparatively short periods of time—and modifications so lasting as to be transmitted by heredity.²

The foregoing considerations serve to illustrate some of the senses in which a *de novo* origin of disease may still be contemplated as a possibility without any departure from the doctrine of 'no life without antecedent life,' as applied to the microphytic causes of disease.

To recapitulate. It seems that although diseases due to highly specialised obligate parasites whose life processes are dependent upon residence in a human host probably always descend from antecedent cases of the same disease, there are yet possible ways in which, independently of any 'spontaneous generation' of microbes, other diseases might, in one or another sense, be said to have a new beginning for human beings.

Illustrations, which, however, do not profess to be exhaustive of such ways, are as follows:—

1. Human disease due to a microbe capable of thriving upon the tissues

¹ See Dr. Louis Parkes' paper on 'The Relations of Saprophytic to Parasitic Micro-organisms,' *Trans. Epidem. Soc.* 1891.

² See Dr. Sims Woodhead's paper on 'The Relation of Modification of Function of Micro-organisms to the Virulence and Spread of Specific Infective Diseases' (*Trans. Epidem. Soc.* vol. x., N.S., 1890-91). In connection with variations in virulence of infectious diseases, Dr. Woodhead refers expressly to modification of 'function' of micro-

either of man or of other animals, might in passing from lower animals to man, seem to have a new beginning for man in that it would be untraceable to previous human disease.

2. Human disease dependent upon a microbe capable of thriving either upon man, or altogether independently of living human or animal bodies—as, for instance, on living vegetable or dead animal or vegetable matter—might also, when this microbe again attacked man after a more or less prolonged saprophytic existence, have a new beginning for man.

3. Should a microbe, already pathogenic, be capable under the influence of particular environment of still further evolving pathogenically, a new variety of disease might be expected to arise; and in this way a definite disease might arise from an indefinite malady.

4. Should a saprophytic organism be capable in certain environments of acquiring pathogenic property, an entirely new disease might arise, and in this way a new and definite disease would have *de novo* origin.

In so far, therefore, as the suggestions put forward have been valid, it would appear that the question, in perhaps its most important aspect, is not one of 'spontaneous generation' but of evolution. And while 'spontaneous generation' seems more than ever untenable, evolution in connection with epidemic diseases, or more strictly their causes, is day by day forcing itself more prominently upon our consideration. The older notion of the absolute immutability of species being now indefensible, we cannot fail to be impressed by the variations from time to time exhibited by epidemic diseases; variations so considerable as to render the classification, even at times of whole epidemics, and frequently of individual cases, an impossibility, except in a most provisional and tentative way. It would seem, too, that the fixity of type of different diseases differs in degree, a circumstance which may not unreasonably be looked upon as pointing to a gradual specialisation of the causes of disease by a process of evolution. Again, we observe that certain diseases possess points of similarity to one another, and this we are now able to regard as suggestion of descent from a common stock. All these, however, are matters rather for future investigation.¹

In the meantime it must be pointed out that in differentiating 'diseases' from one another there is need for caution; and, looked at in the light of the germ theory and of evolution, it may be questioned whether, in some instances, differentiation has not already been carried too far. For the marking off of a disease as distinct and separate from others implies that it is due to a distinct and separate cause—i.e. from the point of view here adopted a distinct species of organism. But as regards many of the epidemic diseases it cannot for a moment be said that this is established. It has to be remembered that what we call 'a disease' is not a specific entity, but a mental conception based upon a rough average of certain morbid manifestations of bodily function and structure, frequently observed to occur in combination, and therefore assumed to be due to a separate cause. But there is obviously danger of attributing to separate causes effects which, although superficially different, may be nevertheless due to the same cause acting under different conditions. It is, for instance, held by some that this error has been committed in the case of membranous croup and diphtheria, and further knowledge may show

organisms, remarking that 'it will at once be accepted that the delicate metabolic changes in an organism are almost invariably brought into prominence long before the coarser morphological modifications can be observed.'

¹ See a paper on 'Specificity and Evolution in Disease,' by Dr. W. J. Collins. London 1884; also a further paper by Dr. Collins on the same subject in *Public Health*, October 1889.

it to have occurred with regard to various other diseases, as, for instance, certain forms of diarrhoea and enteric fever. Indeed, we have yet to learn that some of the diseases which, though alike in kind, differ in malignity, and which are now regarded as due to different species of organisms, are not in reality due to different biological phases of organisms of the same species. In building up an opinion as to the separate nature of any disease, not only its clinical features, but all ascertained facts relating to its natural history, must be fully taken into account. Its period of incubation, duration, period of infectiveness, complications, sequelæ, conditions of occurrence, modes of dissemination, relations (of coexistence, &c.) to other diseases, and particularly its age and seasonal incidence and geographical distribution, must be carefully compared with similar facts as to other maladies.

But a caution of another character must be offered. If it is necessary to abandon the notion of the *absolute* immutability of diseases, it is of the utmost importance to avoid falling into the opposite error of underestimating the relative fixity of type which some of them have evidently acquired. And there is nothing whatever in the suggestions put forward as to the possibility of variations in existing types of disease occurring as a result of variation of function of their microphytic causes, or of the possibility of entirely new diseases arising from the evolution of saprophytic into parasitic bacteria, in the least degree justifying the conclusion that all existing epidemic diseases may have a present day origin of some such kind. Certain diseases may, as previously pointed out, and doubtless do, depend upon highly specialised obligate parasites, incapable now of thriving except in the tissues of human hosts. Such organisms, though themselves a product of evolution, may have been evolved in the remote past under conditions of environment never since, and perhaps never again to be, reproduced.

Notwithstanding, therefore, that all pathogenic micro-organisms are doubtless—especially as regards their pathogenic function—subject, from time to time, to minor variations within the limits of the species, yet many species of such organisms may have long since attained a high degree of fixity of type, and thus exhibit now but little tendency to variation beyond such limits. That such is actually the case with regard to the microphytic causes of some of the diseases under consideration there seems ample epidemiological evidence to show.

In this connection it is important to note that occasional difficulty in tracing particular attacks of a given disease to previous cases is far from justifying hasty conclusion that such attacks have had an independent origin. The probability of such being the case must depend upon a comprehensive view of the ascertained facts with regard to the disease in question—at least, that is, pending definite knowledge of the life history of the microphyte upon which the disease depends. The multifarious ways in which infection may have been carried, and the difficulty of tracing its carriage by many such ways, must be fully taken into account. Allowance must also be made for errors of diagnosis, untreated, and concealed cases, which largely add to the difficulty of following out the causal association between successive and connected attacks. If this difficulty is met with in the early cases of an outbreak, the possibility of tracing back the outbreak to some previous prevalence in a more or less remote neighbourhood, to which it may in reality have been due, is entirely prevented.

Moreover, the presumption that a disease never in the present day arises except as a result of infection, or inoculation, from a previous case of the same disease, will certainly be strengthened if it should be ascertained that the disease in question did not occur in certain districts until introduced from

without; or, if having once been prevalent in such districts, it should have entirely disappeared after measures for the prevention of its spread by infection or inoculation, and for the prevention also of its reimportation, had been taken. Measles and rabies may be mentioned as instances in which these two arguments respectively must be allowed as having considerable weight.

Having now considered some of the aspects of the problem of disease origin, certain other general matters connected with the class of diseases under consideration must be discussed.

Diseases may be Epidemic, Pandemic, or Endemic.—As regards their more general manifestation the diseases now being dealt with are usually described as either epidemic, pandemic, or endemic.

The terms 'epidemic' and 'pandemic' respectively signify the tendency of diseases to spread more or less rapidly in a given community or over a vast area of the world's surface, in either case with a large indifference to local circumstances. In this aspect, therefore, the distinction between epidemic and pandemic disease is one of degree. The term 'endemic' signifies that a disease tends specially to abide among the inhabitants of a particular locality, and is therefore presumably largely governed by local conditions.

In the present day these distinctions are being somewhat reduced in importance, since study of the geographical distribution of disease appears to show that epidemic, and even perhaps pandemic, diseases have a preference for certain areas; while, on the other hand, increasing knowledge of so-called endemic diseases is giving greater prominence to the epidemic character of some of them, as, for instance, cholera. In so far, however, as diseases do display one or other of the characters mentioned, the probable explanation would seem to be that epidemic and pandemic diseases are due to microbes which especially thrive and multiply in living animal tissues, whereas endemic diseases are for the most part due to microbes whose habitat is more largely outside human and animal bodies, and therefore more influenced by local circumstances.

Diseases may spread by Infection, Contagion, or Inoculation.—With reference to the manner in which they are communicated from one individual to another, these diseases are also frequently spoken of as infectious, contagious, or inoculable. The three terms equally imply the transmission of infective material from one person or animal suffering from a given disease to some other person or animal in whom the disease in question becomes thereby established. But they also are intended to indicate distinctions as to the methods by which such transmission is effected, and the means by which the poison gains access to the system of the recipient. In the case of *inoculation* the poison is conveyed, either directly by actual contact with the diseased body, or indirectly by the agency of some surgical instrument or other article, from the person or animal affected to the person or animal previously unaffected, and gains access to the system of the latter through some breach of surface in the skin or mucous membrane. *Contagion*, on the other hand, means transmission of the poison by actual contact, but without any initial breach of surface in the recipient; while *infection* refers to conveyance of the poison in a more indirect way through the medium of air, water, soil, food, clothing, letters, &c., and its entrance to the system of the recipient through one of the mucous tracts, or possibly even through the skin, but in either case, again, without any decided breach

of surface. These terms are useful as broadly indicating different modes by which disease transmission may occur, but care must be exercised in applying them as exclusive modes of dissemination to the different diseases, or in basing disease classifications upon them. Certain infections are, so far as we know, only capable of being transmitted by inoculation, but others, in which inoculation is the normal method of communication, are said to be also capable of being inhaled. Further, some diseases are both infectious and inoculable.

In the present day the tendency is to discourage the use of the term 'contagious,' but if kept within proper limits, it should still have a legitimate application, and there appear no sufficient grounds for banishing it from medical literature. There is a real distinction between the transmission of a disease by actual contact, which is direct, and transmission by air or fomites, which is indirect, and contact certainly plays a larger share in the spread of some diseases than others.

Certain Diseases have special Seats of Invasion.—Certain diseases appear to have special seats or points of invasion, that is to say, the viruses upon which they depend usually attack the body by some special channel or channels, and possess little if any ability of primarily establishing themselves elsewhere. This appears to be the case with regard to the poisons of diarrhoea, cholera, and enteric fever, which make their assault upon the intestinal mucous membrane, and with regard to the poison of pneumonia, which attacks the lungs.

These facts, as Flüge points out, are of considerable importance with respect to the spread of such diseases, for it is not sufficient, in cases of the kind, that the poison should simply be brought to the body, but it must be conveyed also to the particular part of the body which is vulnerable to it. Here we find an analogy in the behaviour of the parasitic fungi which produce the diseases of plants. Some of these attack the flowers, some the fruit, and some the roots.

As regards other pathogenic micro-organisms, such as those causative of anthrax, tuberculosis, &c., the points of invasion are more numerous.

Epidemic Diseases have a Period of Incubation.—When a person has received the poison of one of these diseases a period elapses before definite and recognisable symptoms of the action of the poison manifest themselves. This interval, which is described as the period of incubation, varies considerably as regards different infections, ranging from a few hours in the case of some of them, as perhaps erysipelas, diphtheria, and scarlet fever, to weeks in the case of others, as syphilis and rabies, and even probably years in the case of leprosy. For each different infection, however, the period is comparatively constant; though here, again, variation, within certain limits, occurs in different individual cases of the same infection, and the period is more constant with some infections than others.

It is only by an accurate knowledge of the periods of incubation in the different diseases that we can be enabled to say when a person who has been exposed to a given infection may safely be regarded as having escaped attack, and can therefore be looked upon as, presumably, free from danger to others. Great care, too, is required in studying the periods of incubation in order that fallacies due to multiple or sustained exposure may be eliminated. 'The only cases in which it [a period of incubation] can be positively determined are of course those in which there has been but a single exposure to contagion; but others, in which the exposure (though repeated) began only a few days before the patient's illness showed itself, are valid as

proofs of short incubation ; and yet others in which the exposure ceased many days before he felt ill, are valid as proofs of long incubation.'¹

No detailed explanation of the incubation period can at present be given. It is often broadly stated to be the period occupied by the multiplication of the poison. This, however, if intended to signify the multiplication of micro-organisms, is a very unsatisfactory explanation when we remember, as already stated, that the incubation period extends over weeks in some diseases. Mere multiplication of micro-organisms can hardly be regarded as requiring such lengthened periods of time, and there can be little doubt that some far more complicated process has to be gone through prior to the general infection of the individual. What this process is remains for future study, and it has to be remembered that the process may possibly be a very different one for different diseases.

The following suggestions may be put forward as perhaps worth consideration.

1. If it is a fact that bacteria are incapable of directly penetrating healthy mucous membrane, as Flügge seems to affirm, some little time would doubtless be required for bringing about the local changes at the seat of lodgment, which are necessary for the entrance of the bacteria.

2. Certain diseases at least appear, as already said, to be due to the action of chemical poisons produced by the micro-organisms causative of them, rather than to the simple presence of the organisms themselves. As regards such diseases, a time—and a time which may be different for different infections—is perhaps required for the accumulation of a sufficient quantity of the poison to produce the several phenomena of the disease.

3. The excessively long incubation periods, as in rabies and leprosy, would seem to suggest either some peculiarity in the life history of the micro-organisms upon which such diseases depend, or perhaps the necessity for some gradually effected change in the tissue condition of the recipient prior to the full operation of the pathogenic properties of the micro-organisms. Or, again, it may be that the particular poison locally produced is also locally stored up, and later on discharged from the local tissues and disseminated throughout the system.

Protection.—A very important phenomenon in the natural history of these diseases is that certain of them confer protection or immunity against future attacks upon persons who have once suffered from them. The degree of protection thus imparted varies considerably, however, in different diseases, and in different individuals suffering from the same disease. It also differs, too, according to the age of the individual at the time of primary attack. As regards some diseases it is usually stated that the protection is of life-long duration. This doubtless often is so ; but here it becomes interesting to ask how far the life-long protection, which apparently results from a single attack of one of these diseases, may be due to occasional subsequent exposure of the individual to the same infection, and consequent renewal of protection. It is conceivable, for instance, that a person may derive absolute protection for a certain period. Towards the close of this period he may be again exposed to the same infection, but being still partially protected by the antecedent attack, his re-infection is not followed by conspicuous manifestation of the disease. He may suffer perhaps from a mere undefined malaise which escapes serious attention, but nevertheless serves to renew his protection. In this manner the protection of such diseases as measles and scarlatina may perhaps be renewed from time to time. It is by no means

¹ *The Principles and Practice of Medicine.* O. Hilton Fagge, M.D., F.R.C.P. Edited and completed by P. H. Pye-Smith, M.D., F.R.S. 2nd edit. i. 27.

uncommon to find, for instance, persons in infected households, who have previously suffered from scarlet fever, again exhibiting sore throat and malaise. This doubtless means a re-infection, and renewal of protection; and it becomes an important question how far such persons, while passing through these later mild attacks, are capable of infecting others. In re-vaccination we have experimental confirmation of the renewal of protection.

Another interesting question is whether any degree of protection is capable of being transmitted by heredity. The exceptional virulence of measles when implanted upon virgin soil, as in the case of the Fiji outbreak, suggests the possibility of this being the case, notwithstanding that the mortality of that outbreak was doubtless largely due to a neglect, based upon ignorance and superstition, of all necessary precaution.

The protection afforded by some of these diseases is, however, far less than that afforded by others. Of certain of them, indeed, it is said that they do not protect at all, and that some, as erysipelas, even predispose to future attacks. On the other hand, it has been suggested that all microbic diseases protect for a certain period, though in some instances only a short one; that otherwise it would be difficult to understand such diseases ending except in death, for it has been thought that in the absence of any protection afforded by the disease process a person once infected would keep his disease going perpetually by auto-infection. However this may be, there can be no question that for many people at least, the protection, if any, afforded by an attack of some of these diseases is exceedingly slight, and the question might be raised, as for instance in the case of erysipelas, how far this is due to the disease itself, and how far to peculiarity of tissue of persons especially subject to it. The problem is admittedly a difficult one, and time and observation are wanted for its solution; moreover, it is complicated by the apparent ability of certain infections to lie dormant in individuals, and to recrudescence in them at uncertain intervals. As to this, there seem no *a priori* reasons why microbes should not remain in the system, held in check by the conditions which have brought their obvious manifestations to an end, and consequently conferred a temporary protection on their host. Ultimately the forces may become reversed, and the microbes gain a second ascendancy, to be subsequently in similar manner reduced to temporary quiescence. A thesis of this sort would seem necessary for explanation of the recurrent attacks of ague from which persons frequently suffer years after removal from malarious districts.

Original liability to attack by epidemic disease varies in different individuals, some appearing by nature almost immune, while others exhibit a marked degree of susceptibility. Such contrasts, there can be little doubt, are due to differences—sometimes hereditary and sometimes acquired (in other ways than by attack)—in the tissue condition of the several individuals—and especially perhaps in the condition of the tissues (mucous membrane, &c.) at the points of invasion special to the different diseases. The marked tendency to phthisis observed in certain families probably affords an instance of transmitted liability to infection, while the well-known predisposing influence of unwholesome conditions of life, overcrowding, starvation, &c., illustrates acquired increase of susceptibility to certain infections. In this connection it is worthy of note that the general experience of epidemiologists to the effect that conditions tending to lower vitality predispose to attack by certain diseases, seems recently to have received indirect experimental support.

Susceptibility to certain infections is also no doubt increased by other conditions, such as cold and damp.

Influence of Age and Sex upon Original Liability to Attack.—The age and

sex incidence of the different diseases, as judged mainly by mortality statistics, will be given in the appropriate sections. Speaking generally, it may be said that, as regards certain diseases which mainly affect the young, the age-incidence observed is no doubt in part due to the fact that since so large a number of the individuals of a community are attacked during childhood, the protection thus acquired diminishes the number of adults susceptible to attack. This, however, cannot be regarded as the sole factor, but it probably serves to exaggerate, or perhaps modify, the true age incidence, which there can be little doubt is of a deeper and more fundamental character. Some diseases for instance, on the contrary, appear to pick out adolescents or adults in preference to children, while some, again, of those diseases which especially affect children, as scarlatina, rarely attack very young infants.

Sex incidence in adults is no doubt in part regulated by occupation—that is to say, according to the different occupations of the two sexes, so generally is their chance of exposure to certain infections. At the same time it is now tolerably certain that, independently of occupation, the actual susceptibility of the two sexes varies as regards some diseases.

Influence of Season upon the spread of Epidemic Diseases.—It is well established that some of the diseases under consideration exhibit a decided preference for certain seasons of the year. These seasonal variations are no doubt largely due to climatic influences upon the life phases of the micro-organisms upon which the different diseases depend; that is to say, each species of micro-organism probably has its particular season of the year in which the meteorological conditions most favourable to its development, at all events as regards pathogenic property, are at a maximum. Seasonal variations may, however, be partly, and in the case of some diseases largely, due to the effects of season on the recipients—either the direct effects, as influencing susceptibility, or the indirect effects as modifying the habits of life in such a manner as to facilitate the process of infection from case to case.

Periodicity in Epidemic Diseases.—In addition to the seasonal variations these diseases prevail more extensively in some years than others. This, of course, may be explained, and in part correctly explained, in the same manner as seasonal variations; that is to say, it may be concluded that unusual prevalence is partly, and sometimes perhaps entirely, due to climatic and other conditions which are unusually favourable to the development and dissemination of pathogenic micro-organisms. But as regards the varying prevalence of some of these diseases at least, it is becoming increasingly evident that there is a more or less regular periodicity—a cyclical character, in fact.

This subject was dealt with by Dr. Arthur Ransome,¹ and has again, quite recently, been studied by Dr. B. A. Whitelegge.

Dr. Ransome pointed out that a study of 'the track taken by any of the more common and fatal of the infectious diseases throughout a long series of years' showed them to 'observe definite periodic times or cycles,' which he described as a succession of waves, the periods covered by the waves differing for different diseases. He also showed that as regards some maladies, 'such as scarlet fever, whooping cough, and perhaps also small-pox,' if a sufficient number of years be taken in review, which is rendered possible by the Swedish mortality statistics, there is indication of 'certain larger as well as smaller disease waves.'

Dr. Whitelegge corroborates Dr. Ransome as regards the occurrence of

¹ *Proc. Lit. and Phil. Soc.*, Manchester, Jan. 27, 1880; and *Trans. Epidem. Soc. Lond.*, vol. I, N.S., 1881-82.

the larger and smaller cycles, and by a further study of the facts concerning them, as well as by an extension of his survey to other disease periodicities, he is led to formulate certain general propositions. Disease undulations or waves, he considers, are of two essentially different kinds—the accidental, or, more correctly, ‘superadded’ waves, and the fundamental or true cycles.

The characteristic of the ‘superadded wave’ is that it is not attended by any regular and progressive increase, and subsequent decrease, of virulence. It is a wave of mere prevalence, and is probably but a reflex of changes in the environment. The following Dr. Whitelegge regards as instances of ‘superadded waves’ :—

(a) The weekly wave, which he has shown to occur at Nottingham with respect to scarlet fever. This he considers due to the diminished opportunity on the Sunday for the spread of the disease by school attendance.

(b) The annual seasonal wave, as regards which he finds that the rise in the number of cases does not carry with it a proportionate rise in the number of deaths, nor is the fall in the number of cases attended with an equivalent fall in the number of deaths.

(c) Epidemics caused by specifically contaminated milk or water, inasmuch as they are due to artificially increased opportunities for the spread of infection.

The true fundamental cycle, on the other hand, is characterised by an increase both of prevalence and severity.¹ A typical instance is found in the long cycle extending over a considerable number of years. Study of such cycles shows them to embrace, and indeed to be made up of, a number of the ‘short’ cycles (quinquennial, &c., according to the particular disease), each of which short cycles displays an increase both of prevalence and severity, as compared with its predecessor, until the maximum of the ‘long’ cycle is attained. Then follows a decrease both in the prevalence and severity of each successive short cycle. But in regard to scarlet fever, at all events, these shorter five- or six-year cycles, which make up the larger wave, are themselves demonstrably truly cyclic in character. They are waves of increasing, followed by decreasing, severity. Whether the same can be affirmed of the biennial rhythms of measles seen in certain towns is doubtful, the available statistics being insufficient to determine the point.

The ‘true’ cycles might conceivably be due to corresponding cyclical changes in environment, such as meteorological changes; but Dr. Whitelegge considers it likely that they are of a more fundamental character, probably associated with microphytic evolutionary processes.

MEASLES

Synon.: *Morbilli*; *Rubeola*. Fr. *Rougeole*; Ger. *Masern*; It. *Rosolia*.

History and Geographical Distribution.—There can be little doubt that measles is a disease of ancient origin, though for many generations it was confounded with other maladies, notably small-pox and scarlatina. With small-pox it continued to be confounded until the sixteenth century, and with scarlet fever till the days of Sydenham. Nevertheless it is apparently referred to as ‘hasbah’ in the writings of the Arabian physicians, who

¹ The increase in severity being indicated by a rise in the case mortality, and by the tendency of the disease to attack people at ages usually more or less spared by it as well as persons protected by previous attack. See Milroy Lectures delivered since the above was written. *Lancet*, February 25, and March 4, 11, and 18, 1893.

regarded it (along with scarlet fever, no doubt) as a variety of small-pox. During the Middle Ages the disease would seem to have been widely prevalent in Europe and Asia, generally, according to the accounts, in association with small-pox.

The original seat or native home of measles is unknown, but nowadays the disease has a world-wide distribution, and, now as formerly, in localities not prone to it, its presence might doubtless always be traced to introduction from without. According to Hirsch,¹ it has been four times introduced into Iceland (1664, 1694, 1846, and 1868), and four times into the Farøe Islands (1781, 1846, 1862, and 1875), those localities remaining free from it during the intervals; it reached the western hemisphere 'soon after the arrival of the first European settlers;' it appeared in the Hawaiian Islands in 1848; in the Australian continent, Tasmania, and New Zealand in 1854. There appear to be no grounds for supposing that it had existed in these countries prior to its importation from without, and it would thus seem probable that its general distribution throughout the world had been brought about by human travel and colonisation.

Measles is now well established, occurring in frequent epidemics, throughout most of Europe, Asia, America, and those parts of Africa of which we have definite information on the subject.

In England it is everywhere from time to time prevalent.

During the decennium 1871-80 the registration counties in which the mortality from measles among children under five years of age was above the general average were, beginning with the highest, Lancashire, Devonshire, Monmouthshire, London, Cumberland, Leicestershire, and the West Riding.² It is interesting to note that the high measles mortality was not confined to densely populated counties, and, therefore, cannot have depended entirely upon the increased facilities for spread which aggregation affords. It also appears that the geographical distribution of measles in this country differs considerably from that of diphtheria, and to some extent from that of scarlet fever, though, as regards the latter, the question is complicated by hospital isolation, which has not operated in the case of measles.

Periodicity.—At different times measles has tended to become widely prevalent over large tracts of country, assuming almost a pandemic character. No regularity in the times of appearance of these major epidemics seems to have been made out; and as regards the lesser epidemics of a more local character, the inter-epidemic interval varies greatly. Thus the intervals between outbreaks have been variously stated as from two to six years for different places and by different observers. In view of these discrepancies, and also of the different length of interval between epidemics of measles in particular places, Hirsch disbelieves in any definite periodicity. Without drawing the line too fine, however, it may be said that in large communities the disease tends to occur epidemically at intervals of from two to four years, disappearing more completely between these epidemic visitations than is the case with scarlatina and some other diseases; and that in small communities, especially among the populations of rural districts, the intervals are less regular, and longer.

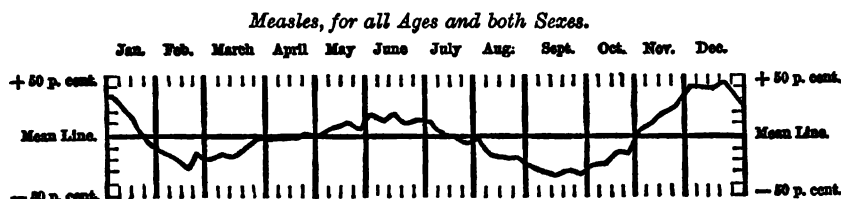
The Influence of Climate.—Upon a cursory survey of the facts it would at first sight appear that measles is uninfluenced by climate; Hirsch, indeed, in view of the practically universal distribution of the disease throughout

¹ *Handbook of Geographical and Historical Pathology*, 1888, i. 156, by Dr. August Hirsch. Translated by Charles Creighton, M.D. New Sydenham Society.

² Forty-seventh Annual Report of the Registrar-General, p. xiv.

the world, concludes that its occurrence 'is quite independent of *climatic influences*.' It is doubtful, however, whether such an inference can be legitimately drawn from the universality of its distribution, which may simply mean that the influence of climate is masked by the high infective power of the disease and other conditions, but some climates may nevertheless be more favourable to its occurrence than others. That climate is not without influence on measles would seem indicated by the consideration, which Hirsch himself points out, that 'wherever it (measles) has occurred, the influence of *certain kinds* of weather, depending on the *seasons*, has been observed to have a marked effect upon the frequency of its outbreak and the extent of its prevalence.'

And of Season.—This influence of season appears to have been everywhere observed, and, taken generally, the warmer periods of the year have been found least to favour the prevalence of measles. In temperate zones, of 580 epidemics of measles in Europe and North America which Hirsch records, 389 occurred in the colder, against 191 in the warmer months. And the same thing has been observed in the tropics. In this country the effect of season upon urban measles has been studied by Dr. Buchan and Sir Arthur Mitchell, and by the Registrar-General. The former, whose conclusions are based upon the mortality statistics of London for the thirty years 1845–1874, as recorded in the weekly returns of the Registrar-General, give the following diagram¹ as illustrating the weekly variations in the London mortality of measles.



In reference to this diagram they make the following remarks: 'The measles curve is remarkable in showing a double maximum and minimum during the year, the larger maximum occurring in November, December, and January, and the smaller in May and June; the larger minimum in August, September, and October, and the smaller in February and March. The most rapid fluctuation takes place in the fall from Christmas to the middle of February, the weekly deaths falling from forty-two to twenty-one. This is one of the steadiest curves from year to year, both maxima being well marked in nearly every one of the thirty years in December and June respectively, and the minima also being well marked.'

It must of course be remembered that the curves under consideration are based on mortality statistics, and that for indication of the development of the measles to which the data refer, the curves should be moved some three or four weeks back throughout the year, to allow for the period of incubation and the period of illness prior to death.

Mortality.—Apart from sex and age influences, which will be dealt with

¹ We are indebted to the courtesy of Dr. Buchan and Sir Arthur Mitchell for permission to reproduce this and other diagrams from their paper on 'The Influence of Weather on Mortality' (*Jour. Scottish Meteor. Soc.*, July 1874–July 1875). The Registrar-General in his Annual Summary for 1890 has published a more recent seasonal mortality curve for measles, based upon the deaths from measles in London during the fifty years 1841–1890. The Registrar-General's curve for the longer period, however, bears a very close resemblance to that given above.

later, measles mortality must be considered from two points of view, e.g. the actual mortality or total deaths, and the case mortality (fatality) or proportion of deaths to attacks. The actual mortality, moreover, to be intelligible, must be reduced to some standard of measurement, as, e.g. the number of deaths per thousand or million of the population. The table below shows the measles mortality in England and Wales for each year from 1888 to 1890, with the exception of the four years (1848-46) for which the causes of death were not extracted by the Registrar-General.

It will be seen that in this country measles is responsible for the deaths of from 6,000 to 14,000 persons each year. It will also be seen that there has been a decided rise in the mortality from measles during the last ten years. How far this is the result of any special advantage measles has of late years gained over us, or how far it is merely the expression of a cyclical character of the disease, is an interesting problem.

Fatality.—The case mortality of measles is capable of varying within very wide limits. By some observers it is stated to average 5, by others 10, per cent. of attacks, while there is plenty of evidence that the fatality of this disease may in a given epidemic range as low as 2 per cent., and in another epidemic be as high as 40 or 50 per cent. Probably there are two main factors in inducing high fatality, the one belonging to measles itself—extra intensity of infection—the other bound up with unfavourable conditions—overcrowding, bad and deficient food, fatigue—of the population invaded; and of course, where these factors are concurrent, the death toll must needs be high. In this way may be explained the high measles fatality from time to time observed among troops on active service, and among prisoners of war. Thus

TABLE I

Year	England and Wales		Average annual death-rate per million living for each quinquennial period	Year	England and Wales		Average annual death-rate per million living for each quinquennial period
	Total deaths	Death-rate per million living			Total deaths	Death-rate per million living	
1838	6,514	426	539	1865	8,562	405	457
1839	10,937	705		1866	10,940	511	
1840	9,326	591		1867	6,588	304	
1841	6,894	433		1868	11,630	528	
1842	8,742	542		1869	10,309	464	
1843	No data; causes of death not extracted by Registrar-General		403	1870	7,543	335	373
1844				1871	9,293	408	
1845				1872	8,530	368	
1846				1873	7,403	316	
1847	8,690	507		1874	12,235	517	
1848	6,867	395	399	1875	6,173	257	385
1849	5,458	311		1876	9,971	408	
1850	7,080	398		1877	9,045	366	
1851	9,370	521		1878	7,765	310	
1852	5,846	320		1879	9,185	362	
1853	4,895	266	425	1880	12,328	478	413
1854	9,277	498		1881	7,300	280	
1855	7,354	391		1882	12,711	483	
1856	7,124	373		1883	9,329	350	
1857	5,969	310		1884	11,324	419	
1858	9,271	476	457	1885	14,495	533	468
1859	9,548	485		1886	12,013	436	
1860	9,557	479		1887	16,765	602	
1861	9,055	460		1888	9,784	347	
1862	9,800	481		1889	14,732	518	
1863	11,349	550	457	1890	12,386	439	468
1864	8,323	397		1891	12,673	436	

in the American Civil War, a mortality of 20 per cent. of those attacked by measles is said to have occurred in two large hospitals. 'In Paris during the siege (January 1871), out of 215 of the Garde Mobile who took measles, 86, or 40 per cent., died; and the mortality reached very nearly the same figure among the French troops who returned to Paris after the Italian War, 40 out of 125 cases dying in one hospital (whose sanitary condition was bad) with severe intestinal symptoms.'¹ Again, according to Masterman, 'at the beginning of the Brazilio-Paraguayan War, an epidemic of measles swept off nearly a fifth of the National Army in three months, not from the severity of the disease, for I treated about fifty cases in private practice without losing one, but from want of shelter and proper food.' So, too, among many uncivilised communities the mortality from measles imported for the first time has been excessive, in some cases whole tribes having been swept away. As instances may be mentioned the outbreaks on the banks of the Amazon, at Hudson's Bay, the Cape, Tasmania, Mauritius, and the Fiji Islands. The excessive mortality in such outbreaks has often been attributed to the fact that the sufferers in question presented a virgin soil to a very intense virus. This may have been, and probably was, one of the factors which contributed to the result; but from official and other reports it appears that important factors were neglect, superstitious practices, and unwholesome conditions of life. Among the small proportion of persons who in some of these outbreaks were treated in hospitals, and were thus more favourably circumstanced than the majority, the mortality was not extravagant. Referring to the terribly fatal epidemic at Fiji, Dr. Squire says: 'The excessive mortality resulted from terror at the mysterious seizure and the want of the commonest aids during illness.' Numbers of the people, it appears, either kept closely shut up in their unventilated houses, or rushed into the streams during the height of the illness. This epidemic carried off from one-fifth to one-fourth of the entire population, but of 148 native constables who were under the treatment of Dr. Cruikshank, only nine deaths were reported, 'most of these resulting from evasion of needful precautions.'²

Influence of Race.—There is not sufficient evidence as to how far difference of race, as such, influences liability to attack by measles or mortality from that disease. Neither can anything definite as yet be said as to the influence of sex and age so far as liability to attack is concerned. But both sex and age appear to influence mortality, and therefore probably also liability to attack.

Of Sex.—Speaking for England and Wales, the rate of mortality among children under two years of age is greater for males than females. At all age-periods above two years it is greater among females. Owing to the fact of the main incidence of the disease falling upon the early years of life, the mortality is, on the whole, somewhat greater among males than females.³

Of Age.—As to age in this country, about 98 per cent. of all deaths from measles occur among children under ten years of age, 90 per cent. among those under five, 75 per cent. among those under three, and 60 per cent. among those under two, the maximum mortality as well as the maximum rate of mortality being in the second year of life. This very marked incidence of measles upon the young is no doubt largely owing to the frequent epidemicity of the disease, which results, at any rate as regards towns, in a very large proportion of persons being attacked during childhood, either to die then or survive protected against further attack. For where measles

¹ Hirsch, *op. cit.*, i. 168.

² Paper read before the Epidemiological Society by Dr. Squire, *Med. Times and Gas.*, 1877, p. 323.

³ Fifty-first Annual Report of the Registrar-General, p. xxiii.

has been introduced among unprotected communities, as in the case of the Farøe Islands, it can attack most persons brought in contact with it, irrespective of age. Even in this country, adults who have hitherto escaped the disease are often seen to take it readily if exposed to the infection, and some of them suffer from it severely. Notwithstanding all this, however, it is extremely likely that measles, although capable of freely attacking unprotected persons of any age, yet has an affinity for the early years of life. And there seems no doubt at all that the disease is more dangerous in delicate children and in infants under four or five years of age (excluding the first six months) than in the latter stages of childhood.

Cause and Mode of Dissemination.—Measles is unquestionably due to a poison which is capable of being transmitted from the sick to the healthy, and of multiplying in the system of the recipient. Hence the probability of this poison being a micro-organism, though no such organism has at present been demonstrated to stand in a causal relation to the disease.

Whatever may have been the remote origin of the disease, there is no evidence that it now ever occurs except as a result of direct or indirect infection from a previous human case, and all that has been said with regard to its general history points to this as the main, if not the only, mode of its occurrence in modern times. In view, however, of recent evidence in the case of some other diseases, the possibility of measles affecting the lower animals as well as man, and being communicable from them to him, must not be lost sight of.

The poison of measles is held to be given off by the breath and mucus—possibly also by desquamating cuticle, though this is less certain. The recipient is no doubt, as a rule, infected through the respiratory tract. Generally the disease is transmitted directly from case to case; but the poison is probably capable of being air-borne, especially in ill-ventilated rooms, to a greater extent than has often been supposed. The poison also can cling to surfaces, and so may be carried by fomites. There is at present no evidence of its being conveyed by water, milk, or food.

The infection is freely given off during the early catarrhal stage of the disease, before the eruption appears; throughout the illness, and to some extent during convalescence. The periods of greatest infectiousness, however, are during the pre-eruptive stage, and while the rash is present. This early infectiousness of measles is a very potent factor in the dissemination of the disease, for during its pre-eruptive stage, children, though in a highly infectious condition, are commonly allowed to mix with others under the impression that they are simply suffering from common colds. In this way there can be no doubt that schools, churches, and other places of public resort play an important part in the spread of measles.

Periods of Incubation and Infectiveness.—In the naturally acquired disease the usual period of incubation is probably about eleven days, though there is not uncommonly a variation of several days on either side of this. Panum, in the Farøe Islands, found that the *rash* usually appeared on the fourteenth day after a single exposure. Goodhart¹ says that, 'though liable to modification within limits of three or four days either way, the incubation period centres round ten days.' According to Finlayson,² who has collated the opinions of recent authorities on this and allied matters, Murchison stated the incubation period as ten to eleven days; Vacher as three to seventeen, usually ten; Squire as eight to eighteen, usually eight to twelve;

¹ J. F. Goodhart, M.D., F.R.C.P., *Diseases of Children*, 2nd edit., p. 128.

² James Finlayson, M.D., the *Glasgow Med. Jour.*, May 1889.

Richardson ten to fourteen; Stephenson fourteen days; Clement Dukes ten to fourteen, usually eleven; Newsholme ten to fourteen, usually twelve to fourteen.

As has been said, the infective period, i.e. during which the patient is capable of infecting others, must be considered as at all events beginning with the very earliest symptoms, some days before the rash appears, and it perhaps extends through most or all of the incubation period. The subsequent duration is variously stated by different observers as somewhere between two and four weeks from the date of appearance of the rash, though it is generally agreed that infection is usually over by the end of the fourth week, provided all cough and desquamation have ceased.

Protection.—One attack of measles usually confers a lasting protection against future attack. This was illustrated in the outbreak of 1846 in the Farøe Islands, under circumstances which gave to the experience the conclusiveness of a carefully planned experiment. The disease had been unknown in the islands since 1781, a period of sixty-five years. During the six months following its reimportation in 1846, according to Panum,¹ no less than 6,000 persons, out of a total population of 7,782, contracted the disease. But every one of the old persons who had been attacked during the previous visitation escaped in 1846. Further, that their immunity was not simply due to age was evident from the fact that the other old persons who had been alive in 1781, but had then escaped, now took the disease, practically without exception. Second attacks, however, do sometimes occur. Occasionally also relapses are met with.

RÖTHELN

Synon.: *German Measles, False Measles, Epidemic Roseola, Rubeola, Rubeola sine Catarrho, Rubella*; Fr. *Rougeole*.

About the middle of the eighteenth century—that is, shortly after the complete separation of scarlet fever from measles—mention began to be made in medical literature, under the titles of ‘roseola’ in England and France, and ‘rubeola’ in Germany, of a malady seemingly different from scarlet fever and from measles, but having some of the characters of both.² From that time to the present, outbreaks have now and again been met with which, from a clinical view, it has similarly been found difficult, or impossible, to regard as either true scarlet fever or measles, and accordingly a belief in the existence of a third exanthem of this kind has gradually gained ground, until at present it is very generally held.

Many have regarded this complaint as a hybrid of scarlet fever and measles, but the view most generally adopted by modern authorities is that, although superficially resembling both scarlet fever and measles, it is an entirely distinct and specific disease.

Apart from its special clinical features, for which the reader must be referred to works on general medicine, this disease is held by such authori-

¹ *Archives Générales de Médecine*, April 1851.

² According to Prof. Thomas (*Ziemssen's Cyclopædia*), some affirm that an ailment of this kind was referred to by the Arabian physicians as ‘hhamikah.’ Prof. Thomas also refers to the possibility of the ‘benignant “Rossalia epidemics” of earlier centuries’ having been rötheln. But this, he says, cannot be ascertained owing to lack of accurate descriptions.

ties to occur in epidemics, to cause little or no mortality, to be usually unattended by sequelæ, to protect against itself, but not against either scarlet fever or measles, 'nor do attacks of either of these diseases in any way modify the liability to this one.'¹ It is said to spread by infection, but the infection is stated to be 'apparently less active than that of measles, and less persistent than that of scarlet fever, for more escape in a house or school during an epidemic of rubeola than during one of measles, and cases seldom occur after an interval of cessation.'² Its period of incubation seems to be somewhere about a fortnight; and the period of infectiveness from two to three weeks. According to Dr. Squire, rōtheln is, like measles, 'contagious even before the rash is thrown out. . . .' In this country, or at least in London, it is apparently most common from March to June.³ In an epidemic of what was apparently rōtheln at Calcutta, recorded by Surgeon-Major McLeod, the cases occurred in the hot, rainy, and cold seasons, but those in the cold season presented 'severer throat symptoms and a more acute character generally.'⁴ As regards age there seems difference of opinion: some observers describe it as most common among adolescents and adults, while Professor Thomas, on the other hand, describes it as 'especially a disease of childhood, attacking indiscriminately boys and girls, older and younger children down to sucklings.' He, however, adds that 'adults up to about forty years are not unfrequently affected.' Sex is said to have little influence on its incidence. It is described as occurring in the British Isles, on the Continent, in Egypt, in India and America. 'Necessary relations between epidemics of rubeola and those of other infectious diseases,' according to Thomas, 'do not exist.'

The consensus of modern authorities as to the existence of a malady of the above sort seems to be too strong to allow of serious doubt upon the point; but to the independent mind a perusal of the literature of the subject may perhaps justify suspicion that the attitude adopted by some authorities upon this matter is somewhat too exclusive and absolute. It has to be noted that some of the epidemics which have been recorded as examples of this disease, by apparently careful observers, have differed conspicuously from the characters now generally regarded as indicative of the malady in question. In some of such epidemics a considerable mortality seems to have occurred, and in other respects the disease has differed from that now regarded as true rōtheln. It is easy, of course, to assert that such outbreaks as do not exactly coincide with the modern conception of rōtheln are but unrecognised outbreaks of either scarlet fever or measles, but there is a decided suspicion of *petitio principii* about such a procedure. It may, for instance, be that rōtheln, admitting it to be a separate disease, is susceptible, under certain conditions of environment, of greater variations than it is customary to admit—indeed, more recent experience seems in favour of this being the case. Or, again, it may be that in addition to the separate malady having the characters usually ascribed to rōtheln, there are other maladies also capable of separation from measles and scarlet fever. In a sense, too, the separate and specific character of rōtheln is perhaps exaggerated, for it seems now to be usually held that it has no definite re-

¹ William Squire, M.D., article on 'Rubella,' *Quain's Dictionary of Medicine*, vol. ii. Dr. Clement Dukes also found that thirty-nine out of sixty-three cases had previously had measles.

² Fagge, *op. cit.*, i. 224.

³ See Dr. Hopwood's article on Rōtheln in *Fowler's Dictionary of Practical Medicine*.

⁴ 'On the Prevalence of Epidemic Roseola in Calcutta,' by Kenneth McLeod, A.M., M.D., F.R.C.S., *Epidem. Soc. Trans.*, 1884-85.

lationship to ordinary measles. This of course may be so, but the two diseases would certainly seem to bear a strong family resemblance, and it would appear not unlikely that r  theln, although a relatively separate and moderately stable malady, may still be genetically related, in the sense at least of descent from a common stock, to measles or scarlet fever—probably the former. Such other epidemics, too, as those above referred to, which seem to have differed somewhat from ordinary scarlet fever and measles, as well as perhaps from r  theln, may possibly have been still other descendants from the common stock; or they may perhaps have been even more closely related to measles or scarlet fever, from one or other of which maladies they may have been recently evolved under special circumstances of environment.

Neither would there seem any inherent impossibility in the notion of some of such disease varieties, including r  theln, being of a hybrid character, as has frequently been suggested.

It might be suggested as a criticism upon the hybrid theory that in bacteria sexual processes are absent. This, however, can by no means be safely affirmed.

Neither scarlet fever nor measles appears decidedly to protect against r  theln, nor does that disease, it is said, confer any immunity against them. But it must be borne in mind that protection is a relative matter; and it does not follow, because in one epidemic of r  theln a certain number, and even a comparatively large number, of persons attacked have previously suffered from measles, that the escape of some other persons in such epidemic may not have been due to protection afforded by previously acquired measles. If it is true that 'more escape in a house or school during an epidemic of rubeola than during one of measles,' this may possibly, though of course not necessarily, be due to the cause referred to. Lastly, it would be premature to allege that diseases descended from a common stock must necessarily protect against each other.

Whatever its origin, however, there seems no doubt, as already said, that cases, both sporadic and epidemic, of an ailment, having some of the appearances of measles and some of those of scarlet fever, are frequently met with in this and other countries; but at present little can be said with confidence as to their epidemiological characters, and the whole subject requires careful study.

Even Professor Thomas, one of the most distinguished and ardent supporters of the separate and specific character of this disease, remarks: 'There are few diseases in regard to which opinions vary so much as about rubeola, or rather about that which is designated as rubeola by various authors. The work of separation and discrimination, which has by degrees given us a definite conception of the other acute exanthems, has by no means reached its final result as regards this.'

There is one matter, however, which, from a public health point of view, is important, and that is the certainty that in practice 'r  theln' is too frequently pressed into the service as a cloak to difficulty in diagnosis. "German measles," says Dr. Goodhart, 'is a term which is terribly abused. A doubtful rash makes its appearance, and the medical man, instead of saying he is not certain of its nature, calls it German measles. "Then it is not scarlatina?" ask the parents. "No," says the doctor; and the parents, thinking nothing of measles, take no precautions. Any hospital physician sees many such cases, and knows also very well—considering the rarity of the actual disease—that, when he has to do with what is called German measles, it is more probable than not that the nature of the malady is scarla-

tinal, and that in this direction he must look for the explanation of whatever sequelæ he may meet with.' The disastrous consequences which follow the course above indicated need not be pointed out.

SCARLET FEVER

Synon.: *Scarlatina*; Fr. *Scarlatine*; Ger. *Scharlachfieber*; It. *Febbre Scarlatina*.

History and Geographical Distribution.—The complete differentiation of scarlet fever was set on foot by Sydenham, whose description of the disease as 'febris scarlatina' was based upon experiences of it as it occurred in London from 1661 to 1675. In this connection it is interesting to note that Sydenham makes no mention of sore throat as one of the symptoms of scarlet fever (Fagge). Subsequently the distinct and separate character of scarlet fever was more fully established by Heberden, Fothergill, Willan, and Withering. But though not differentiated until the latter half of the seventeenth century, scarlet fever was described by Ingrassias as early as 1556, and somewhat later by Döring, Sennert, and other writers. According to Hirsch,¹ 'the oldest notice relating probably to an epidemic of scarlatina dates from Sicily, 1548;' but Hirsch is of opinion that the disease was prevalent on the continent of Europe long before the period from which we derive the earliest medical accounts of it.

Prior to the time of Sydenham, scarlet fever had been regarded as a variety of measles, and even Morton, a contemporary of Sydenham, held to this view and described it as '*morbilli confluentes*.'

Though it is evident from what has been said that scarlet fever has long been prevalent in Europe, its introduction into many other parts of the world seems, as in the case of measles, to have taken place in comparatively recent times. Thus it was apparently unknown in North America until about 1735, and it did not become prevalent in South America until 1830. It first reached Australia and Polynesia in 1848 (Hirsch).

At the present day scarlet fever is most widely distributed in the north-western countries of Europe. In Russia also it appears to be somewhat widely prevalent. It occurs, though apparently to a lesser extent, in Italy, Turkey, Greece, and some of the Mediterranean islands. It is widely diffused over North and parts of South America, but it does not appear to have been frequently prevalent in Australia; and, although often imported, it has never firmly established itself in Asia, except along the coast of Asia Minor, or in Africa except perhaps in Algiers. As regards the broad geographical distribution of scarlet fever, Hirsch remarks that 'the area of diffusion of scarlet fever is much smaller than that of small-pox or of measles; that the continents of Asia and Africa, which, as we have seen, are among the chief seats of these two diseases and especially of small-pox, have been visited by scarlet fever at the utmost to a very small extent, allowing even for imperfections of record.'

As regards England, the Registrar-General points out in his 47th Annual Report (page xiv) that for the years 1871–80 the mortality from scarlet fever among children under five years of age was above the general average in the following counties, beginning with those having the highest mortality: Durham, Lancashire, Northumberland, West Riding, Staffordshire, War-

¹ See Hirsch, *op. cit.*, i. 172.

wickshire, Cheshire, South Wales, Worcestershire, Derbyshire, and Cumberland. 'It thus appears,' he remarks, 'that even after due correction is made for age distribution, scarlet fever is for some reason or other most destructive in the industrial, and especially the mining counties. . . . The explanation that naturally suggests itself is that probably the population in industrial and mining counties live in more than averagely close aggregation, and that the spread of infection is thus facilitated.'

'If, however, this were the true and complete explanation we should expect the geographical distribution of other infectious diseases to tally with that of scarlet fever.' But this, the Registrar-General remarks, 'is not true as regards diphtheria; nor does it seem altogether true as regards measles.'

The contrast between the geographical distribution of diphtheria and scarlet fever is a matter of very great interest, and has also been especially pointed out by Dr. G. B. Longstaff,¹ and by Dr. Edgar Barnes,² who has published maps showing the marked difference between the distribution of diphtheria and scarlet fever during the years 1860-86.

Periodicity.—Although more or less constantly present in large communities in which it is firmly established, scarlet fever at times assumes a wide epidemicity. It extended, for instance, over Denmark, England, Germany, and France in 1825-26, and again, as well as over Ireland and Russia, in 1892-95; over Germany, Denmark, England, and Scotland in 1846-49; over North America in 1821 and 1851, and South America in 1831-37 (Hirsch). From a study of the Swedish mortality records Dr. Arthur Ransome considers that, as regards scarlet fever mortality, 'not only a short cycle of four to six years may be traced, but also a long undulation of fifteen or twenty years or more; which may be likened to a vast wave of disease upon which the lesser epidemics show like ripples upon the surface of an ocean swell.'³

Dr. Whitelegge finds that at Nottingham scarlet fever exhibits a weekly cycle, the notified cases falling to a minimum upon the Wednesday. This he regards as probably due to the diminished likelihood of infection through the agency of school attendance upon the Sunday.

Mortality.—The following table, compiled from the annual reports of the Registrar-General, shows year by year the mortality from scarlet fever in England and Wales, during registration times.

It will also be seen that diphtheria deaths were included by the Registrar-General with those from scarlet fever until the year 1855. But subsequently to that date the returns form a continuous series comparable with one another *quæ* scarlet fever; and it will be at once observed that there has of late years been a most satisfactory decline in the scarlet fever death-rate. It must not, however, be too confidently assumed that this decline is permanent, for, as Dr. Thorne⁴ remarks: 'There is, perhaps, no disease concerning which it can be said with less certainty that diminutions year by year in its fatality foretell a permanent lessening in its prevalence than is the

¹ The Geographical Distribution of Diphtheria in England and Wales. Sup. Report of the Medical Officer to the Local Gov. Board for 1887. Republished in 'Studies and Statistics.'

² 'The Etiology of Diphtheria,' by E. G. Barnes, M.D., *British Medical Journal*, July 28, 1888.

³ Arthur Ransome, M.D., 'On the Form of the Epidemic Wave, &c.,' *Trans. Epidem. Soc.*, 1881-82.

⁴ R. Thorne Thorne, M.B., F.R.S., 'The Progress of Preventive Medicine during the Victorian Era.' Being the Inaugural Address delivered before the Epidemiological Society of London, Session 1887-88.

TABLE II.—*Showing year by year the total deaths registered in England and Wales during registration times as due to scarlet fever; with the corresponding death-rates per million living and the average annual death-rate for each quinquennial period.*

England and Wales				England and Wales			
Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period	Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period
1888	5,802	880	797	1865	17,700	837	982
1889	10,325	666		1866	11,685	546	960
1840	19,816	1,256		1867	12,300	567	
1841	14,161	889		1868	21,912	996	
1842	12,807	794		1869	27,641	1,244	
1843	Including Diphtheria	Including Diphtheria	797	1870	32,548	1,446	759
1844				1871	18,567	815	
1845				1872	11,922	515	
1846				1873	13,144	562	
1847				1874	24,922	1,050	
1848	14,697	857	884	1875	20,469	851	680
1848	20,501	1,178		1876	16,893	691	
1849	13,128	747		1877	14,456	585	
1850	13,370	753		1878	18,842	753	
1851	13,634	758		1879	17,613	694	486
1852	18,887	1,035	907	1880	17,404	675	
1853	15,699	853		1881	14,275	548	
1854	18,528	995		1882	13,732	521	
1855	16,929	894		1883	12,649	475	
1856	13,557	705	806	1884	10,863	402	241
1857	12,646	652		1885	6,355	233	
1858	23,711	1,212		1886	5,998	218	
1859	19,310	976		1887	7,859	282	
1860	9,681	485		1888	6,378	226	
1861	9,077	451	982	1889	6,698	235	241
1862	14,834	728		1890	6,974	242	
1863	30,475	1,478		1891	4,959	171	
1864	29,700	1,418					

case in regard of scarlatina. Not only do different outbreaks vary very greatly as regards mortality, but the epidemic prevalences tend to occur in cycles; and an abatement extending over a few years has been known to be followed by a wide and fatal diffusion of the infection. And not only so, but the more recent diminution in the amount of fatal scarlatina may be in noteworthy part matter of diagnosis. . . . But, after making every allowance, there remains the important fact, that ever since the decennial period 1861-70 there has been a very general and fairly steady diminution in the fatality of scarlatina in this country, until, in 1885, the rate of death from that cause was less than a quarter of that which formerly prevailed; and it is impossible not to regard so long-continued and marked an abatement as an indication that some of the means conducing to the spread of this very fatal fever have been materially restricted.'

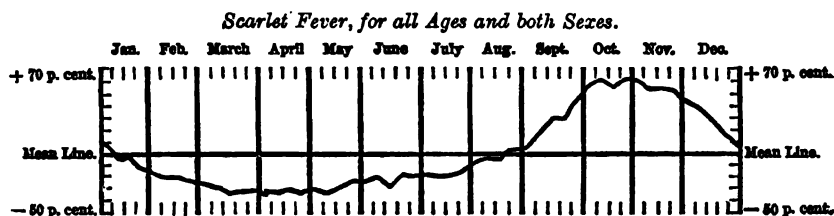
Fatality or Case Mortality.—The fatality of scarlet fever varies largely in different epidemics and even at times during the different stages of the same epidemic. It may range between 8 and 30 per cent. of attacks. Probably 10 per cent. is near the average rate for hospital practice; but since hospital cases are doubtless above the average as regards severity, especially if allowance is made for the mild cases among the general public which escape identification, it is likely that 10 per cent. is too high as a general estimate.

Of the causes of the decided variations in the malignancy of different epidemics little is known. Judging from the evidence, it would seem clear

that although, other things being equal, unwholesome conditions of life almost certainly have some influence in this respect, and the same is probably true of season of the year, yet these are far from being the controlling factors.¹ The type of an epidemic is, in fact, evidently something much more fundamental—something, we may hazard the suspicion, intimately concerned with the evolutionary phases of the species of micro-organism upon which the disease depends.

Influence of Climate.—It appears highly probable that scarlet fever is influenced by climate. Not only is it clear, as will be shown later, that in this country its prevalence is to a considerable extent regulated by season, which is suggestive of the influence of varying climatic conditions, but the fact that the disease has not established itself in the tropical or sub-tropical portions of Asia and Africa would certainly seem to imply that there is something in the climate of those countries which is unfavourable to its development. It would not by any means appear to exclude this inference to refer, as Hirsch does, to the fact that scarlet fever 'has often been found epidemic in the tropical countries of South America,' for climate includes many factors other than mere temperature. The subject requires much closer study before a definite conclusion can be reached; but it is probable that temperate and humid climates are most favourable to scarlet fever.

And Season.—As regards season, Hirsch found that of 485 epidemics of scarlet fever in Europe and North America, 29·5 per cent. attained their maximum in the autumn, 24·7 in winter, 24 in summer, and 21·8 per cent.



in spring. In London, according to Dr. Buchan and Sir Arthur Mitchell, whose curve is here given, the mortality from scarlet fever is at its lowest in April, and attains its maximum in October, again falling rapidly in December.

It must not, however, be assumed that the seasonal prevalence of scarlet fever is the same for all countries. According to Dr. Whitelegge, for instance, the mortality curve for New York is practically the reverse of that for London, the maximum being in April, and the minimum in September.

But for England it may be confidently stated that both the prevalence and mortality of scarlet fever are greatest in the autumn and least in the spring.

Dr. Ballard found that of 8,850 cases of scarlet fever recorded in the books of the Poor-law medical officers and the various medical institutions in Islington for the twelve years 1857–68, 17·7 per cent. occurred in the first quarter, 17·8 in the second, 29·9 in the third, and 35·1 in the fourth.

Dr. Longstaff² has constructed the following diagram, which shows the close similarity between the curve based upon the weekly average of London scarlet fever deaths and that of the admissions to the Metropolitan Asylums Board hospitals of scarlet fever patients, for the ten years 1875–85.

It must be remembered, however, that the admissions to hospital must not offhand be assumed to be a correct measure of the general scar-

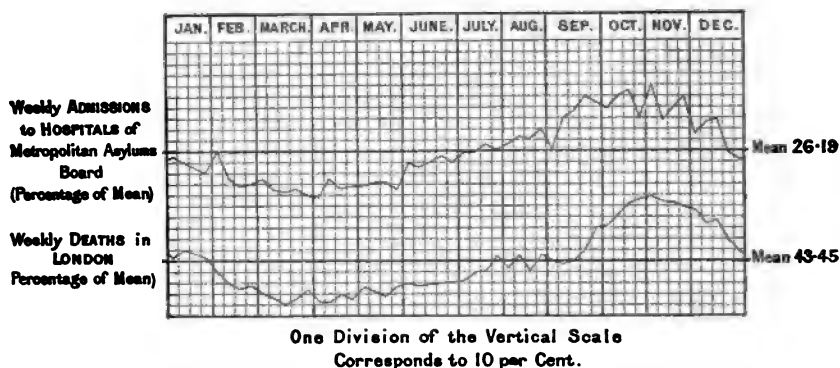
¹ See Hirsch, *op. cit.*, i. 187.

² *Studies in Statistics*, p. 405. Reproduced by Dr. Longstaff's permission.

latinal prevalence, for it is doubtless the more severe cases that seek admission to hospital. But this possible source of fallacy is in great part eliminated by a table constructed by Dr. Whitelegge, which is based on the notification returns of twelve large English and Scotch towns, and which also shows the prevalence of scarlet fever to be greatest in the autumn.¹

As to the influence of particular meteorological conditions upon scarlet fever prevalence and mortality, the evidence put forward by different observers is conflicting. But Dr. Longstaff² has pointed out that there appears to be an inverse relationship between the mortality from scarlet fever, and certain other diseases, and the rainfall. 'The same result is obtained,' he says, 'whether the amount of the fall or the number of days on which it falls

SCARLET FEVER,
LONDON.
Weekly Average 10 Years 1875-1884.



is considered, the connection being somewhat closer in the latter case.' This accords with Dr. Ballard's general conclusion, that a temperature above the average for the season, and a dry state of the atmosphere with little rain, favour the prevalence of scarlet fever more than the reverse conditions.

From what has been said above, it is clear that in this country both the prevalence and mortality of scarlet fever are greatest in the autumn and least in the spring. But what shall we say as to fatality, i.e. the proportion of deaths to attacks? Is scarlet fever more fatal as well as more prevalent in autumn than in other seasons? Distributing into the seasons in which they happened the 1,998 scarlet fever deaths which occurred in the entire parish of Islington during the twelve years 1857-68, and distributing also in similar fashion the 8,850 cases of scarlet fever which occurred in the public practice of the district during the same period, Dr. Ballard, by calculating for each quarter the proportion borne by the deaths in the entire parish to the cases in the whole public practice, arrived at the conclusion that in Islington parish, during the twelve years in question, scarlet fever was, on the whole, less fatal and serious during the third or summer quarter, and most fatal in the first or winter quarter, and that the fatality of the spring and autumn was equal.

Dr. Whitelegge, again, points out that the seasonal curve of notified attacks differs but little in outline from the mortality curve, but that at almost every point the deviation from the mean line is less in the death curve than in the attack curve; in other words, the mortality rises and falls proportionately less than the cases do, indicating that at the season of the year in which the disease is most prevalent it is least fatal, and *vice versa*.

¹ *Epidem. Soc. Trans.*, N.S., vol. vii.

² *Op. cit.*, p. 322.

On the other hand, some observers have been led by their personal experiences to regard the autumn as the season in which scarlet fever is most severe as well as most prevalent. Thus Dr. Gresswell¹ quotes Fothergill, to the effect that he met with more cases of malignant scarlet fever 'from September to December inclusive than in all the other months together.' Willan wrote of scarlet fever: 'This disease, as is usual in the summer months, exhibits a mild train of symptoms, and soon terminates favourably, without producing any material affection of the throat. For some years past it has always been most virulent and dangerous in the months of October and November, but generally ceased on the first appearance of the frost.' Other observers have found 'that in summer the affection of the throat was frequently so trifling as not to demand any particular attention. Haygarth found the disease most virulent in October and November.'

As regards the 588 patients (admitted to the Asylums Board hospitals between early September 1887 and early February 1888), of whom Dr. Gresswell had sole charge, it appears that the severity of the disease 'was greatest among persons attacked in October, and the first three and a half weeks or so of November; and that it began to fall off rapidly afterwards.' This increased severity made itself apparent not only in the death returns, but in the relative frequency of the graver manifestations of the disease. 'I shall show,' says Dr. Gresswell, 'that after a certain date the symptoms, which had up to that date evidenced a general progressive increase of relative frequency and intensity, remarkably ameliorated; and that this amelioration commenced definitely in the latter part of November.' Dr. Gresswell then illustrates by statistical table, and graphically by a chart, the relation to season of the complications and more serious phases of the disease as observed in his 588 patients. In reference to the table and chart he points out that 'though in some points the clinical statistics of the September, October, and November cases were much alike, the relative incidence of many complicating conditions increased from September into October or November, and fell away suddenly in December. . . . The graver manifestations, in fact, became relatively more frequent, and I may add more marked, from September into October or November; and then they suddenly fell off; not merely in regard to two or three determinations, but in regard to most of them—changes which were also expressed in the monthly mortality.'

It has to be borne in mind, of course, that Dr. Gresswell's scrutiny of this question only extended to a limited number of cases at a particular period of an epidemic; and whether further inquiry, of similar exactitude, based upon a larger series of attacks, will support the view that the fatality of scarlet fever, like its prevalence and mortality, is greatest in the autumn months remains to be seen.²

But that the *prevalence and mortality* of scarlet fever in this country are at their lowest in the spring, and thenceforward rise steadily through the summer, attaining their maximum in the autumn, we have seen to be facts beyond doubt.

The Influence of Age and Sex.—The relations of age and sex to attack and death by scarlet fever are dealt with at length in the 49th Annual Report

¹ *Natural History of Scarlet Fever.* By D. Astley Gresswell, M.A., M.D.

² Since the above was written, Dr. Whitelegge has explained the results of his further researches into the subject of epidemic disease periodicities. These results tend to confirm his previous suspicion that the seasonal increase in the mortality of certain diseases, including scarlet fever, is due to increased prevalence of such diseases, and not to an increase in their fatality, i.e. case mortality.

(1886, page xiv) of the Registrar-General. It is impossible here to give the details of the Registrar-General's investigation, but the important conclusions at which he arrives are thus stated :—

'1. The mortality from this disease is at its maximum in the third year of life, and after this diminishes with age, at first slowly, afterwards rapidly. 2. This diminution is due to three contributory causes: (α) the increased proportion in the population at each successive age period of persons protected by a previous attack; (β) the diminution of liability to infection in successive age periods of those who are as yet unprotected; (γ) the diminishing risk in successive age periods of an attack, should it occur, proving fatal. 3. The liability of the unprotected to infection is small in the first year of life, increases to a maximum in the fifth year or soon after, and then becomes rapidly smaller and smaller with advance of years. 4. The chance that an attack will terminate fatally is highest in infancy, and diminishes rapidly with years to the end of the twenty-fifth year, after which an attack is again somewhat more dangerous. 5. The female sex throughout life, the first year possibly excepted, is more liable to scarlet fever than is the male sex. 6. But the attacks in males, though fewer, are more likely to terminate fatally.

'Now it is sometimes said that the separation from its family of a child who is attacked by scarlet fever is scarcely worth the trouble and expense it involves, seeing that the rest of the children, though they may escape on that special occasion, are almost certain to contract this very common disease at some future time, and may therefore as well, if not preferably, have it at once. The results, however, to which our statistical inquiry has led us, are completely subversive of such a position. They show—independently of the plain fact that a very large proportion of persons go through life without ever contracting the disease—that the longer an attack is deferred, the less likely it is to occur at all; and not only so, but that, even supposing it to occur eventually, the less likely it is to end fatally.'

The influences of age and sex in relation to scarlet fever have also been dealt with by Dr. Whitelegge¹ upon the basis of 6,288 cases of scarlet fever notified in the three large towns of Nottingham, Salford, and Leicester. The results, thus independently arrived at, substantially coincide with those of the Registrar-General, except that in Dr. Whitelegge's cases the case mortality was rather higher in the second year of life than in the first. The practical conclusion from Dr. Whitelegge's investigation is that 'in shielding a child against infection during the first few years of life there is a double gain; every year of escape from scarlet fever renders him less and less susceptible, until finally he becomes almost insusceptible; and, secondly, even if he should ultimately take the disease, every year that the attack is deferred reduces the danger to life which it brings.'

Cause and Dissemination.—That the essential cause of scarlet fever is a micro-organism there can, on general principles, be little doubt; but with respect to this branch of the subject, see p. 152, *ante*. As regards dissemination, it has long been known that scarlet fever may be spread by direct infection from case to case, and also by indirect infection through the medium of infected clothing, furniture, books, letters, &c. There are cases on record, too, which seem to place beyond doubt the possibility of the poison being handed on from infected linen to other linen in laundries, and in this doubly indirect way spreading the disease. Moreover, it has frequently been observed that the scarlet fever poison is capable, under certain circumstances, of retaining its infective power for considerable periods of time, altogether

¹ *Epidem. Soc. Trans.*, N.S., vol. vii., 1887-88.

outside the human body. A fresh outbreak of the disease, for instance, has been observed to follow upon the disturbing of some previously infected garment which had been put away in a drawer for months.

Until recent years the above methods of dissemination, i.e. immediate infection from a previous human case, or mediate infection from antecedent human cases, through the agency of fomites, were generally regarded as exhausting the possible methods of scarlet fever transmission. And farther it was apparently not contemplated that the mediate method referred to involved any multiplication of the organism outside the human body, but simply its storage and conveyance. In the year 1870, however, Dr. M. W. Taylor opened the way to a very great advance in our knowledge of the natural history of scarlet fever. While studying an outbreak of that disease which occurred in the town of Penrith, he observed that the main incidence of the disease was upon the customers of a particular dairy. Following up the suggestion thus gained, he found that, prior to the general outbreak in the town, a child at the dairy in question had suffered from scarlet fever, and that not only was the milk taken into the infected dwelling before distribution to the customers, but that the child's mother, while acting as sick-nurse, also at times milked the cows. These facts, coupled with the circumstance that many of the sufferers in the town had no communication with the dairy except through the medium of the milk, seemed irresistibly to point to milk as the vehicle of the infection. Subsequently other observers recorded like experiences elsewhere, and as a result Mr. Ernest Hart was able, in 1881, to refer to fifteen epidemics of scarlet fever in which there was evidence of dependence upon infected milk service. So far, however, the milk was generally looked upon as having become accidentally infected by human agency. Later investigations by Mr. Power and Dr. Klein seem to have shown beyond doubt that human scarlet fever may be produced by milk which owes its infective property to an ailment of the cow. But with respect to this, and the subject of milk-scarlet fever generally, the reader must be referred to Dr. Klein's article (p. 146, *ante*).

The infection of scarlet fever seems to be given off by the breath, the secretions from the nose, mouth, pharynx, ears, and perhaps kidneys, and also by desquamating cuticle. It may apparently cause disease either by being inhaled or swallowed. There is no evidence of its being conveyed by water, and inasmuch as the disease does not appear to spread in the neighbourhood of fever hospitals, it would seem that the infection is not capable of being conveyed any great distance by air currents.

According to Prof. Thomas, several observers have successfully inoculated persons with scarlet fever virus, the inoculation being in some cases followed by general, and in others by local scarlet fever. Children so inoculated were said not to contract the disease when subsequently exposed to infection. He also states that a disease corresponding to scarlet fever in man has been met with by different observers in the horse, 'cats, dogs, swine, and other domestic animals.' Attacks of what appears to be scarlet fever occasionally follow surgical operations, and it is usually said that such injuries increase susceptibility to that disease. At the same time it must be remembered that some of such cases are probably not scarlet fever at all, but septic conditions. Cases of the kind have been observed under circumstances which seemed to put scarlet fever infection out of the question. It appears, too, according to McLeod,¹ that so-called surgical scarlet fever is met with in India, where scarlet fever is practically unknown.

Period of Incubation and Infectiveness.—The period of incubation of

¹ *Epidem. Soc. Trans.*, N. S., vol. iv., 1884-85.

scarlet fever is almost always, if not invariably, less than a week, and it may be as short as a few hours. Usually, however, it is somewhere between two and four days.

The infective period extends from the earliest symptoms to the end of convalescence, but it is greatest when the fever is at its height. No patient can ever with safety be allowed to mix with the healthy until the expiration of at least six weeks, no matter how slight the attack; and it will often be necessary to extend the period of isolation to eight or nine weeks, or even longer, for it must be continued until all traces of desquamation have ceased. Moreover, it must be remembered that discharges from the ear and nose are capable, for a time at least, of spreading the disease, and the same is possibly true of albuminuria. It also has to be borne in mind that recrudescence of the disease—especially recurrent sore throat and albuminuria—sometimes occurs, and it is doubtful how late such true recrudescences may take place, and renew the infective condition of the patient.

Protection.—One attack usually confers immunity throughout life, though well-marked second, and even third, attacks occasionally occur. According to Dr. Squire,¹ persons too who have previously suffered from the disease, 'when much exposed to it, may have sore throat or other signs of partial sickening sufficient to start the disease elsewhere.' Such modified second attacks quite likely renew the protection of the persons thus affected.

Relation to other Diseases.—It has been stated by Löschner, Köstlin, and others that scarlet fever epidemics especially tend to follow upon outbreaks of measles. This, however, is denied by other authorities. If any relationship of the kind, beyond a purely casual one, occurs, the probable explanation would seem to be that suggested by Köstlin, viz. that measles increases the susceptibility to scarlet fever, and, indeed, this would appear not to be unlikely.

Scarlet fever is sometimes found closely associated with diphtheria.

The belief in a close relationship between scarlet fever and a form of puerperal fever, in the sense of the infection of the former being capable of originating the latter, has been widely held. It is certain that in some cases in which recently confined women have been freely exposed to the poison of scarlet fever, no harm has resulted. Nevertheless, the danger of such an occurrence has been maintained by high authorities, and is probably a real one, even if it may have been somewhat exaggerated. It derives some indirect support, too, from the circumstance that scarlet fever seems undoubtedly to exercise an injurious influence upon vaccination, apparently giving rise to irregularity, and in some cases playing a part in the causation of septic mischief with erysipelatous manifestation.

TYPHUS FEVER

Synon.: *Petechial Fever, Gaol Fever, Ship Fever, Spotted Fever.* Fr. *Typhus*; Ger. *Exanthematischer Typhus, Flecktyphus*; It. *Tifo*.

History and Distribution.—Typhus has only in our own day been clearly differentiated from other fevers, notably enteric and relapsing fevers, with which it had been previously confused. There can be no doubt, however, as to its having existed for a number of centuries, and Murchison believed it to have been referred to by Thucydides. Hirsch, after pointing out that much of

¹ 'Scarlet Fever,' Quain's *Dict. of Medicine*.

the 'war or famine sickness' of history was doubtless 'a mixture of various kinds of disease, such as diarrhoea, dysentery, scurvy, typhus, and frequently also malarial fever and typhoid,' refers to an outbreak of fever at a monastery near Salerno in the year 1088 as one of the earliest records of typhus having any degree of definiteness. The more reliable history of typhus, however, seems to date from the sixteenth century, during which period Hirsch finds 'numerous accounts of the disease from almost every part of Europe.' Throughout the seventeenth, eighteenth, and earlier part of the present century it appears to have been widely prevalent in Europe, visiting at one or another time practically every country, including Iceland. Great Britain and Ireland suffered extensively; and indeed Ireland occupies a conspicuous place in the history of this disease. 'In no part of Europe,' says Hirsch, 'does typhus bear the character of an endemic malady so decidedly as in Ireland.' Severe epidemics, many of which appear to have spread from that country to England and Scotland, occurred in Ireland in the years 1708-10, 1718-21, 1728-31, 1770-72, 1797-1802, and during the present century 1816-19, 1821-22, 1826-28, 1836-37, 1846-47, 1862-64. Russia, Italy, and Spain have also suffered heavily. Hirsch considers that, speaking generally, the 'period of typhus' came to an end about the year 1815, and he states that since that time 'the disease on European soil has only once, in 1846-47, attained the same general diffusion which the history of pestilence presents to us so often in former centuries.' During more recent years the prevalence of typhus fever in Europe has markedly abated, and the disease has tended more and more to limit itself to particular areas.

Beyond Europe, typhus has prevailed more or less extensively in Persia, North China, Egypt, and North America, in which latter country it would seem to have been largely a result of immigration. On the other hand, Japan, Australia, New Zealand, and the greater part of Africa, including Cape Colony, are said to have been exempt from the disease. In India the disease seems to be at times observed.

The most important fact brought out by a study of the history of typhus fever is its practically invariable association with overcrowding, starvation, and general misery, whether as a result of war, famine, or more continuous social defects in the communities affected. This is seen to be the case both by a wide survey of the history of the disease and by a study of its behaviour in a given country. But this matter will be referred to later.

Mortality.—The more recent history of typhus fever in England may be sufficiently inferred from Table III. (p. 276), in which it will be seen that with the exception of a temporary check in 1882-88 the registered mortality from this disease has steadily declined.¹ The figures in the table are taken from the Annual Report of the Registrar-General for 1890 (pp. xxxviii-xl). The typhus deaths were not abstracted prior to 1869.

Case Mortality.—As will be seen immediately, age is such an important controlling factor as regards the fatality of typhus fever, that the two matters cannot be usefully discussed apart. Murchison, however, found that of 18,268 cases of typhus at all ages admitted into the London Fever Hospital, 3,457, or 18·9 per cent., ended fatally. But he points out that these, being hospital cases, were doubtless above the average as regards severity, and he

¹ It is doubtful, however, whether the actual typhus mortality during recent years is not somewhat understated in these death returns. An inquiry in the winter of 1886-87 by Mr. Spear showed typhus to be existing in no less than seventeen English towns, and in many instances the deaths had been returned as due to other causes. See later. But notwithstanding this there is no doubt that a marked abatement has occurred in recent years.

gives 10 per cent. as a general estimate of typhus fatality. But this will vary in different epidemics.

TABLE III.—*Showing the total Deaths from Typhus Fever for each Year from 1869 to 1891 and the corresponding Death-rate per Million living.*

England			England		
Year	Total deaths	Death-rate per million living	Year	Total deaths	Death-rate per million living
1869	4,281	193	1881	552	21
1870	8,297	147	1882	940	36
1871	2,754	121	1883	877	33
1872	1,864	80	1884	328	12
1873	1,638	70	1885	318	12
1874	1,762	74	1886	245	9
1875	1,499	62	1887	211	8
1876	1,165	48	1888	160	6
1877	1,104	45	1889	137	5
1878	906	36	1890	151	5
1879	533	21	1891	137	5
1880	530	21			

Influence of Race, Age, and Sex.—There is no sufficient evidence that race exercises any influence over liability to typhus. It is true that the Irish have suffered excessively from this disease, but the circumstance is probably due to the relatively unwholesome conditions under which the poor of Ireland habitually seem to have lived, whether at home or abroad.

No age is exempt from typhus fever, but persons between the ages of fifteen and twenty years are most liable to attack. As regards fatality, however, the matter is very different, and the risks to life, in the event of attack, increase largely with age.

Murchison gives the case mortality of the higher ages as 85·89 in persons between 30 and 40; 48·48 in persons between 40 and 50; 53·87 in those between 50 and 60; and 67·04 in those over 60. As regards the earlier periods of life he says: 'The rate of mortality [fatality] was somewhat greater during the first than during the second ten years of life. Thus, the mortality during the first five years of life was 6·69 per cent.; in the second lustrum it fell to 3·59; between ten and fifteen it was only 2·28 per cent.; and between fifteen and twenty 4·46 per cent. After twenty it went on progressively increasing.'¹

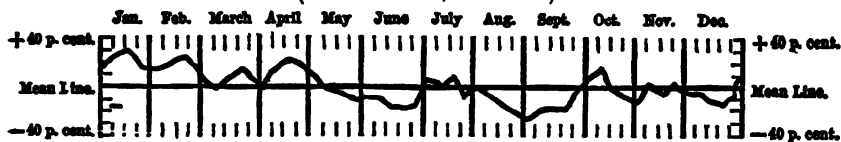
Sex is said to have little influence upon liability to attack, and the total deaths are also pretty equally divided between the two sexes. It would appear, however, that the fatality is usually somewhat greater for males at all ages taken together, than for females. Murchison's cases gave a male fatality of 19·6 per cent. of attacks, and a female fatality of 18·2 per cent. From five to fifteen years the fatality among females was twice as great as that among males, but after fifteen years the male fatality was the greater at every period of life.

Influence of Climate and Season.—From what has already been said with regard to the history and distribution of this disease, it will have been seen that it is mainly a disease of temperate and colder climates; for, although it is true that it has prevailed in some tropical countries, it seems even there to have occurred in the colder seasons of the year, and in the more elevated localities. In England both the prevalence and mortality of

¹ *The Continued Fevers of Great Britain*, p. 236. By Charles Murchison, M.D., F.R.S. 3rd ed.

the disease have probably, upon the whole, been greater in the late autumn, winter, and spring, than in the summer and early autumn.

Typhus—for all ages and both sexes (Buchan and Mitchell)
(London deaths, 1869–1874)



As regards prevalence, this is attested to by the records of admissions to fever hospitals. As regards mortality, the same result is indicated in the above curve.

Dr. Longstaff¹ found that for the ten years 1875–84 the mortality was above the mean from January to April inclusive, again slightly in June, and lastly through October, November, and December. And the maximum was in November instead of January as shown above. On the whole, therefore, it would seem that typhus bears a less constant relation to season than is the case with several of the other epidemic diseases.

Cause and Dissemination.—It is inferred on general grounds that typhus fever is caused by a micro-organism, but so far this has not been established.

With regard to accessory causes, we have already seen that history discloses an association between destitution and overcrowding, and the rise of outbreaks of typhus, so close as to render it impossible to escape the view of a causal connection subsisting between the two phenomena. Indeed, so striking is the association that it has frequently been maintained, and among others by no less an authority than Murchison, that the disease may arise *de novo* from such conditions alone.

In the introductory section of this paper an attempt has been made to show that such a doctrine, if intended to signify the origination of the assumed microphytic cause of disease independently of parent microphytes, is untenable. But several other modes, by which a disease, as such, might practically have *de novo* origin, have also there been indicated, and it is possible that one or other of such ways may have to do with the beginning of typhus.

Another matter of interest with respect to the origin of typhus outbreaks is the possibility of their being initiated by a 'revivification of long dormant contagion,' and Mr. Spear states that he found reason for attributing recent disease to this cause.

But whatever may be the truth as to the life history of the typhus virus, there is no question of the influence of overcrowding and poverty as most important etiological factors in this disease. All authorities are agreed upon that point, and it may be confidently asserted that, apart from such circumstances, typhus would never arise. When once started, however, the disease is highly infectious, and although in the main it still tends to cling to the overcrowded homes of the destitute, it frequently passes beyond those limits, for any who are brought within the range of infection may become attacked, and act as foci for the dissemination of the disease elsewhere. Fortunately there is a circumstance with respect to this infection which largely tends to limit the ravages of the disease, and that is that it does not appear capable of travelling far through the air without losing its potency. Although highly active in the immediate vicinity of the patient, it soon appears to be rendered harmless by diffusion through the atmosphere. With abundant ventilation

¹ *Studies in Statistics*, p. 402.

of the sick-room, assuming, of course, proper isolation of the sick, there need, therefore, be little danger of the disease spreading, except among the medical attendants and nurses, whose duties necessitate frequent and close contact with the sufferers. That both these classes of persons inevitably incur considerable risk is, however, but too plainly testified to by the records of fever hospitals.

The poison of typhus may be conveyed in clothing, but there is no evidence of its being disseminated by water, milk, food, or domestic animals. It is believed to be given off by the breath and skin of the patient, and typhus patients emit a peculiar odour, perceptible at a distance of a foot or two, which has been thought to be associated with the infection.

Periods of Incubation and Infectiveness.—The period of incubation of typhus fever varies considerably in different cases, and this is probably due to the quality of the poison as regards concentration; probably also to the dose of the poison received and the condition of the recipient. Most authorities, however, including Murchison, give the usual period as about twelve days. Some have stated it as shorter: thus Dr. Beveridge gives it as 'about a week,' and Liebert as five to seven days. Murchison considered that the infection is comparatively slight during the first week of the illness, but that the disease is most contagious from the end of the first week up to convalescence. It is therefore impossible to lay down a definite rule as to the actual length of time that infection will last in different cases, but probably it is never safe to allow a patient to mix with others in less time than a month from the date of attack, and a longer period of isolation will often be necessary.

Protection.—One attack of typhus fever usually protects against the disease for the future; but although second attacks are unfrequent they do occasionally occur, and in exceptional instances the disease appears to confer practically no immunity at all, the individual contracting it whenever exposed to infection. Such cases, however, are very rare.

RELAPSING FEVER

Synon.: *Famine Fever*; *Seven-day Fever*; *Bilious Remittent Fever*; *Bilious Typhoid Fever*; *Febris vel Typhus recurrens*. Fr. *Fièvre à rechute*, *Typhus à rechute*; Ger. *Das recurrirende Fieber*, *Hungerpest*; It. *Tifo Recidivo*.

History and Geographical Distribution.—It is generally considered that the first reliable reference to relapsing fever is that of Ruttty, who, in his 'Chronological History of the Diseases of Dublin' (1770), described an epidemic, evidently of this disease, as having occurred there in the year 1789. There can, however, be little doubt that relapsing fever had occurred in earlier times. Murchison, indeed, considered that an epidemic in the island of Thasus, recorded by Hippocrates, 'resembled it very closely in most of its characters, including an intermission of five or seven days between the febrile attacks, jaundice, epistaxis, tendency to miscarry, &c.'

Subsequently to 1789, further epidemics occurred in Dublin, in 1745, 1748, and 1764-65. The first recognisable description of relapsing fever in the records of Scotland seems to date from 1741. In the year 1799, and again in the years 1817 and 1826, the disease occurred in both Ireland and Scotland. It then apparently disappeared until 1841, when it broke out in Scotland, appearing the following year in Ireland (Hirsch). In 1848 it was also widely prevalent in Scotland. So complete seems to have been its cessation in that country between 1826 and this period, the Murchison says

it was now 'regarded by many as a new disease.' It was at this time that the doctrine of relapsing fever as a distinct and separate disease was definitely promulgated by Dr. Henderson, of Edinburgh, and other Scotch physicians; though, according to Murchison, Dr. O'Brien, of Dublin, had drawn a distinction between relapsing fever and typhus as early as 1828. Prior to this, relapsing fever had most commonly been regarded as a variety of typhus, with which disease it was usually found to be associated in epidemics. By some observers, however, it has been looked upon as a malarious form of yellow fever.

Further outbreaks occurred in Ireland and Scotland in 1847-48, and the disease now appeared in different English towns, including London, Croydon, Liverpool, and Manchester. Between 1868-78 it was again prevalent in Scotland and England, the earliest cases in London being 'chiefly in a quarter inhabited by Irish and by poor Jewish emigrants from Poland' (Hirsch).

Outside Britain the disease seems first to have been observed in Russia, where it apparently occurred in single epidemics in 1838 and 1840, and on an extensive and widespread scale in 1868-64. In 1878-79 it broke out again among the Russian troops in Bulgaria. It was observed in Germany in 1847, and was widely diffused in that country in 1868, appearing again in 1871-72 and 1878-79. There are a few references to the disease in the Scandinavian kingdoms and Belgium, but apparently none as regards France, Switzerland, Italy, Spain, and Portugal. Similarly, the Australian colonies seem to have been so far exempt. The disease has, however, occurred in North America, India, China, and Egypt.

Mortality.—In Table IV. will be found the deaths recorded in England and Wales as having resulted from relapsing fever in each of the years from 1869 to 1890, the deaths being given for males and females separately. From this table it will be seen that the mortality from relapsing fever has during the period in question always been insignificant, even in epidemic

TABLE IV.—*Showing the Deaths, at all Ages, of Males and Females registered in England and Wales in the Years 1869-1891 as due to Relapsing Fever.*

Year	Males	Females	Total	Year	Males	Females	Total
1869	72	67	139	1881	6	9	15
1870	301	327	628	1882	4	9	13
1871	155	172	327	1883	7	9	16
1872	25	38	63	1884	6	6	12
1873	21	20	41	1885	5	5	10
1874	13	10	23	1886	8	4	12
1875	25	27	52	1887	3	6	9
1876	17	13	30	1888	4	4	8
1877	22	18	40	1889	1	2	3
1878	11	14	25	1890	2	1	3
1879	10	12	22	1891	4	7	11
1880	9	12	21				

years, as compared with that of typhus, enteric, and some of the other fevers. It will also be observed that it has diminished during successive years in a manner similar to that of typhus.

Fatality.—The fatality or case mortality of relapsing fever is low. Dr. Grimshaw gives it as from 1.2 to 2 per cent. for London, and as up to 4 and 4.5 per cent. for other places, the average rate being about 4 per cent. Murchison's statistics give the rate for Great Britain as 4.08 per cent.

Influence of Sex.—More males appear to be attacked than females. This Murchison considered due to the fact of there being a larger proportion of

males among the class most prone to the disease, viz. the vagrant class. But although more males appear to be attacked, more females die of the disease, so that the case mortality among the latter, at all ages taken together, is higher than that among the former. According to Murchison's London Fever Hospital figures, the case mortality was 1·64 per cent. for males and 2·15 for females; 'but this result is attributable to a larger proportion of the males being under thirty years of age. Under fifty the mortality was greater among females, but above fifty it was much greater among males.'

And Age.—As with typhus and enteric fever, so with relapsing fever, the largest number of attacks occur in the age period fifteen to twenty years. But the subsequent fall, especially during the years twenty-five to forty-four, is less sudden than in the case of enteric fever, and, in this respect, more resembles that of typhus.

The following table, which is compiled from three tables given by Murchison, shows the percentage of attack at each period of life by each of these three diseases. The percentages are based upon 18,188 cases of typhus, 2,111 of relapsing, and 5,911 of enteric fever.

TABLE V.

Age	Percentage at each period of life, of attacks at all ages			Age	Percentage at each period of life, of attacks at all ages		
	Typhus	Relapsing	Enteric		Typhus	Relapsing	Enteric
Under 5 years	1·29	1·84	·98	From 50 to 54	4·35	3·78	·60
From 5 to 9	6·59	5·96	9·44	„ 55 to 59	2·42	1·84	·83
„ 10 to 14	12·06	11·08	18·16	„ 60 to 64	2·20	2·55	·83
„ 15 to 19	16·16	19·18	26·86	„ 65 to 69	1·08	·56	·08
„ 20 to 24	18·28	16·81	19·69	„ 70 to 74	·46	·28	·00
„ 25 to 29	9·52	9·80	10·15	„ 75 to 79	·17	·04	·03
„ 30 to 34	8·36	8·43	5·36	Above 80	·01	·00	·00
„ 35 to 39	8·08	6·82	8·40	Age not specified	·00	·00	1·80
„ 40 to 44	8·80	6·72	2·09				
„ 45 to 49	5·72	4·26	1·08	—	99·90	99·85	99·88

The fatality of relapsing fever is very low during the early years of life, but, like that of typhus, markedly increases with advancing years. Murchison gives the following London Fever Hospital figures:—

Under 80 years, 1,366 cases,	7 deaths, or	·51 per cent.
Above 80 „	745 „	32 „
„ 50 „	191 „	18 „
„ 60 „	72 „	9 „
		12·50 „

Influence of Season.—Relapsing fever seems to exhibit a considerable independence of season. On the whole, perhaps its attacks are somewhat more common in the winter than during the warmer periods of the year. The mortality also appears to be greatest in the winter.

Cause and Modes of Dissemination.—The phenomena of relapsing fever are believed to be bound up with the presence in the blood of a particular spirillum (see page 171).

The predisposing causes of relapsing fever appear to be identical with those of typhus, viz. overcrowding, filth, and starvation. Murchison maintained that starvation was the main factor, relapsing fever differing somewhat from typhus in this respect; but the experiences of other observers throw great doubt upon this, for in some notable epidemics starvation, according to the records, certainly cannot be said to have been a conspicuous etiological factor. Rather it seems that overcrowding, and all which that entails, is the important element, though, no doubt, starvation, fatigue, and the like are powerful

contributory causes when present. As regards the influence of overcrowding there seems no doubt.

When once established, relapsing fever is highly infectious. 'The mode of communication,' Murchison remarks, 'is probably the same as in typhus—that is to say, the poison is conveyed through the air, or by fomites, from the sick to the healthy, and actual contact is not necessary.'

With regard to the distance to which the poison will travel through the atmosphere, Murchison further says: 'It is only they who are in close communication with the sick, or who visit, or reside in, their badly ventilated dwellings that suffer. With free ventilation the disease almost ceases to be communicable.' Medical men and nurses are, as in typhus, particularly liable to contract the disease, and laundresses also frequently contract it from washing the clothes of patients.

There are no grounds for thinking that soil, or topographical or geological circumstances, have any influence over the rise and spread of the disease.

Period of Incubation.—More exact data are wanted with respect to the period of incubation. Murchison considered that, on the whole, it was shorter than that of typhus. Like that of typhus, however, it seems to be subject to considerable variation. The Silesian physicians gave it as from fourteen to twenty-one days, and Lebert as from three to seven days. Among the twelve cases in which Murchison considered that he definitely fixed it, there were variations from 'attack immediate on exposure' up to fourteen days.

Protection.—Relapsing fever appears to afford comparatively little protection against subsequent attack. Certainly, second attacks are of much more common occurrence than in the case of typhus.

Relation to other Diseases.—Relapsing fever has generally been observed to occur in epidemic association with typhus, and the most usual course of events in these mixed epidemics has been, as Murchison shows, that 'the proportion of relapsing cases has been greater at the commencement than towards the close of the epidemic, and with the advance of the epidemic typhus has taken the place of relapsing fever.'

The commonly accepted explanation of the frequent association of these two diseases is that their *predisposing* causes are similar, the diseases themselves being held to be specifically distinct, and therefore dependent upon different *essential* causes.

The belief in the separate nature of the two diseases appears to be based mainly upon the following considerations: (a) that they present marked clinical differences; (b) that the one disease does not give rise to the other; (c) that one does not protect against the other; (d) that Obermeier's spirillum is not observed in the blood of typhus patients, but is always present in that of relapsing fever patients during the relapse.

These arguments, if all well founded, must reasonably be allowed considerable weight. Nevertheless, as elsewhere pointed out, it is by no means impossible that diseases possessing relatively specific characters may yet have some genetic relation to one another, and thus it must not be too hastily concluded on the ground of clinical, or even apparent bacteriological, differences that such diseases are entirely unrelated.

Looked at from this standpoint, it is difficult to avoid being struck with the similarity, as regards epidemiological features, of these two diseases. They admittedly occur under much the same conditions, and relapsing fever is usually associated with typhus. They both seem to be entirely independent of soil, and largely independent of season. In their modes of dissemination they are strikingly similar—so similar, indeed, that Murchison, a firm sup-

porter of the 'separate' doctrine, says of the behaviour of the relapsing fever infection that 'the remarks made on this subject under the head of Typhus apply with equal force to relapsing fever.' Considering the peculiarities of the typhus infection, this is a suggestive circumstance. The diseases also resemble each other in the marked increase of their fatality with advancing years of life; and there is, too, a general resemblance between their age incidence in the matter of attack. Their latent period seems to be similarly variable. The geographical distribution of *recognised* relapsing fever, it is true, is less wide than that of typhus; but, as Murchison remarks, it is 'much wider than it was once supposed to be.' Moreover, relapsing fever was first recognised, and has been most conspicuously present, in countries especially prone to typhus, notably Ireland and Russia. Lastly, it seems doubtful whether the arguments mentioned above as forming the basis of the doctrine of the separateness of these two diseases are all founded upon fact.

The assumption that the one disease does not give rise to the other appears to be based on the statement that, during mixed epidemics, cases of both kinds are not found coming from the same households. But it is pointed out by Fagge that, according to Murchison's later records, this is not strictly true. Then, again, the great frequency with which mixed epidemics have been observed to consist mainly at the outset of relapsing fever and later of typhus may possibly be indication of 'progressive development.' Even, however, if both these positions should be untenable, it may still be that the two diseases are evolution varieties of a common stock, but breeding true, and consequently each producing only its own kind.

As regards the statement that the diseases do not protect against each other there seems also some doubt. It is true that well-authenticated cases of persons having suffered from both these diseases have been recorded by different observers. But such cases do not prove the proposition in question. Second attacks of both typhus and relapsing fever are known to occur, and protection is a matter of degree. On the other hand, the late Dr. J. C. Steele observed 'that in the epidemic of 1848 persons who had previously suffered from typhus were not attacked by relapsing fever; and the same thing is said to have been noticed recently in epidemics at St. Petersburg and at Breslau, . . . while Lebert draws attention to the fact that in his cases of typhus, among those who had already passed through relapsing fever, the mortality was only half as great as in the population generally. Do these facts,' it is very naturally asked, 'indicate that after all there is between the two diseases some connection the nature of which is not at present understood?'¹

INFLUENZA

Synon.: *Epidemic Catarrh*. Fr. *La Grippe*; Ger. *Influenza*; It. *Influenza*.

History and Distribution.—When influenza reappeared in England at the close of the year 1889 there was a marked tendency among the public, and even the medical profession, towards the view that we had amongst us some entirely new scourge—so unfamiliar had we become in this country with the character of the malady. Yet, curiously, there is perhaps no disease which has so frequently swept over the face of the globe in pandemic waves.

Zuelzer, in his article on influenza in Ziemssen's 'Cyclopædia,' expresses

¹ Fagge, *op. cit.*, i. 154.

the opinion that that disease can only with certainty be traced back to the beginning of the sixteenth century, though he points out that epidemics of catarrhal fever which may have been influenza are recorded as early as the ninth century. Hirsch,¹ on the other hand, considers that 'the disease may be followed into the remotest periods from which we have any epidemiological record at all;' and in a paper read before the Society of Medical Officers of Health in February 1890, Dr. W. H. Clemow² referred to various interesting early epidemics dating back from the year 1481 to the time of Hippocrates, some of which would certainly seem to have been influenza. Ignoring earlier records, which were thought too deficient in detail to be of much use for epidemiological purposes, though 'they certainly relate to influenza,' Hirsch gives a long list of epidemics extending over the period from 1173 to 1875, and affecting at one or another time every quarter of the globe.

But the first extensive pandemic of which we have reliable information seems to have occurred in the year 1510, and Zuelzer remarks that with that year 'begins a series of epidemics, the wide distribution of which has been reached by no other acute infectious disease.'

The chief epidemics in this country during the present century occurred in the years 1808, 1833, 1887-88, and 1847-48. But in addition to these major epidemics, Dr. Parsons, in his report to the Local Government Board on the influenza epidemic of 1889-90, points out that in the earlier years which followed the major epidemics of 1887 and 1848 minor periodical recurrences at intervals of three or four years may be traced in the death returns of the Registrar-General down to the year 1858.³ Whether the lesser prevalences should be regarded as the periodical flickerings of the dying virus left in this country by the larger epidemics, or whether they should be considered in connection with the prevalence of the disease in other countries during the same years, is doubtful, for it seems, according to Hirsch, that influenza was epidemic in Germany in 1841; in Germany, France, Russia, and North America in 1848-44; generally over the western and eastern hemispheres in 1850-51; generally over Europe, including the Farøe Islands and Iceland, in 1855-56; and generally again over the western and eastern hemispheres in 1857-58. Since that time influenza is recorded by Hirsch as having occurred in Australia, North America, Bermudas, Netherlands, Cape of Good Hope, and Iceland between 1860 and 1862; New Caledonia, France, California, and Switzerland in 1863-64; France, England, Mauritius, Germany, and Belgium, 1866-67; Turkey in 1868; universally over North America in 1873, and widely over Europe and North America in 1874-75. But notwithstanding this influenza prevalence in other countries, it would appear tolerably clear from the Registrar-General's returns that England enjoyed a comparative immunity from the disease for a number of years prior to 1889. The present epidemic, of which, during the past three winters, we have had such a painful experience, would, however, seem to be one of the most severe and widespread ever recorded. Its original progress over the globe was also more rapid than that of previous epidemics.

Geographical Distribution and Influence of Race, Climate, and Season.—A glance at the history of this disease, whether it be at the general history or the history of this latest epidemic, shows influenza to have a world-wide distribution, and consequently to affect persons of all nationalities. Climate, season, and weather also appear to have no controlling influence over its

¹ *Op. cit.*, i. 7.

² 'Epidemic Influenza,' by Dr. W. H. Clemow, *Public Health*, April 1890.

³ See Table VI., p. 285.

spread, and Dr. Parsons points out, with respect to the 1890 outbreak, that, like former epidemics, it 'prevailed nearly at the same time at places in the north and south hemispheres, i.e. in opposite seasons of the year. It has prevailed in the cold of Russia and the heat of India; in the moist climate of the British Isles and the dry air of Egypt. In Spain its advent was preceded by a month of cold, dry, frosty weather, and at New York by the mildest and moistest season on record.' Speaking of former experience, Hirsch says that, in its origin, influenza is independent 'of the seasons and of the influences of the *weather*; and it is in that respect that it is marked off most essentially and most decidedly from epidemic bronchial catarrh.' Zuelzer expresses the same view. Telluric influences appear also without notable effect upon influenza.

As to the original home of the disease nothing is known. But in the northern hemisphere influenza epidemics have exhibited a tendency to travel from east to west. This is stated by Zuelzer, Watson, and others; and Hirsch, although he appears to consider that such a 'progress of the disease in a definite line from east to west' has been exaggerated, admits that it cannot be denied that 'some pandemics, regarded as a whole, do afford evidence of the sickness travelling in the alleged direction, from east to west.' The present epidemic certainly followed this course.

Periodicity.—Of any regular and sustained periodicity history seems to afford no evidence, though the annual recurrences of the disease during the last three or four years might perhaps point to an annual developmental cycle of the poison. The apparent tendency of the disease on previous occasions to recur at intervals of three or four years has already been referred to.

Variation in Type.—Influenza epidemics differ in type from time to time, and similar differences are commonly observed among different cases of the same epidemic. Thus the prominence of ordinary catarrhal symptoms may vary considerably. In the present epidemic such symptoms have been less conspicuous than on former occasions, though it has been especially pointed out by observers of previous epidemics that catarrh, in the ordinary sense, may be entirely absent. Again, the gastro-intestinal, pulmonary, and nervous symptoms may each be more or less prominent at different times, though all, and especially the last, seem to be present in every epidemic.

Different epidemics have, moreover, preserved the same general characters, such as rapidity of dissemination, general independence of climatic, seasonal, age, and sex influences, relative suddenness of onset as regards attack, and low case mortality.

It has been stated above that when the present epidemic appeared in 1889 many hesitated to regard it as influenza. This, however, was largely due to the erroneous conception of influenza which prevailed as a result of the improper application of the term 'influenza' during non-epidemic periods to ordinary catarrh. But, as Dr. Parsons remarks, 'few persons who have read the history of the previous epidemics of influenza, and compared it with that of the epidemic of 1889-90, can doubt that the disease with which we have been visited is the same as that which has prevailed so extensively in former periods.' Certainly influenza presents certain points of similarity to dengue, and some persons were at first not unnaturally inclined to believe the present epidemic to be one of that disease. Dengue, however, is essentially a disease of hot climates and seasons, is seldom fatal, is unattended with pulmonary complications, almost always presents a rash, and is frequently followed by profuse desquamation. Other clinical distinctions also seem to exist between the two.

Mortality.—In Table VI., page 285, will be found year by year the total

TABLE VI.—*Showing year by year the Deaths registered in England as due to Influenza, the corresponding Influenza Death-rate per Million of the Population, and also the Death-rates from diseases of the Respiratory and Circulatory Organs.*

England and Wales				
Year	Total deaths from influenza	Death-rates per million from		
		Influenza	Diseases of the respiratory organs	Diseases of the circulatory system
1838	806	55	—	—
1839	887	59	—	—
1840	1,030	67	—	—
1841	1,659	106	—	—
1842	883	53	—	—
1847	4,881	285	2,980	580
1848	7,963	460	2,587	556
1849	1,611	92	2,586	618
1850	1,880	78	2,184	637
1851	2,152	120	2,705	657
1852	1,859	76	2,646	699
1853	1,789	99	3,118	760
1854	1,061	58	2,856	734
1855	3,568	193	3,439	786
1856	1,029	55	2,812	726
1857	1,393	73	3,057	775
1858	1,794	98	3,399	852
1859	1,112	57	3,069	879
1860	1,130	58	3,484	956
1861	746	38	3,233	909
1862	915	45	3,358	930
1863	919	45	3,308	959
1864	804	39	3,363	1,089
1865	596	29	3,291	1,078
1866	651	31	3,592	1,059
1867	607	29	3,312	1,076
1868	806	14	2,847	1,049
1869	703	32	3,594	1,144
1870	615	28	3,626	1,161
1871	848	15	3,569	1,177
1872	278	12	3,147	1,193
1873	266	11	3,632	1,244
1874	245	10	3,797	1,289
1875	449	19	4,232	1,331
1876	203	8	3,656	1,333
1877	205	8	3,547	1,413
1878	195	8	3,830	1,445
1879	266	11	4,332	1,513
1880	171	7	3,614	1,372
1881	99	4	3,428	1,369
1882	90	3	3,565	1,376
1883	107	4	3,675	1,473
1884	72	3	3,342	1,506
1885	138	5	3,737	1,613
1886	83	3	3,641	1,647
1887	85	3	3,626	1,666
1888	92	3	3,502	1,695
1889	55	2	3,309	1,664
1890	4,523	157	4,120	1,757
1891	16,686	572	4,474	1,826

influenza deaths recorded by the Registrar-General in England and Wales during registration times, and the corresponding death-rate per million of the population. The death-rates per million living are also given for diseases of the respiratory organs and diseases of the circulatory system. It will thus be seen that a rise in the deaths from influenza has generally been attended by a rise in the mortality from lung disease and sometimes heart disease. During the years 1890 and 1891 this is especially con-

spicuous, but it may be traced elsewhere in the table.¹ The mortality attributable to influenza must therefore not be measured solely by the deaths registered as due to that cause, but the indirect effect of the malady as expressed in the increased mortality from certain other causes must be taken into account. Accordingly the Registrar-General estimates that, during 1890, 'the total number of deaths due directly or indirectly to the epidemic influenza was not merely 4,528, but 27,074, or 941 per million living.'

Case Mortality.—The actual mortality from influenza, in such an epidemic as we are now passing through, is therefore a sufficiently serious matter. The fatality, however, i.e. the proportion of deaths to attacks, is low, and this seems to have been a constant characteristic of influenza. Dr. Parsons found that among the in-patients treated in eight large London hospitals, from which during the year 1890 he obtained returns, the fatality was 84.5 per 1,000 cases. Among the total patients at the same hospitals, 'in' and 'out,' amounting in number to 5,516, it was only 1.6 per 1,000 cases. In the Army, where the patients' ages and circumstances are favourable for recovery, there were 9 deaths among 8,108 cases, or 1.1 per 1,000. In the more recent epidemics the case mortality seems to have been greater.

Influence of Age and Sex.—The mortality from epidemic influenza is greatest during the middle and later periods of life, and in this respect there is a marked contrast between that disease and what in non-epidemic periods is loosely called influenza, for in the latter condition the mortality is mainly among children and old people. The difference in the mortality of these two maladies at different ages is well shown in the following table from Dr. Parsons' report:—

TABLE VII.—*Proportion at the several Ages to 100 Deaths from Influenza at all Ages, in London.*

Period	Percentage at several ages							Total
	Under 1	1-5	5-20	20-40	40-60	60-80	80 and above	
1876-89	22.6	16.0	3.4	3.4	10.0	26.9	7.5	100
First quarter, 1890 . .	5.3	4.3	4.7	24.7	36.2	22.4	2.5	100

In the epidemic of 1847-48 the age mortality differed somewhat from that during the first quarter of 1890, in that the greatest proportion of deaths occurred in the age period 60-80, instead of 40-60, as shown in the table. But it also differed from that of the influenza of non-epidemic times 'in not showing the high proportion of deaths in early childhood.' As regards liability to attack, Zuelzer, referring, of course, to previous epidemics, says:

Everybody is agreed in stating that epidemics attack the population without distinction of age, sex, constitution, or condition. . . . That children cannot be regarded as in any special degree exempt is proved by the large percentage attacked in different schools and training ships.

Cases of supposed influenza in new-born infants have also been reported.

With respect to the incidence of the disease upon the different sexes, it appears from the Registrar-General's reports that in the epidemic years 1847-48 there were 6,819 deaths of females registered as influenza, and 6,025 of males. Dr. Peacock attributed the excess of female deaths to the

¹ It is somewhat masked by the steady increase, from other causes, in the mortality from heart and lung diseases. It will best be appreciated by comparing the mortality from these diseases in influenza years with that of the years immediately succeeding them.

fact of the disease being mostly fatal to persons of advanced age, and it has already been seen that the greatest proportion of the deaths in that epidemic was at the age period 60–80. In the year 1890 we see from the Registrar-General's annual report that of the 4,528 influenza deaths registered, 2,108 were of females and 2,415 of males. The mortality for that period was therefore greatest among males; but the greatest proportion of the 1890 influenza deaths occurred in the age group 40–60; and further, the proportion of deaths in the 20–40 period was, for London, greater than that among persons in the 60–80 group. This, therefore, would seem to support Dr. Peacock's view. Moreover, in 1890 the female mortality was greatest at all ages over sixty-five, but at all other age groups, except one or two of the earlier ones, where the numbers were small, the mortality was higher among males—probably as a result of greater exposure to infection and cold.

On the whole, then, it would seem that the mortality from influenza is little influenced by sex, except perhaps in connection with differences as regards occupation. Neither do the sexes seem to differ in liability to attack if similarly circumstanced. The number of male and female clerks attacked in the Post Office in 1890 were in proportion to the numbers of each sex.

Protection.—Influenza is frequently said to confer no protection against future attack. Certainly, a large number of persons suffer a second and third time under circumstances which point to a reinfection. At the same time, it must be borne in mind that the fact of many persons undergoing second or third attacks does not prove that other persons have not been protected, or even that the persons suffering from more than one attack have not been protected for a period during the interval, except in cases in which the second attack follows closely upon the first; and cases of the latter sort are open to the suspicion of being the result of relapse or recrudescence.

Cause and Dissemination.—Various hypotheses have been framed as to the cause of influenza, but the most probable one, in view of recently acquired knowledge, both with respect to the origin of other diseases and the behaviour of influenza, is that it depends upon a micro-organism. Moreover, there would appear good grounds for thinking that a particular bacillus has been shown to stand in causal relation to that disease by Pfeiffer and Kitasato.

But, however that may be, it seems now established that influenza is an eminently infectious complaint—a fact which, although often maintained by individual authorities in the past, has been frequently denied by others. It is impossible here to enter into the evidence on this point in detail. The following is, however, the summary of Dr. Parsons' conclusions on this matter:—

'In view of the circumstances—1. That the progress of the epidemic was contrary to the prevailing winds, and that it was independent of season or any particular kind of weather; 2. That it has not been shown to have travelled faster than human beings could travel; 3. That it has not occurred among persons placed under circumstances precluding its communication by human agency; 4. That, as a general rule, in each country it has appeared first in the capital, or the ports of entry, or the frontier towns in communication with countries previously invaded, and that the towns, as a rule, have been affected earlier than country places; 5. That neighbouring communities have in certain instances been affected only at considerably different dates; 6. That many instances are recorded of the disease having been introduced into a district, and spread to persons in contact with the patient, and sometimes afterwards to others; 7. That persons brought much into contact with others—e.g. people going daily to business in towns, have generally

been the first to suffer; their households, and people locally employed, being affected later; 8. That in public services and establishments persons employed together in large numbers in enclosed spaces have suffered in larger proportion than those employed few together, or in the open air; 9. That in institutions in which the inmates are brought much into association the epidemic has more quickly attained its height, has prevailed more extensively, and been sooner over than in those in which the inmates are more secluded from one another; I am of opinion that the epidemic has been propagated mainly, perhaps entirely, by human intercourse; though not in every case necessarily from a person obviously suffering from the disease. I see no sufficient ground for believing in that world-wide spread by atmospheric agencies which has been so generally assumed. I do not say that the contagion once imported into a locality may not propagate itself outside the human body in such media as damp ground or air contaminated with organic exhalations, but the fact of adjoining communities suffering at different dates seems opposed to the notion of the poison travelling far through the air. I do not find sufficient evidence that the recent epidemic has anywhere commenced suddenly with a large number of simultaneous cases, unprecedented by any previous ones, and I think that the rapidity with which influenza develops into an epidemic may be accounted for by its short period of incubation, by the comparatively general susceptibility to the disease, and by the existence of numerous slight and unrecognised cases. I do not, however, wish to exclude the possibility that the specific germ of the disease may multiply in appropriate media, e.g. in damp, organically polluted confined air, outside the human body.¹

The question, too, of the possible communicability of influenza from animals to man, or *vice versa*, has to be considered, and it is suggestive to find among the records of influenza epidemics from the earliest times reference to concurrent illness among domestic animals, notably horses, dogs, and cats.

With respect to this subject Hirsch says: 'Even in the oldest epidemiological records, there are indications of these coincidences, both as regards time and place, as well as of the identity or at least similarity of the form of disease; and the number of these observations is so remarkably large that the suggestion of an etiological and perhaps also pathological connection between the epidemics on the one hand, and those epizootics on the other, may be regarded as provisionally proved, although it ought not at the same time to be left out of sight that the notion of "horse influenza" has remained to the present day a somewhat vague one with veterinary surgeons, and that very various diseased processes appear to have been included therein.' Sir Thomas Watson says: 'It has been observed also that shortly before, or during, or soon after, the prevalence of these epidemic catarrhs epizootic diseases have raged; various species of brutes, and of birds, have been extensively affected with sickness.'

Certainly influenza or 'pink eye' was prevalent among horses in different parts of England either before, during, or after the epidemic of 1890; but as regards this epidemic Dr. Parsons is of opinion that the origin of influenza in the human subject from a similar disease in the horse is rendered improbable by the following considerations:—

1. That 'influenza' among horses has frequently prevailed at times and places when there has been no human epidemic; 2. That in the late epidemic persons having to do with horses were not observed to be specially

¹ See also the evidence brought forward by Dr. Sisley on 'Epidemic Influenza.'

or earliest affected ; and that cases of apparent transmission of the disease from the horse to man were of somewhat rare occurrence ; 8. That in many places where the late influenza epidemic prevailed (e.g. at Newmarket), the absence of any similar disease among horses was affirmed.

Dr. Parsons, however, reports that in many places where influenza was prevalent pet dogs, cats, and caged birds, especially those living indoors, were noticed to concurrently suffer from similar symptoms.

The likelihood of the microbe having ability to live for a considerable period outside the body seems supported by the fact that after an epidemic the disease tends for a time to recur at intervals in the same locality, as we have unfortunately experienced of late. In reference to this, Sir Thomas Watson says : 'The locality does not appear to be thoroughly cleared of the poison for some time ; or perhaps a more cautious statement of the fact would be, that the disorder generally shows itself again in succeeding years, but in a milder and less general form.'

Periods of Incubation and Infectiveness.—It is not easy to fix the period of incubation of a disease which spreads so rapidly as influenza, owing to the difficulty of eliminating the fallacy of multiple exposure. The evidence collected however, makes it certain that the period is commonly a short one, and the most usual time would seem to be two to three days, though it may be longer or shorter. Influenza is infectious quite in the early stage, and certainly as long as the eighth day, and perhaps longer.

WHOOPIING COUGH

Synon. : *Pertussis*. Fr. *Coqueluche* ; Ger. *Keuchhusten* ;
It. *Tosse Convulsa*.

History and Distribution.—There seems no sufficient evidence that whooping cough is a disease of great antiquity. Hirsch gives an epidemic in Paris in the year 1678 as the first reliable record of the disease. In England whooping cough was referred to in the seventeenth century by Sydenham and Willis.

At the present day the disease has practically a world-wide distribution, but is more constantly prevalent and more severe in temperate climates than in tropical regions. So far, however, there is no evidence that race influences liability to attack, for there are accounts from India, Egypt, and other countries, of coloured children suffering equally with Europeans.

But, although the disease appears, at one or another time, to have visited most parts of the world, its introduction to some countries, as Australia and New Zealand, appears to have occurred during the present century, as a result of importation. Similarly, in other localities having but little communication with the rest of the world, as Iceland and the Farøe Islands, it has only occurred at apparently rare intervals when introduced from without.

Mortality.—In the following table will be found the mortality recorded from this disease in England and Wales from 1898 to 1891.

A glance at this table will show that the mortality from whooping cough in this country is very considerable indeed ; of late years this disease has destroyed more children than any of the other so-called zymotic diseases, with the exception of diarrhœa.

Case Mortality.—The case mortality of whooping cough is not high, and is sometimes stated at about 5 per cent. But in addition to the number of

children who die from this disease, a very considerable number are more or less permanently injured by it.

Influence of Season.—The effect of season upon whooping-cough mortality

TABLE VIII.—Showing year by year the Deaths registered in England and Wales from Whooping Cough, and the corresponding Death-rates per Million of the Population.

England and Wales							
Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period	Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period
1838	9,107	596	504	1867	11,873	548	545
1839	8,165	526		1868	9,228	419	
1840	6,132	389		1869	10,966	498	
1841	8,099	508		1870	11,901	529	
1842	8,091	502		1871	10,360	455	
1847 ¹	9,260	540	480	1872	13,806	596	499
1848	6,862	394		1873	9,612	411	
1849	9,622	548		1874	10,362	437	
1850	7,770	437		1875	14,280	594	
1851	7,905	440		1876	10,556	432	
1852	8,022	440	511	1877	11,358	460	527
1853	11,200	609		1878	17,784	710	
1854	9,770	525		1879	12,752	508	
1855	10,185	541		1880	13,662	530	
1856	9,225	483		1881	10,830	415	
1857	10,138	526	498	1882	15,259	579	459
1858	11,648	598		1883	10,471	393	
1859	8,976	456		1884	11,476	425	
1860	8,555	429		1885	13,106	481	
1861	12,309	612		1886	12,936	470	
1862	12,272	602	516	1887	11,251	404	444
1863	11,275	547		1888	12,287	436	
1864	8,570	409		1889	12,225	430	
1865	8,647	409		1890	13,756	478	
1866	15,764	786		1891	13,612	468	

in London will be seen from the curve on the next page. This curve shows the London mortality from whooping cough is at its minimum in September, rising steadily through November and December to its maximum in April, thenceforward steadily declining through the warmer months. It is usually said that the prevalence of whooping cough in this country, like the mortality, is greatest about the months of March and April. It will be noted that this curve is almost exactly the reverse of that for scarlet fever.

The relation to season thus indicated does not seem to hold for all other countries. According to Hirsch, in Sweden, for instance, both the prevalence and mortality of the disease appear to be greater in the summer and autumn than in the winter. As regards the effect of weather, Dr. Goodhart² remarks: 'Atmospheric changes have a most important bearing upon pertussis. It has been repeatedly noticed in the whooping-cough ward at the Evelina Hospital that the children are worse, even when otherwise doing well, when the wind turns cold or suddenly changes; and it is notorious that the disease runs a much less determined and persistent course in summer than in the colder seasons of the year.'

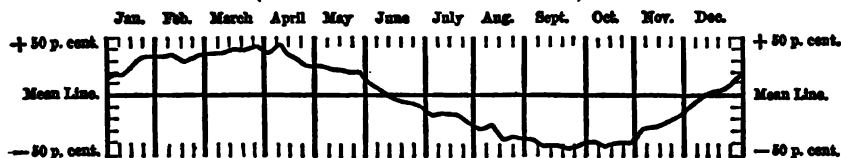
Influence of Age and Sex.—The mortality from whooping cough is decidedly greater during the first year of life than during any subsequent year.

¹ Deaths not abstracted by Registrar-General for years 1843–46.

² *Op. cit.*, p. 282.

Reference to the Registrar-General's annual reports will show that between 40 and 50 per cent. of the whooping-cough deaths at all ages occur within

Whooping Cough—for all Ages and both Sexes (Buchan and Mitchell)
(London Deaths from 1845 to 1874).



this period. During the second year the mortality is considerable, but it falls rapidly during subsequent years, and after the tenth year is insignificant.

Of the total deaths, over 90 per cent. at all ages occur during the first five years of life. The incidence of the disease as regards attack seems also to be mainly upon the earlier years of life, though no age is entirely exempt. Of 814 cases collected by Dr. Goodhart, 801 occurred in the first six years of life, sixty-two of these being in the first year. The mortality among females is greater at all age periods than among males. One attack of whooping cough usually confers immunity against a repetition of the disease.

Periods of Incubation and Infectiveness.—Period of incubation is stated by some observers to be from four to six or seven days, but it appears liable to variation. The late Dr. Murchison reported some cases which appear to be free from any likelihood of fallacy, in which the incubation period was apparently a fortnight.¹ Other more recent authorities have also given fourteen days as a common period. Whooping cough is highly infectious from the very commencement of the attack, before the whoop manifests itself, and remains infectious for some six or eight weeks, as a rule. Most authorities seem agreed that the infective period may be considered to have ceased at the end of six weeks from the commencement of whooping, provided all spasmodic cough has ceased.

Cause and Dissemination.—The epidemic and infectious character of this disease, together with the facts of its having an incubation period, and affording protection against a second attack, are circumstances which seem to justify the conclusion from analogy that it is of microbic origin, but at present no micro-organism has been shown to stand in causal relation to it. The disease is, so far as at present known, invariably spread by infection from case to case. Such infection, however, need not necessarily be direct, as the virus may be carried in clothing, &c. Owing to the early infectiveness of whooping cough, it is, like measles, largely spread by the attendance at schools, and other public gatherings, of children who are sickening for it, but who have not, so far, manifested the characteristic symptoms. There is no evidence that this disease is ever disseminated by the agency of water, milk, or domestic animals. Neither does it appear to have been shown to have any relation to any particular topographical or geological circumstances.

Relations to other Diseases.—Whooping cough is usually said to occur frequently in association with measles. It seems probable, however, that this association has been exaggerated, and as to the order of sequence in which these two diseases occur with respect to each other, there seems also difference of opinion. Dr. Goodhart remarks that 'when an appeal is made to statistics, the association appears to be less common than I had anticipated. Of 805 cases of pertussis of which I have notes, measles is only mentioned as recent in fourteen.' Dr. Goodhart also found that in all these cases the

¹ *Trans. Clin. Soc.*, xi. 1878.

measles preceded the whooping cough. Other observers, on the other hand, have described the whooping cough as preceding the measles. Hirsch found that in 495 epidemics of whooping cough, a coincidence with epidemic measles was noted ninety-four times. 'In fifty-eight of these the diseases occurred together; in eleven whooping cough preceded measles; and in twenty-five it came after.'

MUMPS

Synon.: *Parotitis*; *Cynanche Parotidea* (Cullen); *Parotitis Epidemica*; *The Branks*. Fr. *Les Oreillons*; Ger. *Ziegenpeter*; *Bauerwetz*; It. *Parotitide*.

History and Geographical Distribution.—According to Hirsch, epidemic inflammation of the parotid gland is described 'in a masterly fashion' by Hippocrates, who records, among other things, the liability of the testicle to become inflamed during the course of the malady. The distinctness of epidemic from symptomatic parotitis seems also to have been clearly recognised by the early Greek and Roman writers generally, as well as by the mediæval practitioners. Evidently, then, mumps is a disease of considerable antiquity.

In the present day its geographical distribution is practically coextensive with the habitable globe. In its diffusion mumps is at times limited to particular areas, or particular sections of the community, such as the children at certain schools, or the occupants of particular barracks, workhouses, or prisons. But at other times its epidemicity is more marked and it extends over wide areas of country. Sometimes it displays a tendency to recur in a locality at short (as yearly) intervals, but as a rule a longer period elapses between successive epidemics.

Influence of Climate and Season.—That no particular climate will prevent the occurrence of mumps is evident from the records of epidemics of the disease in Iceland and Lapland on the one hand, and in India, Arabia, and the West Coast of Africa on the other—as well, of course, as throughout the countries in the temperate zone. Nevertheless, as a rule, mumps is decidedly most prevalent during the cold and wet seasons of the year.

Mortality.—The mortality from mumps is insignificant. From the Registrar-General's annual reports for the ten years 1881–90 it appears that among the entire population of England and Wales some eighty deaths only are annually registered as due to this disease. Even this, however, probably overstates the actual mortality from mumps, for there can be little doubt that some of the deaths so registered are due to other maladies, notably diphtheria.

Fatality.—In view of the wide prevalence of the disease in this country, and of the few deaths ascribed thereto, it is clear that the fatality is insignificant.

Influence of Age and Sex.—Mumps is said to be most common in children from ten to fifteen years of age, but the greatest registered mortality is among the very young. Thus, of the 800 odd deaths from mumps recorded by the Registrar-General in England and Wales during the ten years above referred to, about 68 per cent. occurred during the first five years of life;¹ and

¹ It may be suspected that not a few of these deaths were in reality due to other causes, notably diphtheria.

during those years the deaths were most numerous in the first year, diminishing with each succeeding year of the lustrum. The minimum was reached in the age period 15-20 years, after which there was a slight increase. Deaths occurred at all the age periods up to, and including, the period eighty-five years and upwards.

As regards sex, the deaths were decidedly more numerous among males than females, consisting of 472 for the former sex and 365 for the latter. In only two of the years did the female mortality exceed that of the male, and in one they were equal. In the remaining seven years the male deaths preponderated. This is in accord with the view expressed by different observers, that the disease is more common among males than females.

Cause and Mode of Dissemination.—It may be inferred from analogy that mumps is, in all probability, a microbic affection. So far as is known, it spreads only by infection from case to case, and it would therefore seem likely that the micro-organism upon which it is here provisionally assumed to depend belongs to the class of obligate parasites. The infection is believed to be given off by the breath. No relation between mumps and any particular telluric conditions has been made out.

Period of Incubation.—The period of incubation seems most usually to be from a fortnight to three weeks. It is probably seldom much less than twelve days.

Relation to other Diseases.—Mumps has often been considered to have occurred in association with measles and with diphtheria, and sometimes, though less frequently, with scarlet fever. But whether there is any relationship, other than a casual one, between mumps and the diseases in question is doubtful. Hirsch considers the observations too few to justify a conclusion on the point. As regards diphtheria at least, belief in concurrence of mumps therewith has often no doubt been due to error of diagnosis.

Protection.—One attack of mumps usually confers immunity, but second attacks do sometimes occur. Dr. Pye-Smith mentions an instance which came under his observation in which 'a boy had mumps three times during his school life.'

DIPHTHERIA

Synon.: *Angina maligna*; *Cynanche maligna*; *Putrid sore-throat*.

Fr. *Diphthérie*; Ger. *Diphtheritis*; It. *Difterite*.

History.—It is only possible here to indicate a few of the more salient points of the history of diphtheria. Certainly diphtheria, or a disease very nearly allied to it, may be traced back to antiquity; and although Hirsch appears doubtful whether it is referred to in the Talmud and the Hippocratic writings, as has been alleged, he states that 'in the writings of some of the later Greek physicians, particularly Aretæus and Aetius, we meet with descriptions of an affection of the throat as to the identity of which with angina maligna there can be hardly any doubt.' As regards the writings of the Arabians and mediæval physicians of the West, Hirsch considers reference by them to 'angina' as often of doubtful import, but thinks 'there are accounts of epidemic forms of sickness in those ages given by some of the chroniclers, which may perhaps be taken as relating to malignant sore-throat.' In England there seems to have been an epidemic of angina in

1889, which carried off a large number of children ;¹ and in the Rhine districts there was, according to Hirsch, prevalence of an ' unknown ' sickness in 1517, ' so that men's tongues and throats were covered as if with a fungus and turned white, and they were neither able nor inclined to eat or drink from pails in the head not unattended with pestilential fever.'

In following the history of this malady into more recent times, two circumstances attract the attention—viz. the considerable extension which has apparently occurred in the geographical distribution of malignant throat ailment—at least as regards its conspicuous prevalence—and the variations which such ailment has also exhibited from time to time in the matter of prevalence generally.

Considering these two points together, we find that angina maligna was more or less continuously prevalent in Spain from 1588 to 1618, and re-appeared in various parts of the country throughout the remainder of the century. Besides Spain, it also, rather later in point of time, visited Portugal, Southern and Central Italy, and the adjacent islands. At the beginning of the eighteenth century it again broke out in Spain. In 1786 it seems, judging from the accounts of Noah Webster, to have been extensively prevalent in England and America. About 1745–50 the disease again occurred in Portugal and Italy; and now also, for the first time, come reliable records of its epidemic prevalence in France (north-east) and Holland, also of further outbreaks in England (at Liskeard and other places in Cornwall). About 1752 it was prevalent in Switzerland, Germany, and New York, and in 1755 in Sweden. Still later in the century the disease was epidemic in Portugal, the North of France, Holland, Germany (Osnabrück), the Northern States of America and England (London and Chesham).

Then, except in the case of France, seems to have come a marked remission, and Hirsch remarks that ' as the eighteenth century was drawing to a close, Angina maligna retired into the background among epidemic diseases which then held the stage.' This remission apparently lasted for the first half of the present century, when the disease again appeared on an extensive scale, assuming ' the character of a true pandemic, a character which it has maintained to the present day.'

It must not, however, be concluded that, except in the case of France, the disease entirely disappeared during the first half of the present century. Sporadic cases and even small epidemics occurred from time to time in most of the countries which had been previously visited; but that such outbreaks must have been on a much smaller scale than during the preceding century Hirsch infers both from the meagre writings on the subject and from the fact that when, about 1860, the disease again became widely prevalent, it was ' expressly affirmed by many observers in all sorts of places . . . that they had no knowledge of the disease when they first saw it.' As regards Great Britain, cases apparently occurred in Dublin, Glasgow, Edinburgh, Warwickshire, Surrey, and Kent during the earlier years of the century; and from 1845 to 1856 many scattered cases occurred in London, Kent, Lincolnshire, Herefordshire, Staffordshire, Norfolk, Devon, and Cornwall. In 1849 there was a decided epidemic at Haverfordwest, and in 1855 one at Launceston.

France, it has already been said, was exempt from the general remission. Throughout the period in question epidemics of diphtheria were both extensive and frequent in that country, although for the most part confined to the North. In 1821 Bretonneau, who had had opportunities of studying epidemics of malignant angina at Tours (1818–21), first propounded his doctrine of the essential identity of various throat ailments characterised by what he

¹ Webster's *History of Epidemic and Pestilential Diseases*, Hartford, 1799, i. 148.

then regarded as a membranous inflammation. To this condition of the air-passages he gave the name of *diphthérite* (διφθέρα, skin). Later, however, upon concluding that the morbid process was not an inflammatory one, he adopted the name of *diphthérie*. In connection with the persistence of the disease in France, and as bearing upon the question of the transport of the virus by human agency, it is interesting to note that the French troops suffered from diphtheria in the Crimea.

Proceeding to the latter half of this century we find, according to Hirsch, that 'this new era in the history of Angina maligna begins for the larger part of Europe and North America at almost all points with the years 1857 and 1858, a little earlier in some countries than others. . . . ' The diffusion of the disease, too, seems to have been far wider than hitherto. As regards France, it was no longer confined to the North. In England, as will be seen from Table IX., the mortality was excessively high in 1858-59.

Beyond Europe and North America, the disease about this period also became more or less extensively prevalent in parts of India, China, Australia, the West Indies, Argentine Republic, Peru, and parts of South Africa, though it is true there are accounts of earlier outbreaks in several of these localities.

From the above historical sketch it would seem clear, even allowing for the imperfect records of earlier times, that this disease has vastly extended its epidemic area, and that, during what may be called its historic period, it has exhibited one decided remission and a subsequent sudden, and almost world-wide, increase in epidemicity. It is striking also to observe how tenaciously diphtheria has adhered to certain localities, and how often places which had been previously affected by it were among the first to be attacked in later epidemics. As regards the very general prevalence of diphtheria which set in between 1855 and 1860, no doubt increased means of communication may have had some influence; but it would seem unlikely that greater facilities for locomotion constituted the only, or even the main, cause. Moreover, this increase of epidemicity has to be considered along with the previous remission and the still earlier prevalence of the eighteenth century. Such variations would seem incapable of being explained solely by variations in social circumstances, whatever minor part such circumstances may have contributed to the larger result; and we appear driven to the conclusion that the main factors were such as were bound up with conditions proper to the virus itself. The tendency of the disease to reappear in localities previously affected might mean simply that such localities offered a specially favourable environment of some kind to the cultivation of the virus whenever introduced from without; or it might mean that the diphtheria micro-organism, having once been introduced to such localities, has not ceased to exist during the non-epidemic periods, but has simply either more or less died down, or, perhaps, living a saprophytic existence, largely lost for the time being its pathogenic function. Subsequently, upon the re-occurrence of suitable conditions, meteorological or other, it has taken on fresh activity, or acquired a renewal of pathogenic property.

Of the more modern diphtheria prevalence in England we have now a broad indication in mortality statistics. The diphtheria deaths have only been abstracted by the Registrar-General since the year 1855, up to which time the few that occurred were included with those of scarlet fever.

The registered mortality in England from this disease rose gradually during the years 1855, 1856, and 1857, from 20 to 82 per million, and then with a bound in 1858 and 1859, in which years it was as high as 389 and 517 per million respectively. In reference to the excessively high rates in these two years, however, it has to be noted that during those years, and

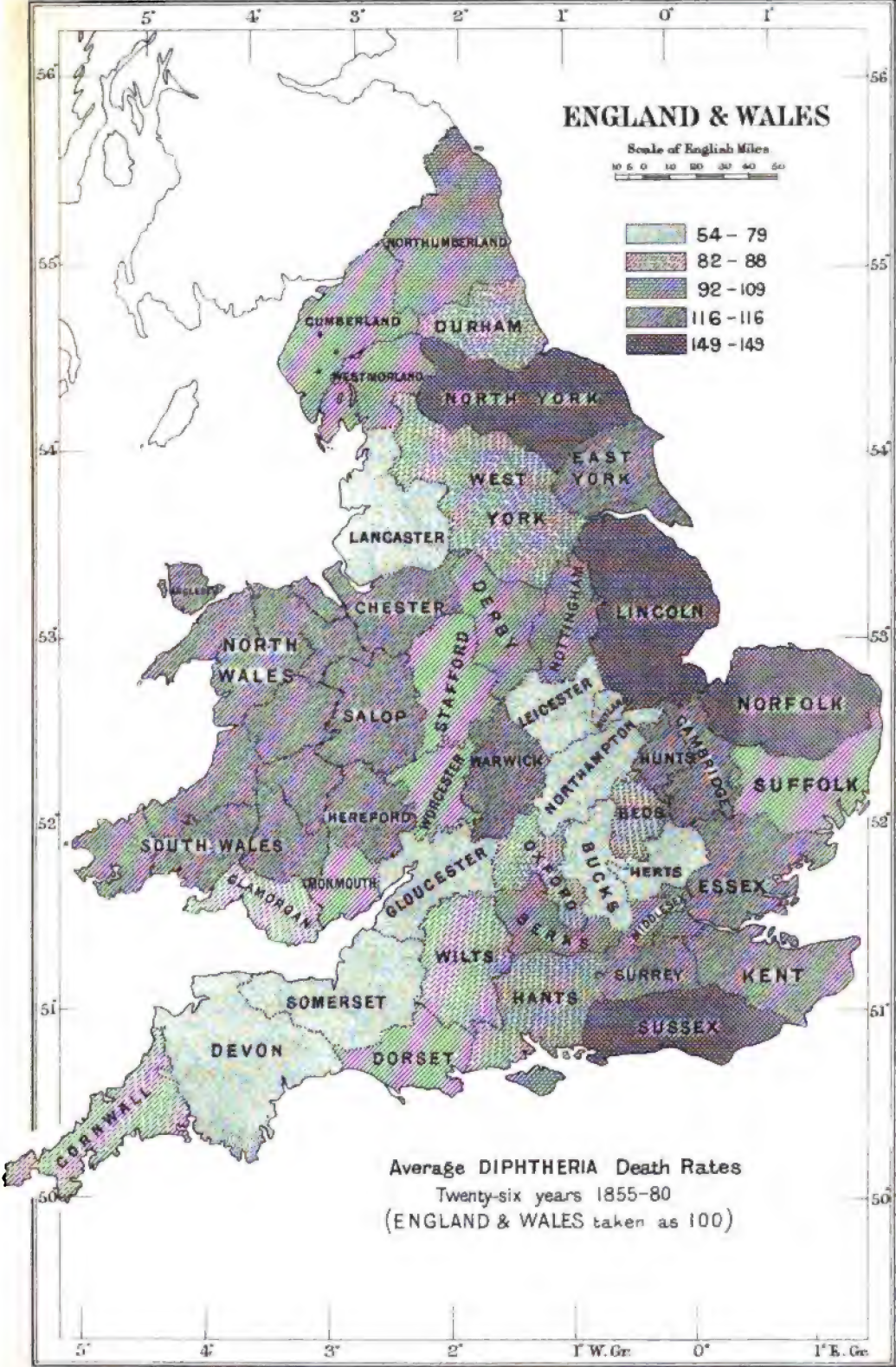
especially the former, scarlet fever was also widely prevalent, and some allowance should doubtless be made for confusion between the two diseases. Nevertheless, it has been seen above that at this time diphtheria was becoming conspicuous over a large part of the civilised world, and it is certain that it showed a marked increase in this country also. Shortly prior to the more general extension of epidemicity in England, diphtheria, as we have seen, had occurred in various parts of the country, and was supposed to have been imported from France. Such may have been the case, but it is worthy of note that Cornwall, in which county the disease had occurred in the previous century, was now one of the earliest invaded. In the year 1860 the mortality fell to 261 per million, i.e. just half what it had been during the preceding year. For the decennial period 1861-70 it further fell to 187, and in the succeeding decennium it still further fell to 121. During the period 1881-90, however, there has been a decided increase in diphtheria mortality, the rate rising to 162.

One important point in connection with the behaviour of diphtheria in this country requires particular attention. Until recently diphtheria, according to all authorities, was especially a disease of sparsely-populated localities, but one of the most striking characteristics of its modern behaviour is its conspicuous invasion of the towns. Considering first the case of the metropolis, it appears as regards the period 1861-70 that, while the diphtheria mortality rate per million living was, for England and Wales, 187, it was for London only 179. But in the next decennium the rates were 121 and 122 respectively, and in the decennium 1881-90 they were 168 for England and Wales, and 259 for London. Thus, during the later period, although there was a decided increase in diphtheria mortality in the country generally, this increase was relatively far greater in the metropolis. But this urban invasion is by no means limited to London, as Dr. Longstaff has shown in his paper on 'The Geographical Distribution of Diphtheria in England and Wales.' Taking the Registrar-General's mortality returns for the twenty-six years, 1855-80, and dividing these years into three periods, and by also dividing the various counties in England and Wales into dense, medium, and sparse areas, according to the relative density per acre of their population, Dr. Longstaff was able to show that although during the whole period the sparse districts suffered most heavily from diphtheria, yet in each successive period the diphtheria mortality of the towns, relatively to that of the rural districts, had become greater.

In this paper Dr. Longstaff, in a map, here reproduced by his permission, shows that 'the distribution of diphtheria is apparently *sui generis*; the mortality from the disease clearly is not regulated by the same causes as influence the general mortality.'¹ It also differs vastly from that of the other zymotic diseases, as, for instance, diarrhoea, measles, and especially scarlet fever. The latter disease is most prevalent in the mining and manufacturing districts, some of which have particularly low diphtheria rates. The contrast between the distribution of these two diseases has also been pointed out by Dr. Edgar Barnes.

Periodicity.—Hirsch considers that the history of diphtheria indicates a cyclical character in the epidemicity of the disease, though 'the several cycles have extended over periods of various length, many of them only a few years,

¹ During the period in question (1855-80), the greatest diphtheria mortality occurred in the North Riding of Yorkshire, Lincolnshire, Norfolk, and Sussex; and the next greatest in East York, Cambridgeshire, Hunts, Essex, Kent, Surrey, Warwick, Wales (except Glamorgan), and some adjoining counties. In striking contrast was the low mortality of Devon, Somerset, Lancashire, West York, and various midland counties. This is illustrated by Dr. Longstaff in a shaded map.





and others lasting several decades.' Looked at broadly, it may be that the prevalence of the eighteenth century, the remission during the first half of the present century, and the subsequent renewal of pandemic prevalence now going on, are the manifestations of an inherent periodicity with a rise and fall extending over a number of years.

TABLE IX.—*Showing the Deaths registered as due to Diphtheria in England and Wales during the years 1855–1890; the Annual Death-rate per Million living for England and Wales during the same Years; and the Annual Diphtheria Death-rates per Million living for London during the Years 1859–91.*

England and Wales			London	England and Wales			London
Year	Total deaths	Death-rate per million living	Death-rate per million living	Year	Total deaths	Death-rate per million living	Death-rate per million living
1855	385	20	—	1874	3,560	150	122
1856	608	32	—	1875	3,415	142	167
1857	1,588	82	—	1876	3,151	129	109
1858	6,606	339	—	1877	2,731	111	88
1859	10,184	517	284	1878	3,498	140	155
1860	5,212	261	174	1879	3,053	120	155
1861	4,517	225	239	1880	2,810	109	144
1862	4,908	241	255	1881	3,153	121	172
1863	6,507	315	275	1882	3,992	152	222
1864	5,464	261	207	1883	4,218	158	244
1865	4,145	196	144	1884	5,020	186	241
1866	3,000	140	152	1885	4,471	164	227
1867	2,600	120	145	1886	4,098	149	212
1868	3,013	137	158	1887	4,443	160	235
1869	2,606	117	107	1888	4,815	171	219
1870	2,699	120	104	1889	5,368	189	290
1871	2,525	111	105	1890	5,150	179	281
1872	2,152	98	80	1891	5,086	178	240
1873	2,531	108	95				

Mortality.—In the above table will be found the total deaths from diphtheria registered in England and Wales annually from the year 1855 to 1891. The annual death-rates per million living are also given; and for the purpose of indicating the relative increase during late years in the mortality from this disease in London, as compared with that of the country generally, the annual death-rates for London have been added.

Case Mortality.—The case mortality of diphtheria varies so greatly that it is impossible to form any useful estimate on the subject. In well-pro-nounced diphtheria, and especially in certain epidemics, it is unquestionably high, but it is impossible to gauge at all the number of slighter cases of diphtheritic throat illness, which, as will be seen later, so frequently accompany or precede diphtheria epidemics.

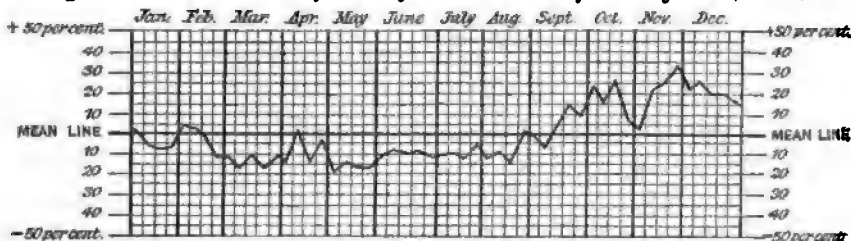
Influence of Climate and Seasons.—Diphtheria would seem to be capable of occurring in any climate, but it is far more common in temperate and cold regions than in the tropics. In this country the mortality from diphtheria is very regularly at its highest during the December quarter, and lowest during the summer months. This is shown for London in the curve (p. 298) published by the Registrar-General in his Annual Summary for 1890.

There is also evidence from notification returns that the prevalence of diphtheria, as well as the mortality, is greater during the autumn and winter than during the warmer period of the year.

The same general relation of diphtheria prevalence and mortality to the different seasons of the year would also seem to hold as regards other countries—at least in temperate climates. Hirsch, for instance, found that

the recorded diphtheria attacks in Sweden during the ten years 1861-70 were considerably more numerous in the winter quarters than in the summer.

Diphtheria—London Deaths for all Ages and both Sexes for Thirty Years, 1861-90.



He also found this true of the registered deaths (for differing periods of years) in a number of separate towns, including Berlin, St. Petersburg, Vienna, and Philadelphia. How far the influence of season upon diphtheria prevalence is direct, operating upon the activity of the diphtheria microbe, and how far it is indirect, and operates by increasing the susceptibility of individuals, is doubtful, but it appears probable that it acts in both these ways. There can be little doubt that anything which tends to damage the integrity of the mucous lining of the throat, such as ordinary catarrhs, would so far predispose to attack by diphtheria, given the presence of the efficient cause of that disease; and it is also possible that the influence of cold and damp may increase the general susceptibility of the body to infection. Nevertheless, it seems in a high degree probable that the activity of the diphtheria organism is also largely controlled by season.

Influence of Race.—The balance of present evidence would seem to be to the effect that there is no racial immunity to this disease.

Influence of Sex.—For the first two years of life the mortality from diphtheria is greater among males than among females. But from the third year until about the thirty-fifth it is greater among females. After that period the male mortality seems again to be, if anything, slightly in excess of the female.

The excess of female mortality, at certain ages, at least, is no doubt largely due to greater exposure to infection—that is, to the closer and more continuous contact with the sick to which females are exposed as compared with males; but Dr. Arthur Downes¹ has pointed to the very early ages at which the excess of female mortality is discernible as perhaps indicating that some further explanation is required. As regards this, Dr. Thorne remarks that 'the excess of diphtheria death which attaches to females over males from three to fifteen years of age, increases precisely as the age advances which fits them more and more to take some share in the care of home, and of relations during periods of sickness.'² And he further remarks that 'something may depend upon the full significance of the term "domesticity," and upon its taking account of those acts of affection and tenderness which, in their relation to the sick, characterise females during the period of girlhood, as well as in mature womanhood.' Both these considerations are deserving of considerable weight, especially as regards a disease such as diphtheria, in the dissemination of which close contact is known to play a conspicuous part; but it still appears to remain doubtful whether increased exposure to infection can be regarded as entirely accounting for the excess of female mortality, especially in the very early years of life.

¹ *Practitioner*, vols. xxxi. and xxxii.

² *Milroy Lectures*, 1891, p. 35.

Influence of Age.—The mortality from diphtheria is far greater in the age-group 0–5 years than at any other age-period. For the years 0–5 taken separately, it is decidedly lowest in the first year. Between the second, third, and fourth years there appears to be but little difference, though it is probably at its maximum in the fourth year. After that it begins to decline. During the next five years, however, i.e. the age-group 5–10, the mortality is still about double that at all ages, but it thenceforward falls considerably, and after the fifteenth year is comparatively insignificant. As regards liability to attack, Mr. Power has shown in his reports to the Local Government Board upon diphtheria outbreaks at Brailes (1876), at Radwinter (1877), and at Pirbright (1888), that the attack incidence of the disease, even apart from the influence of school attendance, is greatest upon children between the ages of three and twelve years. A similar result is also brought out by Dr. Thorne in his report upon diphtheria at Coggeshall in 1877. These reports are of especial interest with respect to the age-incidence of diphtheria, and also with respect to the influence of school attendance upon the dissemination of that disease, a subject which will be referred to later.

Cause.—The dependence of diphtheria upon an organism seems now to have been completely established, but for the evidence upon this point reference must be made to Dr. Klein's article. Certain factors which may be regarded as having indirect bearing upon the causation and spread of diphtheria must, however, be referred to here.

Looking at the geographical distribution of diphtheria in England at the present day, and remembering also that at different times outbreaks of this disease have occurred in localities exhibiting almost every conceivable geological variety, one would naturally conclude that geological circumstance had no influence upon diphtheria prevalence. And in the main, perhaps, this is true, as regards the more conspicuous geological features. But there is, nevertheless, a growing opinion among those who have had practical experience of diphtheria outbreaks, especially in rural districts, that a general dampness of soil, whether due to the quality of the soil itself or to its topographical arrangement, does favour diphtheria prevalence; though its influence in this respect may, of course, be masked by other dominant factors. Dr. Charles Kelly, among others, has drawn special attention to this.

Besides an influence due to general dampness of soil, there is a considerable accumulation of evidence in support of the view that special and continuous dampness of dwelling is similarly concerned with diphtheria. This appears to have been first insisted upon by Dr. Thursfield (*Lancet*, 1878), and has since been supported by other observers.¹

As regards the actual dissemination of diphtheria, direct infection from case to case plays an important part. In this process the virus is, doubtless, often given off in the breath from the throat of the sufferer, and inhaled by the recipient; but actual contact, as in kissing, and the conveyance of the virus upon drinking-vessels and spoons, is no doubt responsible for many cases of infection. In addition to the throat and air-passages of the recipient, the poison may attack other mucous surfaces, as the conjunctiva, and wounds of the skin.

In connection with the spread of diphtheria by infection, the influence of school attendance is a matter of great importance. 'That some such influence obtained was one of the earliest observations following an inquiry into the etiology of the disease in this country,' writes Dr. Thorne in his 'Milroy Lectures;' and he then proceeds to discuss fully the nature and extent of this influence. For the detailed evidence upon this subject the reader must

¹ See also Dr. Copeman's article on 'The Influence of Soil on Health,' vol. i. p. 338.

be referred to the lectures in question, but the conclusions at which Dr. Thorne arrives are summed up as follows:—‘School influence would appear, then, to be operative for mischief in a number of ways:—

‘1. It brings together those members of the community who are, by reason of age, most susceptible to diphtheria. 2. The children thus brought together are placed, and remain for many hours of the day, in exceptionally close relation with each other. In this connection, it must be remembered that we are dealing with a disease, the communication of which from person to person is largely dependent on the closeness of the mouth and nares of the recipient individual to the faucial mucous surface of the individual by whom the infection is imparted. And when it is remembered that children are so placed in elementary schools as necessarily to be for long periods inhaling the lung exhalations of their fellows, and that collective singing and a kindred form of general intoning often form part of the school programme, it will not be difficult to understand how especially favourable such circumstances are to the transmission of a specific infection which has a primary seat upon the mucous membrane and in the secretions of diseased fauces. 3. The closer the aggregation, and the greater the lack of ventilation, with corresponding hindrance to the free movement of air, the greater is the risk. 4. Such faulty sanitary circumstances of the school-house and its surroundings, and such other conditions as tend to a condition of general ill-health, in that they induce sore-throat, favour the reception, by children so suffering, of any imported diphtheria infection. 5. There are ample grounds for believing that the aggregation of children in elementary schools constitutes one of the conditions under which a form of disease of particular potency for spread and for death may be, so to speak, manufactured. 6. The danger of school aggregation is by no means limited to the period in which the throat disease is acute. “Recrudescence” of the throat mischief in the individual probably tends to be favoured by conditions intimately associated with school attendance. And similarly, even in the stage of the disease after paralytic neuroses have supervened, infection may be communicated. . . . 7. The practices of kissing and of transferring sweetmeats from mouth to mouth—practices which are more common amongst girls than with boys—as also the joint use of drinking-cups, &c., must, according to experience, be credited with assisting in the diffusion of diphtheria amongst schoolfellows.’

The conclusion expressed by Dr. Thorne in No. 5, above, with reference to the manufacture, under certain conditions of aggregation, of a form of disease of particular potency for spread and for death, opens up a consideration of great importance and interest with respect to the etiology of diphtheria.

It has again and again been observed that outbreaks of typical and fatal diphtheria have been preceded by a more or less widespread prevalence of throat illness of a minor sort, and that even when true diphtheria has appeared upon the scene, the cases of throat illness in different families in the affected locality have presented great variety of type and severity, some of these being true and unmistakable diphtheria, others less typical, and at all events unrecognised as diphtheria at the time of their occurrence, though followed by the neuroses characteristic of that disease; and others, again, cases apparently of quite trivial throat ailment. Moreover, such outbreaks of diphtheria following upon minor throat illness have frequently occurred in particularly isolated localities, and under general conditions which seemed entirely to preclude the likelihood of their having resulted from the importation of specific infection from elsewhere; the whole circumstances seeming strongly to point to the view of the more severe malady having ‘grown’ out of the latter. And so long ago as 1878 Dr. Thorne, referring to such ex-

periences, remarked that they appeared 'to indicate the possible occurrence of what may perhaps be looked upon as *the progressive development of the property of infectiveness.*'

It is, no doubt, true that recently acquired knowledge of the etiology of diphtheria may be regarded as indicating the *possibility* of the specific microbe having been conveyed to isolated localities in ways not hitherto suspected, as, for instance, by domestic animals, or even birds. And it has also to be remembered that there is reason for believing that the poison of diphtheria may remain dormant in the soil or elsewhere for long periods of time, to be again called into activity upon the occurrence of suitable conditions of environment; so that some of the outbreaks of diphtheria in isolated localities, though not traceable to recent throat illness there or elsewhere, may still be connected with past diphtheria in the neighbourhood in question. But, at the same time, the frequency with which true diphtheria is observed to follow upon a prevalence of minor throat ailment is so great as to render it difficult to avoid the conclusion that the former is at times connected with the latter by an evolutionary process. And it has to be borne in mind, moreover, that even in the case of diphtheria occurrences dependent upon the return to activity of 'specific' micro-organisms left in the locality by some previous outbreak of diphtheria, as suggested above, such micro-organisms may, by a more or less prolonged dormancy or saprophytic existence, have largely parted with their virulence; and among the conditions of environment necessary for full establishment of pathogenic function, passage through a succession of especially suitable hosts may hold an important place.

In connection with the sudden appearance of diphtheria in isolated localities, the question of the aerial conveyance of the virus naturally arises. Upon this point the evidence is conflicting. It must be admitted that many cases occur in rural districts which seem, at first at least, strongly to suggest the conveyance of the poison through the atmosphere for considerable distances. On the other hand, the general behaviour of diphtheria in the dwelling and the hospital would seem opposed to this view, for although the infection is no doubt capable of being conveyed through the atmosphere, of the room for instance, for certain distances, yet close association and even actual contact with the sick seem to play a far larger proportionate share in the spread of diphtheria than in that of many other infectious diseases. Moreover, although, as Dr. Thorne says, 'no limit can as yet be assigned to the conditions, as to distance or otherwise, under which the diphtheria contagion can travel through the atmosphere and yet retain its potency for harm,' it will be seen from what has been said above as to other possible modes of origin of diphtheria in isolated localities, that 'it is pretty certain that many occurrences of diphtheria which some years ago might with plausibility have been held to be due to an aerially transmitted infection, would now admit of a different explanation.'

In addition to the methods of diphtheria dissemination already described, it is certain that the infection may adhere to clothing and the like, and thus be transmitted from place to place. It is now, also, well known that the diphtheria virus may be distributed by milk. This was first established by Mr. Power, in 1878, with respect to an outbreak of the disease in North London.¹ Since that time several other outbreaks in association with particular milk services have been brought to light, notably one at Hendon and one at York Town and Camberley, also reported upon by Mr. Power in 1888 and 1887 respectively. It is impossible here to enter into the details

¹ *Report to the Local Government Board on an Epidemic Prevalence of Diphtheria in North London*, 1878.

of these outbreaks, and the reports themselves should be studied. It is sufficient now to say that they have been the means of establishing beyond doubt the fact that milk may act as the vehicle for the dissemination of diphtheria infection. Mr. Power was also able to show good reasons for thinking that the quantity of the milk consumed in the different households, and the storage of such milk before consumption, were important factors in determining the incidence of the disease upon the various households receiving the infected supply. The quantity of milk consumed, he suggested, might operate either as *quantity* or by leading to the more frequent use of milk, and consequent repetition of the act of infection. The storing of the milk, he considered, might be harmful, as giving time for the multiplication of specific micro-organisms, and this Dr. Klein has since shown to be the case.

It is important, however, to note that in none of the instances above referred to could any means be ascertained by which it was likely that the suspected milk supplies had become infected by human diphtheria. This, as well as certain circumstances connected with the outbreaks referred to, raised the question as to whether the cows themselves, suffering, perhaps, from an ailment so slight in appearance as to pass unnoticed by those about the farm, might not have imparted infective quality to the milk. According to the recent investigations of Dr. Klein, it would seem that this question has now been answered in the affirmative.¹

And this leads to the matter, already several times referred to, of diphtheria in the lower animals.

Of late years, evidence as to the participation of the lower animals in diphtheria has been accumulating. Various observers in studying outbreaks of human diphtheria have met with cases of concurrent throat illness of a like kind among domestic animals and birds, particularly cats, sheep, horses, fowls, turkeys, and pigeons; and in some instances strong evidence has been forthcoming that the animal disease in question was derived from or communicated to the human subject.² To the experimental proof of the occurrence of diphtheria in cows reference has already been made, and it may now be added that Dr. Klein has also established, by laboratory experiment, the fact of the occurrence of diphtheria in cats.

Drainage defects, filth accumulations, and the like, have very generally been held to have concern in the origin and propagation of diphtheria. For years, indeed, it was commonly believed that circumstances of this kind constituted the main cause of diphtheria, which was accordingly regarded as essentially a filth disease. It must be admitted that a view so widely held, not only by the public, but also by the medical profession, must, in all probability, have had some basis in fact. Nevertheless, the knowledge gained more recently as to the natural history of diphtheria has largely tended to modify the belief in question.

It may be conceded at once, as being in a high degree likely, that such unwholesome conditions do, by lowering vitality, both predispose to infection and add to the severity of the disease, if contracted. Moreover, it would seem reasonable to suppose that drain air, or, for that matter, coal gas, would, by giving rise to a relaxed and unhealthy condition of the mucous lining of the throat, and by thus inflicting injury at the special seat of invasion of diphtheria, increase the liability to attack by that disease in the event of exposure to specific infection. And, further, the possibility of the diphtheria

¹ See page 161, *ante*.

² See Report by Dr. George Turner in the *Report of the Medical Officer to the Local Government Board*, 1886. Also Report by Dr. Bruce Low on an Outbreak of Diphtheria at Enfield in the *Report of the Medical Officer to the Local Government Board*, 1888.

bacillus finding its way into sewers, and hence into houses imperfectly disconnected from such sewers, cannot be denied. But, on the other hand, it has to be remembered that modern inquiry has tended to emphasize the share taken by more direct infection in the dissemination of diphtheria—either infection from recognisable forms of the disease, or from those minor, and perhaps unrecognisable, forms which it is now known may be capable of giving rise to severe and fatal diphtheria. And other means of infection, which were hitherto unknown, such as by specifically contaminated milk, or domestic animals, have also to be borne in mind. It is therefore certain that, as in the case of supposed infection through the medium of wind currents, so here, many cases of diphtheria which it has been customary to attribute to drain defects—and drain defects, moreover, which notwithstanding modern improvements may probably still be detected in very many houses as well where diphtheria is absent as where it is present—might now, with due care, be traced to one or other of the causes indicated. Lastly, if we take a broad view of the matter, we see two strong grounds for discrediting the popular notion that drainage defects and other filth nuisances play a prominent part in the causation of diphtheria: 1. Diphtheria is steadily increasing, in spite of the considerable and progressive diminution in the unwholesome conditions of the sort in question, whereas the reverse is the case with regard to enteric fever, a disease which is known to be controllable by sanitary improvements. 2. The geographical distribution of diphtheria in England lends no support to the view that that disease especially affects those areas in which filth nuisances abound or defective sewers are the rule. In referring to the results of his studies of the geographical distribution of diphtheria, Dr. Longstaff says: ‘The practical deduction suggested by these facts is, that the cause, or causes, of diphtheria should not be sought for primarily in any high development of civilisation, such as sewers, but rather in some condition associated with a more primitive mode of life. Again, privies and ashpits can hardly be important agents in breeding or disseminating the disease, or we should expect to find diphtheria exceptionally prevalent in those Northern towns where such nuisances reach their worst, whereas the contrary is the case.’

It has been suggested that diphtheria may be disseminated by the agency of drinking-water, but the evidence at present is against such being the case.

Assuming that the diphtheria bacillus may at times find a habitat in the surface soil, a rise in the subsoil water may, by expelling the ground air, lead also to the expulsion of the bacillus, and consequently have concern in the origination of some diphtheria outbreaks, as suggested by Mr. M. A. Adams; and this is a matter well worthy of future study.

Relation of Diphtheria to other Diseases.—Diphtheria, or what is certainly, from a clinical point of view, diphtheria, and is followed by the characteristic paralysis, is not infrequently observed in concurrence with, but more frequently in sequence to, both measles and scarlet-fever, and less frequently perhaps, enteric fever. Some interesting cases of associated scarlet fever and diphtheria will be found recorded in a paper by Dr. H. F. Parsons in the *Epidemiological Society ‘Transactions’* for 1888–84, and the meaning of their association is also there discussed. Speaking generally, it may be anticipated that diseases, such as scarlet fever and measles, which especially lead to more or less temporary damage to the mucous membrane of the throat would predispose to the reception of the diphtheria poison, and it seems likely that this is the true explanation of many of the instances of diphtheria following upon the diseases in question. At the same time, the association between

diphtheria and scarlatina, at all events, seems at times to be an extremely intimate one, and in individual cases the co-existence of the two viruses in the same body is at least a possibility. Indeed, there would seem no theoretical ground for altogether denying the possibility of such a co-existence of the viruses extending beyond the single case, and giving a hybrid character to other cases.

When diphtheria is prevalent, there is very commonly an increase in the number of deaths registered as membranous croup. This, however, is apparently a matter of nomenclature and erroneous diagnosis, the so-called croup being, no doubt, laryngeal diphtheria. Similarly, diphtheria deaths are often registered as mumps, laryngitis, and tonsillitis.

Period of Incubation.—Most observers regard the period of incubation of diphtheria as being a short one, usually two or three days, but as liable to vary from one to six days or so. Some authorities, on the other hand, give a much wider variation, as, for instance, from one day to a fortnight. As regards unusually long incubation periods, Dr. Thorne points out 'the possibility of one of those mild sore-throat attacks from which adults at times suffer during diphtheria epidemics, and this without known inconvenience,' having preceded in the individual the attack which attracted notice; the latter being, in fact, recrudescence of the disease in the individual in question. It is also not unlikely that the period of incubation varies with the quality, and perhaps with the dose, of the virus, as well as with the condition, especially as to the throat, of the recipient. In milk epidemics the period of incubation seems usually to be short, a circumstance most probably due to 'conditions, such as storage of the supply, [which] have favoured the multiplication of the bacilli and subsequent formation of chemical poison, as also the direct reception of the poison into the stomach. . . .'

Period of Infectiveness.—This is variously stated by different observers as from fourteen days to eight weeks. Much, of course, must depend upon the circumstances of the individual case; but it may well be suspected that considerable harm in the way of the spread of diphtheria is done by underestimating this period, and allowing diphtheria convalescents to mix with the healthy, and perhaps return to school while still in an infective condition. Moreover, in this connection the observations of Dr. Astley Gresswell, which seem to show that in certain individuals diphtheria may, so to speak, become chronic, and subject from time to time, especially upon exposure to cold and damp, to recrudescence, are of the utmost importance, and should be carefully studied.¹

Protection.—It is usually said that diphtheria confers no immunity against subsequent attack. Certainly, second and third attacks in the same individual are not unfrequently met with, but how far the later attacks are due to a fresh infection, and how far to rejuvenescence of a dormant virus left by an earlier attack, as suspected in some cases by Dr. Gresswell, remains an open question.

¹ 'Diphtheria as a Chronic Malady in Particular Individuals, with Liability in them to Recrudescence' (*Trans. Epidem. Soc., N.S.*, vol. v., 1885-86).

CEREBRO-SPINAL FEVER

Synon.: *Epidemic Cerebro-Spinal Meningitis*; *Cerebro-Spinal Arachnitis*; *Cerebral Typhus*; *Malignant Purple Fever*; *Spotted Fever*. Fr. *Méningite Cérébro-spinale Épidémique*; Ger. *Epidemische Meningitis*; It. *Febbre Cerebro-spinale*.

History.—The more important history of this malady dates from the year 1887. Medical records, however, bear testimony to its prevalence in Switzerland, France, and particularly America, during the early years of the century. Some French writers, indeed, have considered that they could trace it in the literature of far earlier times; but upon this point the evidence, according to other authorities, notably Ziemssen and Hirsch, is of a doubtful nature. However that may be, the disease appeared in epidemic form in the South of France about the year 1887, being apparently at this time first observed among the troops at Bayonne, but almost simultaneously at Foix and Narbonne. During the next five years it extended over a large part of France, spreading, it would seem, from the centres mentioned. In its progress through France its incidence upon the military population was conspicuous, and it is interesting to note, as bearing upon the mode of its spread, that upon several occasions its appearance in particular localities seems clearly to have been due to importation by recently arrived troops. The following is a brief summary of this French epidemic:—

About the time of, or shortly after, its appearance at Bayonne, the disease prevailed up the banks of the river Adour, in the department of the Landes, and among the troops at Bordeaux and La Rochelle. In the following year it broke out at Rochefort among soldiers who had arrived there from the Landes, and in the next year (1889) it appeared at Versailles, again among the same soldiers, who had been moved there from Rochefort. In 1889 it also broke out in the garrison at Metz, and reappeared at Bordeaux; in 1840 it broke out at Strasburg, and a second time at Bayonne; and in 1841 in the garrison at Nancy—being carried in the meantime by detachments of troops to a number of adjacent places, such as Schlettstadt, Hagenau, Buxweiler, and Wasselonne (Hirsch). At Laval the disease had broken out in the garrison in the spring of 1840, and thence, in the following winter, it was seemingly carried to Le Mans, appearing about the same time at Blois, Tours, Poitiers, and other places around. In 1840–41 it was also present in the garrisons of Brest, Caen, and Cherbourg; and in 1842 there were a number of cases in Paris.

It has already been seen, however, that simultaneously, or almost simultaneously, with its appearance at Bayonne in 1887 the disease had also broken out at Foix and Narbonne. From this centre, apparently (1888) it spread to Hers, near Toulouse, and to the troops at Nîmes and Toulon. About a year later it appeared in the garrisons at Avignon, Perpignan, and Montbrison, and in the years 1841–42 at Marseilles and Lyons.

After a general abatement throughout France from 1842 to 1845, several more or less extensive epidemics again occurred in that country between 1846 and 1850. In its reappearance the malady seems first to have broken out in localities which had been affected by it during the previous epidemic of 1887–42, such, for instance, as the garrisons of Avignon, Lyons, Nîmes, Toulouse, and Metz.

During the period under consideration (1837-50) cerebro-spinal fever was somewhat widely prevalent in Southern Italy, where it seems first to have occurred in 1839-40, and to have continued to prevail until 1845. In 1840, also, it broke out in Algiers, probably in connection, as Hirsch remarks, with its prevalence in France, and the French troops at Douera were among those who were early attacked. In 1842 the disease became prevalent in the United States, where it was first observed in Tennessee and Alabama, subsequently becoming epidemic in a number of other States between 1842 and 1850.

During the year 1843 there was a malignant epidemic at Corfu, and a minor epidemic at Gibraltar in 1844. In Denmark the disease appeared in 1845. In the latter year, also, cases occurred in the Dublin and Belfast workhouses,¹ and in Liverpool. A few cases were also observed in Ireland in 1850. From 1850 to 1854 there seems little record of cerebro-spinal fever; but the latter year saw the beginning of another widespread epidemic diffusion of it, and the disease continued active in one country or another until about the year 1876. During this period the countries most severely affected were Sweden, Germany, Russia, Greece, Italy, and especially America. It was present also, but in a minor degree, in Great Britain, Denmark, Norway, France, Switzerland, Austria, Hungary, Turkey, and Portugal, Jerusalem, Persia, and Algiers. It was earliest observed in Sweden, where some cases occurred at Gothenburg in 1854; but it did not become truly epidemic in that country until the following year. It then slowly extended over the south of Sweden, dying down during the latter half of each year, to reappear early in the following year. Each year, too, it travelled farther northwards, until 1868, when it had reached the latitude of 68° N. Subsequently to this the disease was more or less present each year, but with gradually decreasing intensity, and chiefly in localities in which it had previously occurred, until 1865, when it became again more prevalent. But it finally died out in 1867.

Early in 1859 the disease had broken out in Norway at Opdal, in the province of Hedemarken, which adjoins Sweden. If, as there seem grounds for thinking, Sweden had previously to 1854 been free from cerebro-spinal fever, the probability of the malady having at this period found its way there from Denmark, where it had been prevalent a few years before, is obvious.

The country which especially suffered during the period under consideration was the United States of America. The disease broke out in 1856-57, both in North Carolina and the New York State, and between the latter year and 1874, according to Hirsch, 'scarcely a year passed without its being seen over a larger or smaller area, its diffusion from first to last covering the whole country.' In the Civil War, it was particularly prevalent among the troops, attacking also the negroes in the Confederate army.

In Germany, the disease was observed on a small scale in Silesia during the year 1868, and became more conspicuously prevalent in the winter of 1868-64, when it appeared at many different places in Posen, Brandenburg, East and West Prussia,² and Pomerania, remaining 'epidemic therein until the end of the following winter' (Hirsch).

In Hanover and Brunswick it was epidemic in the winter and spring of 1864-65. During the same period, but commencing somewhat later, it was also present in Bavaria, Hesse, Baden, and Würtemberg. By the end of 1866 the German epidemic had spent itself, and the disease became limited

¹ See paper in *Dublin Quarterly Journal* of 1846 by Dr. Robert Mayne.

² A report by Dr. Burdon Sanderson upon the epidemic at Dantzic in 1865, will be found in the Annual Report for that year by the Medical Officer to the Privy Council.

to quite small epidemics and sporadic cases, chiefly in the colder seasons of the year.

As regards Austria-Hungary, some cases occurred in Vienna in the spring of 1868—that is, it will be remembered, the year in which we have record of its appearance in Silesia. Other prevalences occurred at Gömor in Hungary, and Pola and Trieste in Austria, during the three following winters. In the same year it broke out in Russia and Greece, afterwards spreading widely in those countries. In Italy it did not, apparently, during this epidemic, appear until 1878. As regards Great Britain, a rather important outbreak occurred, notably among soldiers, in Dublin and other parts of Ireland, during the winter of 1866–67. Several interesting papers upon this epidemic were read before the Medical Society of the Irish College of Physicians, and a long discussion upon them took place.¹ The outbreak produced considerable sensation, and some observers were inclined, in view of the high mortality which attended it, and the purpuric rashes which occurred in some of the cases, to regard it as a reappearance of the 'Black Death'—a view, however, which was soon abandoned. About the same time some cases were observed near Lincoln;² but the disease does not appear to have been conspicuously prevalent in England.

Subsequently to 1876, cerebro-spinal fever, judging from the scanty records of it, seems to have generally abated. Some minor epidemics have since been observed, as, for instance, that in Fiji in 1885,³ in Dublin in 1885–86,⁴ and in the basin of the Mediterranean in 1887–88.⁵ In 1890, also, some cases, apparently of this disease, occurred in the Eastern Counties of England, especially at Oakley, in Suffolk. The Oakley cases unquestionably constituted a small epidemic of a malady having for its main characteristic cerebro-spinal symptoms—vertigo, headache, great drowsiness, marked retraction of the head, and in some cases opisthotonos and subsequent paralysis. In certain instances other symptoms also suggestive of true cerebro-spinal fever were observed, such as conjunctivitis, herpes of the mouth, and a rash 'like chicken-pox.' There was, too, as Dr. Low points out, 'a definite group of cases, with associated scattered cases in the neighbourhood, but with no apparent connection between them,' such as has been commonly observed in respect to cerebro-spinal fever. In two points, however, the Oakley outbreak differed from some of the more typical epidemics of cerebro-spinal fever as that disease has been seen in other countries, viz. in its low fatality and in the frequency with which multiple cases occurred in the same household. But neither of these differences could be held as establishing a distinction from true cerebro-spinal fever. It has several times been observed that well-marked epidemics of this latter disease have been accompanied by numbers of cases exhibiting somewhat similar symptoms to the typical malady, but of so mild a character as almost to escape attention. There would, therefore, seem no reason why a particular localised epidemic should not consist wholly of such relatively mild attacks. As regards the multiple cases, although it has been stated of certain important and well-marked epidemics of cerebro-spinal fever that multiple cases in households have been conspicuous by their absence, yet this has not always been the case.

¹ *Medical Press and Circular*, June 5, 12, and 19, 1867.

² *Lancet*, 1867, 'On an Epidemic of Cerebro-Spinal Meningitis,' by G. M. Lowe, M.D.

³ See the account in the *Trans. Epidem. Soc.*, Lond., N.S., vol. vii., by Mr. B. G. Corney.

⁴ *British Medical Journal*, June 26, 1886.

⁵ Notes by Dr. Thorne in the *Report of the Medical Officer to the Local Government Board*, 1888.

On the other hand, having in view the prevalence of influenza in this country during the early part of the year 1890, the protean character of that malady, and the fact that in the first instance its main assault appears to be upon the cerebro-spinal system; and coupling with these facts the previous relative freedom of England from cerebro-spinal fever, it might no doubt be suggested that the low mortality and multiple cases in the Oakley outbreak were rather suggestive of a form of influenza in which the cerebro-spinal symptoms were more than usually prominent, than of cerebro-spinal fever. This hypothesis might, perhaps, be thought to be strengthened by the interesting account given later, by Dr. Bruce Low, of another series of cases of 'anomalous illnesses' in certain localities in Northamptonshire. In these latter cases pneumonia was the more prominent feature, and although decided meningeal symptoms were observed, they do not appear to have been so constant or so conspicuous as among the Oakley cases. As at Oakley, they were characterised by a low mortality, and multiple cases in households were common.

Nevertheless, it is to be noted that, as regards age incidence, the Oakley and Northamptonshire cases apparently differed from influenza, and although not in accord with some experiences of cerebro-spinal fever in this respect, the balance of evidence seems to point to that disease rather than to influenza.

Mortality.—Cerebro-spinal fever has at times given rise to considerable mortality. According to Hirsch, 4,577 deaths were officially assigned to this disease in Sweden during the periods 1854–60 and 1865–70, in which it has been seen the disease was prevalent in that country. In the epidemic about the Lower Vistula in 1865, at least 1,000 persons died of the disease in the Circle of Dantzic during the first few months of the year.

As regards this country, reference to the Registrar-General's Annual Reports shows that the deaths recorded annually in England and Wales as due to 'cerebro-spinal fever' during the fifteen years, 1876–1890, have ranged from 18 in 1888 to 58 in 1878. These sporadic cases, however, 'scattered broadcast over the country,' cannot, as the Medical Officer to the Local Government Board suggests in his Annual Report for the year 1888, with any confidence be affirmed as of the same nature as those seen in epidemic form elsewhere, though there is, 'in the fact of the occurrence of any such deaths in England, reason for watchfulness on the part of Health Authorities and their officers.' It may also quite well be, as Dr. Bruce Low suggests, that true cerebro-spinal fever is of more frequent occurrence than is generally supposed, owing to fatal cases being certified as 'fever,' tubercular meningitis, &c., and non-fatal cases being mistaken for some other ailment.

Fatality.—The fatality in well-marked epidemics has usually been very great. According to Boudin's statistics, quoted by Sir John Simon, 809 deaths occurred among 1,804 patients—a fatality of 62 per cent. In the worst epidemics the fatality, Sir John Simon points out, 'seems to have been as high as 80 per 100.' Dr. T. W. Grimshaw, in Quain's 'Dictionary of Medicine,' gives the fatality in some of the American epidemics as 75 per cent., and states that among the Irish Constabulary it reached 80 per cent.

It is, however, not improbable that the estimates referred to are somewhat too high, for, as Sir John Simon points out elsewhere in the same memorandum, while cases of the gravest kind 'are occurring, perhaps, not very numerous in a place, sometimes within the same area, or scattered over a wider one, a large number of other persons will suffer slight indications of similar nervous derangement. Headache, vertigo, muscular discomfort in

the head and limbs, and attacks of chilliness, are the chief of these minor indications, which apparently bear to the graver cases the same sort of relation as epidemic diarrhoea bears to concurrent epidemic cholera.'

If these milder cases could have been estimated, the fatality would probably turn out to be lower than it has usually been stated to be.

Influence of Race, Sex, and Age.—So far as present experience goes, all races would appear to be subject to this disease if brought within the range of infection. In various American epidemics the negroes have suffered excessively, but whether this was due to special susceptibility *qua* race, or to conditions of life, is doubtful.

It is usually stated that the disease is more frequent among males than females; Hirsch, however, is of opinion 'that there are no considerable differences discoverable' in this respect.

As regards age, cerebro-spinal fever has been known to occur in persons of all ages, though least frequently among those beyond middle life. According to Dr. Grimshaw, 'the disease usually attacks those approaching the age of puberty or in early adult life,' and 'robust males between the ages of fifteen and thirty are its favourite victims.' This latter statement, no doubt, seems to derive some support from the experience of military epidemics; but the incidence of the malady upon troops is clearly due to some factor other than age; and where the civil population has been attacked, children have often suffered severely. Hirsch gives a long list of epidemics in which children up to the age of about fifteen years have suffered almost exclusively, and he remarks that 'it has been much less common for patients of from twenty to thirty years of age to outnumber the children and youths.'

Influence of Climate and Season.—Hitherto cerebro-spinal fever has in the main been limited to temperate and sub-tropical latitudes, and there seems little evidence of its having prevailed extensively in the tropics, though, as we have already seen, it has occurred in the Fiji Islands. In all countries in which it has at present been observed in epidemic form it seems to have been decidedly most prevalent in the winter and spring, again and again dying down at the approach of warmer weather, to reappear with the ensuing cold season. In the case of Fiji, also, we learn from Mr. Corney that 'the period at which the epidemic prevailed was the cool season of the year, in 1885, an unusually cool one for Fiji . . .'

Cause and Mode of Dissemination.—One of the most noticeable features in the behaviour of this disease, as judged by its past history, is its marked incidence upon troops and persons living together in public institutions, such as workhouses and prisons. The French epidemic of 1837 and the following years was largely among the military, and in America, Ireland, and other countries, the soldiers have at different times been observed to suffer heavily. Similarly, in certain epidemics it has been the inhabitants of prisons and workhouses that have almost exclusively been attacked.

This circumstance might at first sight be regarded as indicating infection as at least a prominent factor in the spread of the disease. Nevertheless, the detailed evidence, though somewhat conflicting, is generally held to be opposed to this view. Thus, with respect to the Dantzic epidemic, Prof. Burdon Sanderson reports that he met with no facts 'which afforded ground for believing that epidemic meningitis was capable of being communicated by personal intercourse,' while, on the other hand, he found evidence to the contrary effect. In the town of Dantzic, for instance, the average number of persons living in a house was, he states, eighteen, but he did not hear of a single instance, up to the time of his inquiry, of two persons suffering in the same house. Influenced by experience of the same kind, and by the

allied observation that those in attendance upon persons suffering from this disease, and other patients associated with such persons in general hospitals, are but very rarely attacked, most authorities have expressed themselves decidedly against the spread of the disease by infection. In this connection, however, the following remarks by the Medical Officer to the Local Government Board, in his Annual Report for the year 1888, are of much interest: 'The disease that was epidemic in Cyprus [1888] did not appear to be infectious from person to person, and in this respect it corresponded with the outbreaks that were observed in 1864-65 in villages on the Lower Vistula, and that were the subject of report by Dr. Sanderson to the Medical Department of the Privy Council (Eighth Report of Medical Officer). But for some years there have been observed in various countries (Landesbezirke) of Prussia (notably in certain towns of Oppeln County, in Upper Silesia), outbreaks of cerebro-spinal meningitis in which German medical observers believe they have detected a certain infectious quality, and in respect of which the Prussian sanitary authorities enjoin that every precaution be taken that is proper to be taken in the case of definitely contagious maladies.' The evidence at present, therefore, would seem to suggest, either that cerebro-spinal fever varies considerably as regards infectiveness, or that different maladies have so far been confused under one common name.

On the whole, as regards well-marked cerebro-spinal fever, the evidence perhaps rather goes to support the view expressed in 1865 by Sir John Simon, that 'if directly communicable from person to person [cerebro-spinal fever] is communicable only in a very low degree. Such communicability as is familiar to us with typhus, small-pox, and other eruptive fevers, cerebro-spinal fever does most assuredly not possess.'

According to Fagge, 'one suggestion is that a contagious principle is given off by the sick, but that it has to undergo some transformation or intermediate stage of its development, possibly in another animal, before it can infect a human being;' and he adds that it is stated, 'on the authority of Mr. Ferguson, Veterinary Surgeon to the Privy Council in Ireland, that on each occasion when the disease has prevailed in that country it has co-existed with an epizootic of the same nature among pigs and dogs.'¹

But if not directly communicable, in any high degree at least, there are strong grounds for regarding the disease as communicable in the sense of being transportable from place to place by infected persons and, perhaps, infected things. By some the disease has been thought of as due to a widespread influence independent altogether of human movements, and in support of this view it has been pointed out that on particular occasions cerebro-spinal fever has appeared simultaneously at widely separated localities. Obviously it may be true, given the essential cause of the disease at the spots in question (left, perhaps, by some previous outbreak), that certain widespread meteorological conditions may determine a simultaneous outbreak in places remote from one another. Or, again, it may conceivably be that, under the circumstances of modern life, such widespread conditions are capable of imparting to micro-organisms not usually pathogenic, ability to produce cerebro-spinal fever. But it must be remembered that, as regards outbreaks of the kind in question, it is extremely difficult to exclude the possibility of importation of the disease by human agency, and there is nothing improbable in the notion of its simultaneous importation at different places.

On the other hand, a broad view of the history of the disease decidedly supports the view that human intercourse holds a very important place in its spread. Taken generally, the march of cerebro-spinal fever has not

¹ Fagge, *op. cit.*, i. 697.

been particularly rapid, and the disease has largely spread circumferentially from previously invaded districts. Lastly, as regards particular places, there has been very strong suggestion of importation.

Ziamssen, Hirsch, and some other authorities, are agreed that circumstances of life have concern in the prevalence of this disease, and that persons living under conditions of overcrowding, defective ventilation, and filth, are more liable to suffer than those more favourably situated. In many epidemics among the civil population the disease is said to have been confined, or almost confined, to the most insanitary parts of the towns invaded; and when troops have mainly suffered, the officers have often been observed to enjoy a marked immunity. On the other hand, Professor Burdon Sanderson did not associate the outbreak in the Lower Vistula with unwholesome conditions; and as regards the Oakley outbreak, Dr. Bruce Low found the conditions of life among those affected no better and no worse than those of their class generally.

Among other predisposing causes, experience of military outbreaks seems to show that excessive fatigue is of importance, and apparently it has sometimes been practicable to make an epidemic of this disease decline by lightening the men's duties.

Considerable importance has also been attributed by some observers to dampness of soil as an etiological factor in this disease, and it has been suggested, in fact, that cerebro-spinal fever is of malarial origin. Hirsch, on the other hand, opposes the malarial theory, pointing to the prevalence of this disease in high and dry localities. Conversely, he states as regards France, that 'it is precisely the great marshy districts that have been least touched by it.' As pointing to the same conclusion he also refers to the fact of cerebro-spinal fever usually occurring in the winter.

In view of the frequency with which cerebro-spinal fever has been observed to occur in the colder seasons of the year, cold has from time to time been regarded as its ultimate cause. The association between cold and this disease is, however, by no means so constant as to support such a thesis. There are not a few epidemics on record in which the disease has survived the cold of winter, continuing to spread, or in some cases breaking out, in the warm weather of late spring, or even summer (*see* Hirsch). Clearly, then, the relation between cold weather and cerebro-spinal fever, although no doubt in a sense real, is indirect. For the essential cause we must search deeper, and in the present day it will hardly be denied that the general features of the disease in question point strongly to a micro-organism as that cause. We are even perhaps justified in going so far as to suggest that the organism probably belongs to the class of 'facultative parasites,' being capable of thriving and multiplying outside human or animal bodies.

Suggestions of this are found in the fact that the disease has often broken out in districts which have suffered from previous epidemics; that upon its first introduction into a district it has on some occasions exhibited but a minor manifestation during the first season of its appearance, becoming decidedly more prevalent during the next cold season; that while it is not conspicuously infectious, it is apparently transportable; and lastly, that in not a few instances it has shown a decided tendency to cling to, and practically limit its manifestation to, the inhabitants of particular public institutions, such as barracks, and even to particular parts of these institutions. All these facts, though no doubt capable of other explanations, are at least consistent with the hypothesis of the ability of the micro-organism to carry on a saprophytic existence, perhaps in the soil, and perhaps also in the dust and dirt accumulated between the boards of barrack-room floors.

SIMPLE CONTINUED FEVER

Synon. : *Febricula*. Fr. *Fébricule*; Ger. *Febricula*; It. *Febbricola*.

From time to time cases of more or less transient fever are met with which are unattended by any definite and constant symptoms, other than those commonly associated with pyrexia. The difficulty of classing such cases with any of the more specialised fevers long ago gave rise to the notion of a 'simple continued fever'—a notion which apparently dates back to the days of Hippocrates, and has survived to our own time. 'But most physicians now believe that a disease without any morbid anatomy, without any known etiology, and without any definite or characteristic course or symptoms, cannot be admitted into a useful nosology.'¹ Moreover, it is now becoming generally recognised that the cases of feverishness grouped together under one or other of the above names are, in reality, due to a variety of causes. Even Murchison, who himself described a simple continued fever, remarks that 'many cases are designated simple fever, or febricula, which are in reality mild or abortive cases of typhus or enteric fever, or relapsing fever without a relapse, or catarrh with an unusual amount of febrile disturbance.' And such is no doubt the fact, the cases of so-called simple continued fever being largely made up of atypical examples of the particular fever prevailing at the given time and place.

Some cases, too, are doubtless symptomatic of gastric or intestinal disturbance, tubercular mischief, and other specific or non-specific causes.

In the following table, however, will be found the number of deaths recorded by the Registrar-General year by year from 1869 to 1891 as having, in England and Wales, been ascribed to 'simple and ill-defined fever.' Prior to 1869 the Registrar-General did not separate these deaths from those due to the other continued fevers.

TABLE X.—*Showing the Deaths registered in England and Wales during the Years 1869-1890 as due to Simple and Ill-defined Fever.*

Year	Deaths	Death-rate per million living	Year	Deaths	Death-rate per million living
1869	5,810	289	1881	1,159	44
1870	5,254	233	1882	1,016	39
1871	4,248	186	1883	963	36
1872	3,352	145	1884	768	28
1873	3,081	132	1885	662	24
1874	3,089	130	1886	605	22
1875	2,599	108	1887	507	18
1876	1,974	81	1888	436	15
1877	1,923	78	1889	413	15
1878	1,776	71	1890	361	13
1879	1,472	58	1891	325	11
1880	1,490	58			

On reference to the table it will be seen that the registered mortality from 'simple fever' has undergone a very marked diminution during the period in question—a diminution, in fact, very similar to that exhibited by the typhus mortality. This is, no doubt, partly owing to improved and

¹ Fagge, *op. cit.*, i. 207.

more careful diagnosis ; nevertheless, it is probably also in no small degree due to the diminished prevalence of certain other fevers, such as enteric, and more especially typhus fever—diseases which, it cannot be doubted, were responsible for a considerable share of the deaths previously recorded as due to simple fever.

Murchison believed that the recorded 'simple fever' deaths were 'due, for the most part, to enteric fever with latent abdominal symptoms.' This, however, would hardly seem to have been the case.

Dr. G. B. Longstaff¹ has shown that the seasonal mortality curve of simple continued fever is quite unlike that of enteric fever. The curve of the latter disease has a well-marked autumnal maximum, whereas in the case of the simple continued fever curve 'there is not a trace of this.' On the other hand, Dr. Longstaff does find 'some sort of resemblance' between the seasonal curves of typhus and simple continued fever.

For the present the matter of main importance, from a public health point of view, is that mild cases of fever, which do not present any well-defined symptoms, should not be hastily classed as 'simple continued fever'—a separate disease being thus assumed, which, perhaps, has no existence. Careful inquiry will often throw light upon the causation of such attacks. If, for instance, one or other of the recognised fevers be prevalent in the neighbourhood at the time, and more especially if it should be ascertained that the 'febricula' patients have been exposed to likelihood of infection by such fever, there will be grounds for thinking it probable that the apparently simple feverish attacks are in reality but mild and irregular examples of the more serious malady. Under such circumstances, the interests of the public, as well as those of the patients themselves, obviously demand that the cases should be regarded as of the graver sort, all the precautions being observed which would be considered necessary with respect to the particular fever prevailing.

ENTERIC FEVER

Synon.: *Typhoid Fever* ; *Pythogenic Fever* ; *Gastric Fever* ; *Low Fever* ; *Infantile Remittent Fever*. Fr. *Fièvre Typhoïde*, *Dothiëntérie* ; Ger. *Typhus Abdominalis* ; It. *Tifo Enterico*.

History and Geographical Distribution.—There are few more interesting and instructive pages in the history of medicine than that which records the gradual differentiation of enteric from typhus fever—a differentiation only effected after prolonged and painstaking study of the pathology of these two diseases. 'The doctrine,' Hirsch remarks, 'of the so-called "abdominal typhus" counts among the acquisitions which medicine owes chiefly to the methodical investigation of morbid anatomy.'

It is often supposed that enteric fever is a disease of modern times. History, however, does not seem to support this view, but rather goes to show that it is the differentiation of the disease which alone is modern.

The late Dr. Murchison was of opinion that 'some of the descriptions of the Greek writers probably referred to enteric fever,' and he

¹ 'The Seasonal Prevalence of Continued Fever in London' (*Epidem. Soc. Trans.*, N.S. vol. iv.). Reprinted in *Studies in Statistics*.

points out that in the course of two successive autumns Hippocrates 'met with many cases of fever of the continual type, characterised by diarrhoea, offensive watery stools, bilious vomiting, tympanitis, abdominal pain, "red rashes," epistaxis, sleeplessness, or a tendency to coma, delirium, and subsultus, irregular remissions, a lengthened duration, and great emaciation.' Whether or not such early writings are to be taken as referring to enteric fever, the descriptions of various observers in the seventeenth century, notably Spigelius, Panarolus and Baglivi for Italy, and Willis and Sydenham for England, seem to leave little doubt that the disease was prevalent in their day, several of them referring to intestinal lesions and a general course of symptoms highly suggestive of enteric fever. Both Willis and Sydenham, according to Murchison, regarded this fever as differing from *febris pestilens* [typhus]. In the eighteenth century there was an increasing recognition of a form of fever described variously as slow fever, nervous fever, or low continued fever, which differed from ordinary typhus in the insidiousness of its course, the attendant diarrhoea, and the tendency to prove suddenly fatal, and which was accompanied by inflammation or ulceration of the bowels. The more important accounts of this malady came from Hoffmann of Halle (1698-1728), Strother of London (1727-29), Gilchrist of Dumfries (1785), Huxham (1789), Sir Richard Manningham, F.R.S. (1746), Riedel (1748), and Roderer and Wagler, who described an epidemic of *morbus mucosus* at Göttingen in 1760.

That a doctrine of enteric fever as a disease distinct from typhus was at this time gaining ground seems evident from the circumstance recorded by Murchison, that 'Dr. Erasmus Darwin, of Derby, in a letter addressed to Dr. Lettsom in 1787 proposes as a question for discussion at the Medical Society, "Whether the nervous fever of Huxham be the same as the petechial or jail fever,"' and that 'Dr. Willan, in 1799, observed that Cullen had "improperly comprised under the term typhus the slow or nervous fever described by Gilchrist and Huxham, which may rather be considered a species of hectic, and is not received by infection."'

During the early years of the present century an increasing amount of attention was bestowed upon the intestinal lesions met with in cases of fever. These lesions were particularly studied in France by Prost, Pettit, and Serres, the latter observers stating that the lesions were limited to the lower part of the ileum; Bretonneau, who showed that the disease was localised in the solitary and agminated glands of the ileum; and Louis, who first gave to the disease the name of *fièvre typhoïde*.

All these French observers, however, appear, according to Murchison, to have regarded the disease they were studying as really the same disease as the typhus of camps and armies. This was doubtless owing to the fact that during this period enteric fever was common in France, while true typhus had almost entirely disappeared. Hence it was not unnatural that, upon finding intestinal ulceration very generally present in cases which were then regarded as typhus, the French physicians should have been inclined to regard such lesions as associated with that disease.

During the same period, i.e. the first half of the present century, the foundation for the differentiation of enteric fever was being pushed forward in other countries. Hildebrand, in 1810, distinguished between so-called contagious typhus and non-contagious nervous fever; and other German physicians soon after followed on the same lines, among them Schönlein, who introduced for the latter disease the term *typhus abdominalis*, which is still adhered to in that country. In England, during the first twenty years

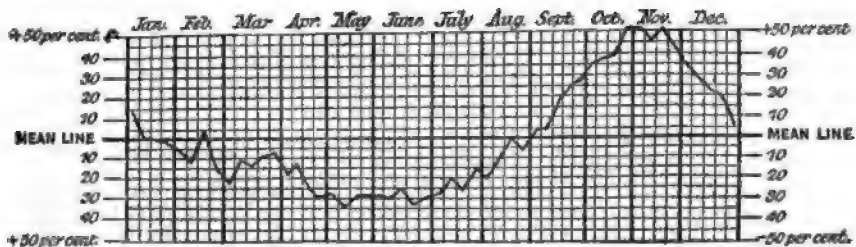
of the century several outbreaks of enteric fever seem to have been recorded, and to have been recognised as differing from typhus. The more important of these were an epidemic at Deal in the year 1806, recorded by Dr. Thomas Sutton; another at Paisley in 1811, recorded by Dr. Muir; and one at Newcastle in 1817, described by Mr. Henry Edmondstone. In 1826, Dr. Hewitt, of St. George's Hospital, published some important observations upon the pathology of this disease, which, Murchison says, met with unmerited neglect. They were 'published almost simultaneously with those of Bretonneau; and, like his, they showed that the seat of the lesion was in the solitary and agminated glands of the ileum.' Later on the separate nature of the two diseases was more fully recognised and insisted upon by Drs. Perry of Glasgow, Lombard of Geneva, Gerhard and Pennock of Philadelphia, Shattuck of Boston, U.S.A., and especially by Dr. A. P. Stewart of Glasgow. But it was reserved for Sir William Jenner, in 1849-51, to finally establish the non-identity of enteric and typhus fevers.

Sir William Jenner indicated more fully and more clearly than had hitherto been done the differences between the two diseases as regards symptoms and pathological lesions, and he also showed that the two diseases occurred independently of one another, were due to separate causes, and that the one did not communicate or protect against the other.

As regards geographical distribution, Hirsch describes enteric fever as an ubiquitous disease, and Murchison was evidently of the same opinion. It is certainly common throughout Europe, including Norway and Sweden, the Farøe and Shetland Islands, and Iceland. It is common also in North America, in Australia, and in India. It occurs in China, in Japan, and in various parts of Africa, notably Cape Town. On the whole, however, it has seemed hitherto to be less common in tropical than in temperate climates. Some allowance, too, has to be made for the probability that certain forms of malarial fever have been frequently mistaken for enteric fever. It seems certain, for instance, that remittent fever in the tropics frequently simulates enteric fever in a remarkable degree, though *post-mortem* examination shows the particular lesions of the latter disease to be absent. Many cases of the so-called typho-malarial fever are no doubt of this kind, as was pointed out in a paper read before the Epidemiological Society of London in 1886 by Dr. J. Edward Squire.

Influence of Season.—The influence of season, both upon the mortality and prevalence of enteric fever in London, is shown in the following curves, which are taken from the Registrar-General's Annual Summary for the year 1890.

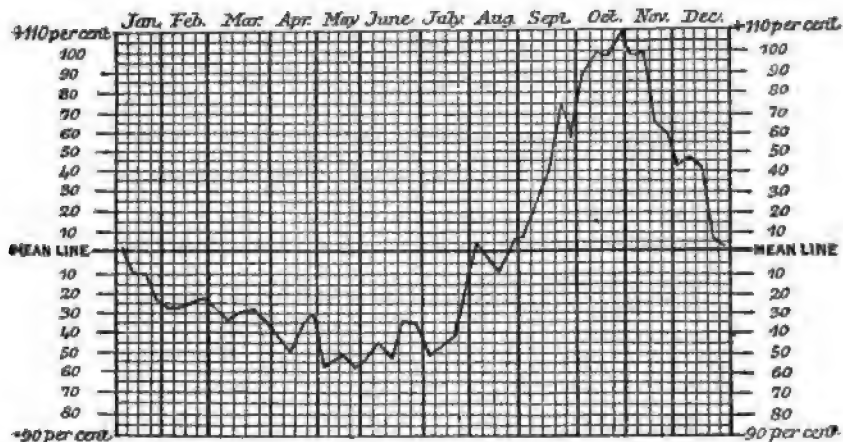
Enteric Fever.—London Deaths, twenty-two years, 1869-90.



For London, therefore, it appears that both the prevalence and mortality of this disease are greatest in the autumn, and least in the spring and early summer. A very similar relation to season obtains, according to Hirsch, for a number of European towns, including Paris and Berlin; and also for

Massachusetts and Boston. In New York, the maximum prevalence, Dr. Whitelegge says, occurs in September.

Mean Admissions of Enteric Fever Patients to the Metropolitan Asylum and London Fever Hospitals. (Sixteen years, 1875-90.)



Mortality.—The deaths from enteric fever have only been given separately by the Registrar-General since the year 1869. In the following table will be found the total enteric fever deaths recorded each year in England and Wales from that time to the present, and the corresponding death-rates per million of the population:—

TABLE XI.

England and Wales				England and Wales			
Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period	Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period
1869	8,660	390	374	1881	5,529	212	216
1870	8,781	388		1882	6,036	229	
1871	8,461	371		1883	6,078	228	
1872	8,741	377		1884	6,880	236	
1873	8,798	376		1885	4,765	175	
1874	8,861	374	277	1886	5,061	184	179
1875	8,913	371		1887	5,155	185	
1876	7,550	309		1888	4,848	172	
1877	6,879	279		1889	5,011	176	
1878	7,652	306		1890	5,146	179	
1879	5,860	281		1891	4,875	168	
1880	6,710	261					

From this table it will be seen that, notwithstanding the considerable increase in population which has taken place during the period in question, the absolute number of deaths recorded in the country as due to enteric fever has latterly diminished considerably; while the mortality, relatively to the existing population, is only about half of that which occurred in the earlier years. And that this reduction in enteric fever mortality has been largely brought about by sanitary improvements there can be no doubt. In the Ninth Report of the Medical Officer to the Privy Council will be found the results of Dr. (now Sir George) Buchanan's well-known inquiry into the effect

upon the death-rate of twenty-five towns in England and Wales of the execution of various sanitary works, notably works in connection with improved drainage and water-supplies. In twenty-one of these towns Sir George Buchanan found an average reduction of over 45 per cent. in the enteric fever mortality subsequently to the execution of the works in question. In three instances there had been a rise in the enteric mortality, but in these cases it was clearly associated with conspicuous defects in the sewerage systems.

Case Mortality.—Of the 5,988 cases recorded by Murchison as admitted to the London Fever Hospital during the twenty-three years, 1848–70, 1,084, or 17·3 per cent., died, ‘but, deducting those patients who were moribund on admission,’ the fatality at that hospital during the years in question was 15·8 per cent., or about 1 in 6. Allowing, however, for the many slight cases which occur outside hospital practice, it may no doubt be safely concluded that the actual fatality is less than that indicated above.

Influence of Race.—Race *per se* seems to exercise no influence over liability to attack by this disease. Negroes, although apparently less liable to suffer in their native countries than non-acclimatised persons living there, become, Hirsch says, ‘subject to the disease in America in no less a degree, *ceteris paribus*, than the white race.’

Influence of Sex.—As regards sex, it appears from the Registrar-General’s Fifty-first Annual Report that, judging from the enteric fever deaths at all ages, taken together, the rate of mortality is slightly higher for males than for females. From the third year until the end of the twentieth, however, the female mortality is decidedly higher than the male. With respect to liability to attack at all ages, taken together, there appears to be little difference between the two sexes, though males are apparently rather more susceptible than females. ‘Enteric fever,’ says Murchison, ‘attacks one sex as readily as the other.’ But according to the Registrar-General, whose conclusion is based upon a study of the admissions to the London Fever and Metropolitan Asylums Board’s Hospitals, it would seem that between five and twenty years of age more males are attacked than females. If this is usually the case, it follows that since more females than males die of enteric fever during the years in question, the fatality among females must be much greater from the fifth to the twentieth year than it is among males. The Registrar-General’s conclusion, however, as to the greater liability to attack of males than females between the fifth and twentieth year is, as he points out, based only upon 5,716 cases.

Influence of Age.—Both liability to attack and to death by enteric fever are considerably modified by age. As to the former, it has been stated by some observers that susceptibility is greatest in the first years of life, and progressively diminishes thenceforward. It has to be remembered, however, that many alleged cases of enteric fever among infants are probably, as the Registrar-General has pointed out with regard to infantile enteric fever deaths, ‘not due to that disease, but to some undetected cause, manifesting itself in feverish symptoms.’ Murchison states that the disease is ‘chiefly met with in youth and adolescence,’ and that was certainly so with regard to his hospital cases. Of the 5,911 cases he records, the largest number, 26·86 per cent., were between the ages of fifteen and twenty, and 66·42 per cent. were between the ages of ten and twenty-five. After the age of thirty the cases became fewer and fewer.

The mortality in this country, that is, the deaths per million living as distinguished from the fatality, is, according to the Registrar-General, at its minimum in the first year of life for both sexes. It rises to the end of the

fifth year, and then falls, 'not inconsiderably for males, but quite insignificantly for females,' till the fifteenth year, after which it rises again, until its maximum is attained in the age period 20-25. Thenceforward it falls, becoming 'comparatively low through the remaining age periods.'

As in the case of typhus, so with enteric fever, the danger to life in the event of attack is greatest at the higher ages, that is, from fifty-five upwards; but, as Murchison points out, there is this important difference between the age fatality of the two diseases—that in the case of enteric fever it 'increases with age to a much less extent than in typhus, and the small rate of mortality (fatality) observed in early life in typhus does not occur in enteric fever.'

Period of Incubation.—The latent period of enteric fever is liable to considerable variation. Murchison and several other authorities give it as most commonly about a fortnight, considering at the same time, however, that it may range from a few days to twenty-eight or thirty. In the Guildford outbreak, reported upon to the Privy Council by Sir George Buchanan in 1867, it was eleven days. In the Caterham outbreak, reported upon by Dr. Thorne to the Local Government Board in 1879, it was fourteen days. A good instance of a shorter period is recorded in the report of the late Mr. Netten Radcliffe and Mr. W. H. Power upon the milk outbreak in the West of London in 1878. The patient, a child whose own family were not consumers of the infected milk, 'spent an afternoon (July 19) with a family supplied from this dairy, and during the visit drank nearly two pints of milk; on July 24 she was attacked by enteric fever.'

Protection.—The extent to which enteric fever confers immunity against subsequent attack is still doubtful. Murchison remarks that 'well-authenticated instances of persons contracting enteric fever a second time are more common than is generally believed,' and he refers to his own experience, and also to 'unequivocal' examples of second attack reported by Michel, Bartlett, Paul, and Dr. W. Budd. On the other hand, he points out that on questioning patients suffering from enteric fever 'it is rarely ascertained that they have had a previous attack;' and he refers to instances recorded by different observers, of second outbreaks in particular localities in which those attacked on the earlier occasion escaped during the later outbreak.

On the whole, there would seem little doubt that enteric fever does confer immunity, but for what length of time is a matter well worthy of further inquiry.

Cause and Dissemination.—The bacteriological evidence with respect to the causation of this disease will be found in Dr. Klein's article (page 168). It is sufficient here to say that the ability of the enteric fever virus to multiply in water and milk is a strong indication that it consists of a living organism. Further epidemiological facts support the view that this organism is decidedly 'facultative,' being capable of thriving and multiplying, not only in water and milk, but also in the soil.

Enteric fever is essentially a communicable disease, though the mode by which it is usually communicated differs widely from that of some other communicable diseases. It is now well established that in epidemics of enteric fever the virus is mainly given off from the body of the sufferer in association with the bowel excreta, and that it finds its way into the body of the recipient most readily by way of the alimentary system, as, for instance, along with water or milk. In this sense enteric fever is classable with cholera rather than with typhus, small-pox, and other so-called infectious diseases. Whether or not enteric fever is at all infectious in the popular sense, has been, and still remains, a subject for discussion. In the early days of the

recognition of enteric fever as a separate disease many observers, especially in France, held it to be highly infectious. Looked at in the light of modern experience of epidemics, however, we can perceive that the succession of a number of cases in the same house or locality, which to the observers in question seemed conclusive evidence of infection, may well have been often instances of multiple exposure at different times to one and the same cause. And nowadays, when the importation of a case of enteric fever into a village previously free from the disease is followed by a number of other cases among the inhabitants of the particular village, we have first of all to assure ourselves that infection of the later cases from the earlier has not been indirect—that is to say, due to contamination of water or milk supply by the bowel excreta of the first case, rather than to direct infection. Similarly, the cases to which the early advocates of the doctrine of infectiousness attached considerable importance—of nurses who contracted the disease while nursing patients in the patients' own homes—we should seek to explain, in the first instance, by exposure of the nurses to the same causes to which their patients owed their attacks. In this course we should be encouraged by the circumstance that the records of both fever and general hospitals seem to indicate that if the patients are treated away from the locality in which they contracted the disease, neither the nurses, nor other patients in the same ward, but suffering from different ailments, contract the disease, except upon very rare occasions.

Thus Murchison points out that 'during twenty-three years (1848-1870) 5,988 cases of enteric fever were admitted into the London Fever Hospital, but only seventeen residents in the hospital contracted the disease, and most of them had no personal communication with patients sick of enteric fever.' One of these was a laundress, who may have contracted the disease by handling the soiled linen of enteric fever patients; and 'twelve of the seventeen cases occurred subsequently to 1864, when various extensions of the hospital buildings led to a serious derangement of the drainage.' Mr. Shirley F. Murphy has since brought down the experiences of this hospital to 1878, with similar results. But Murchison also gives the following striking evidence. 'Since 1861 it has been the practice to classify the patients in the Fever Hospital in this way:—The typhus, relapsing, and scarlatina patients have been kept in distinct wards, whereas the patients suffering from enteric fever have been treated in the same wards with the many patients sent to the hospital who have not been the subject of any form of contagious fever. The two classes of patients have remained together, both during the acute stage of their maladies and in convalescence, in most instances for several weeks. The same night-chairs have been used for both classes, and the employment of disinfectant has been exceptional. The result has been this:—During nine years 3,555 cases of enteric fever have been treated along with 5,144 patients not suffering from any specific fever; not one of the latter has contracted enteric fever.' Dr. Cayley, in editing the third edition of Murchison's great work, remarks that 'the subsequent experience [down to 1882] of the London Fever Hospital is in complete accordance with these statements of Dr. Murchison.' It becomes clear, therefore, that if enteric fever is infectious at all, its infectiousness is, in ordinary circumstances, an insignificant factor in its dissemination. In the few recorded instances in which nurses in hospitals have apparently contracted the disease from patients—that is, the few instances in which no other explanation was forthcoming—it is possible that in dealing with the patients' excreta or the patients' soiled linen the specific poison has become attached to the hands of the nurses (or even become lodged beneath their finger-nails), and so has been conveyed

accidentally, as it were, to their mouths, and thence to their alimentary apparatus.

At the same time, it would not be safe to say that the disease is never conveyed by infection; and some great authorities, even during recent times, have held the view that such does occasionally happen. Sir William Jenner, for instance, has recorded two cases of enteric fever which occurred in medical students who, before the days of the registering thermometer, were engaged in frequently taking the temperature of enteric fever patients.¹ In the circumstances of the case these students must have constantly had their heads in close proximity to the patients, and in the absence of other explanation it would seem likely that to this fact their attacks were due. Even so, however, it does not follow that they acquired infection from the breath or skin exhalations of such patients. There would probably be some slight soiling of the patient's body or bedclothes with the specific bowel excreta, which when dry might become detached and distributed for a short distance upon the displacement of the bedclothes.

But although it is doubtless true that in epidemics the majority of cases of enteric fever are due to specifically contaminated water or milk—matters which will be dealt with shortly—we must not overlook other possible modes of the dissemination of this disease.

It has to be remembered that wholesale dissemination of enteric fever by means of contaminated milk or water supply, in that it gives rise to a large number of cases, tends perhaps to distract our attention from other, but no less real, distributors of the disease. There is no doubt, for instance, that the air of sewers and drains which has become specifically contaminated may, if allowed to find its way into dwellings through defective house connections, cause from time to time enteric fever among the inhabitants of such dwellings.² And this fact has, indeed, in many minds, given rise to the exaggerated idea that sewers, as such, have large concern in the production of enteric fever. That notion has, however, been discounted by Dr. Buchanan's inquiry, already referred to, and by the continued fall in the enteric fever death-rate which has since gone on side by side with the modern extension of sewerage works. It is to defects of the sewerage system, therefore, and not to the system itself, that mischief of this kind is due when it does occur. Nevertheless, defective sewers and house drainage are still, unfortunately, common enough, and must always be taken into consideration in endeavouring to trace the origin of obscure cases occurring in the same locality at irregular intervals of time.

Again, it is now generally admitted that the enteric fever organism is capable of thriving in the soil. It is a common experience in rural districts, and apart from sewerage systems, to observe the disease hang about a particular locality. Once introduced into a village, it will often recur regularly as the autumn comes round, invading households deriving their water supply from different wells, households not possessing community of insanitary conditions of a sort to explain the fever. This seems consistent with a specific organism capable of abiding in the soil, multiplying therein at its own proper season, and so contaminating the soil over considerable areas. In this way many different wells may become specifically polluted, and recurring outbreaks in villages have often been found to cease upon the provision of an entirely fresh water supply from some distant locality.

But if the microbe is capable of living in the soil, it may, as suggested by

¹ Quoted from Fagge, *op. cit.*

² See Reports on outbreaks of enteric fever at Croydon, Worthing, and York, by Drs. Buchanan, Thorne, and Airy.

Lindwurm (*see* page 886, vol. i. of this work), also find suitable conditions of existence in the dirt of rooms, as between the boards or stones of the flooring. To this cause may be due the tenacity with which the disease is well known to cling to certain barracks.

With regard to alleged relationship of variations in the level of the sub-soil water to enteric fever prevalence, the reader is referred to Dr. Copeman's article in the first volume of this work.

There are few facts more certainly established in the etiology of disease than the dissemination of enteric fever by specifically contaminated drinking-water. Quite early in the history of recognised enteric fever outbreaks of that disease were traced to polluted water by Drs. William Budd, Alfred Carpenter, and others.¹ And during more recent years a large number of further outbreaks have been proved to have depended upon the same cause. Space does not permit of even a brief summary of these outbreaks, and reference will therefore be made only to certain selected reports which illustrate particular points in the propagation of enteric fever by water.

In the Tenth Report (1867) of the Medical Officer to the Privy Council will be found some interesting reports of the kind. Thus, Sir George Buchanan describes there a severe outbreak of enteric fever at Guildford which was limited to the portion of the town over which a particular section of the public water-supply was distributed. Inquiry showed that about ten days before the outbreak occurred the portion of the town in question had been exceptionally supplied with water from a new well in the chalk, close to which well a leaking sewer was found to exist. In 1878 further progress was made in our knowledge of the circumstances under which enteric fever may be spread by water. Early in that year Dr. Blaxall was instructed by the Local Government Board to inquire into an outbreak at Sherborne, in Dorsetshire.² He found that the disease had been more or less present in the neighbourhood for some seven years previously, but that a notable increase had occurred in the number of new cases during the months of January and February, 1878; and, further, that during 'the first week in March there was a sudden and very great increase, and during the remainder of the month a gradual diminution.' Various possible causes for this increase having been inquired into and put aside, the process of exclusion led to the public water-supply as the probable source of the mischief. In connection with this water-supply it was ascertained that certain manipulations of the service had closely coincided with the variations in enteric fever prevalence. Thus, during the months of December 1872 and January 1873, the water 'was frequently shut off from the town at a point near the reservoir, and the same thing was done every night in February.' During the month of March this system of shutting off the water at night was discontinued. Thus the notable increase in the cases of fever in January and February coincided (allowing for the period of incubation) with the frequent intermissions in the water-service; the sudden and great increase in the cases in early March followed closely on the regular nightly intermissions of the water-supply; and lastly, the diminution in the number of attacks of fever was coincident with the discontinuance of the system of intermitting the water. Now, in Sherborne at that time this water was in some cases laid on direct to the closet pans by pipes supplied with taps. Some of these taps were broken. The effect, therefore, of turning off the water at the mains

¹ Parkes mentions the 'Schleimfieber' of Göttingen, in 1760, as having been attributed in part to impure water (Parkes's *Practical Hygiene*, p. 66).

² *Reports of the Medical Officer to the Privy Council and Local Government Board*, N.S., No. II. 1874.

would naturally be to establish a direct air communication between the water-mains (through the closet supply-pipes) and certain of the closet pans—in other words, as Dr. Blaxall remarks, 'the system of pipes for the water-supply became the means of ventilating the closet pans; if the trap happened to be broken or out of order, it became a means of ventilating the sewers; and if a pan happened to be full of excrement, that excrement would be sucked into the water-pipe.'

During the following year Sir George Buchanan¹ traced an outbreak of enteric fever at Caius College, Cambridge, to a broadly similar cause. The

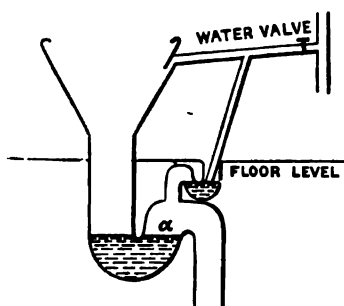


FIG. 1.

outbreak in question was mainly limited to a particular section of the college known as Tree Court. Tree Court derived its water-supply from the general 5-inch main of the Cambridge Water Company, which supplied the College, but by a special pipe from this main 'for Tree Court, and for no part but Tree Court.' Now it turned out that, unlike those in the rest of the College, the water-closets at Tree Court were supplied with water direct from the constant water-service, i.e. without the intervention of any disconnecting cisterns. And in addition to this, a small 'weeping-pipe' was given off from the closet supply-pipe to feed a miniature trapping-bend in the safe waste, which was taken direct into the closet trap. This arrangement is shown in fig. 1, taken from Sir George Buchanan's report. It is evident, then, that in the event of the water being intermitted, and the valve on the closet supply-pipe left open, there were here ample opportunities for admission to the water-pipes of foul air from the closet, or even liquid filth from the closet trap. It was further ascertained that about a fortnight before the enteric fever occurred in Tree Court the water-supply of this particular part of the College had, as a matter of fact, been temporarily cut off. It was also found on examination that the end of the weeping-pipe 'was crusted with a brownish matter,' and that internally it, and the supply-pipe between where the weeping-pipe came off and the valve, had also some little deposit. These deposits were submitted for analysis to Dr. Dupré, who came to the conclusion that 'water impregnated with faecal matter must have entered the pipes.'

But a further link in the chain of evidence as to the origin of *specific* contamination of the water-supply of Tree Court was forthcoming. It turned out that between 'the unventilated sewer in Trinity Street' and the point marked *a* in the diagram there was 'not a single opening nor a single trap; nothing either to get rid of, or to attempt to keep back, the foul air' [from the sewer]. It is clear, therefore, that the water in both the large and small trapping-bends must more or less constantly have been charged with sewer air; and in the case of the smaller one 'a very small excess of atmospheric pressure in the down soil-pipe over the interior of the house would cause sewer air to bubble through the little bend; or some aspirating or syphon action . . . would empty this bend of its water.' Lastly, it was shown that shortly prior to the time of the outbreak in Caius College cases of enteric fever had occurred in houses draining into the Trinity Street sewer above the point at which it received the drainage of Tree Court, so that at the

¹ *Report of the Medical Officer to the Privy Council and Local Government Board, N.S., No. II., 1874.*

particular time in question the sewer air in the Tree Court soil-pipes must doubtless have been charged with the specific poison of enteric fever.

Clearly, therefore, there can be little doubt that when the water-supply of Tree Court was temporarily cut off (some one or more of the valves on the closet supply-pipes having probably been left open), specifically-tainted sewer air, and even similarly tainted water and filth from the trapping-bends had been sucked up into the Tree Court main pipe, and subsequently distributed with the water throughout the Court—'for fever attacked every staircase of the Court.'

The reports above quoted indicate very clearly some of the dangers of intermitting a water-supply. Before proceeding to consider certain other reports which in chronological order should follow, reference must be made to the admirable report by the late Mr. John Spear on an outbreak of enteric fever in the Mountain Ash U. S. D.¹ This outbreak was mainly limited to the area of distribution of a certain section of the public 'constant' water-supply. Careful inquiry enabled Mr. Spear to localise the probable spot at which contamination of the supply had taken place, and examination of the pipes at that spot showed that 'the main was carried, without any special precaution, immediately above, alongside, and even through old rubble drains, and when, in the course of the trenching, pipe-drains were cut through, no trouble appears to have been taken to replace them.' The main, which was leaky, was thus, 'at different points, from time to time bathed in refuse matters, and habitually, at certain points, in sewage-contaminated air.' Intermissions of the water-supply had taken place, and it is evident that, under the circumstances indicated, the pipes would 'take up foul air and liquid from these culverts and from the soil around.' But it is important to remember, as Mr. Spear points out, that such intermissions are 'not by any means essential to the introduction of foreign matters into water-pipes. Under various physical conditions, very powerful insuction of external matters into a full-flowing water-pipe can take place.'

Certain reports may now be quoted which illustrate the important fact that a degree of specific contamination of a water-supply so slight as to escape detection by the ordinary methods of chemical analysis may give rise to widespread outbreaks of enteric fever. The most important of these is the report of Dr. Thorne Thorne on an epidemic at Caterham. This report and its teachings have since been briefly summarised by Dr. Thorne himself as follows:—'In that case 852 cases of enteric fever took place at Caterham, Redhill, and certain intervening places, some 200 of the earlier cases being traced directly to the use, during a particular fortnight, of water derived from a deep well in which a man suffering from that disease had been employed, under circumstances which left no possible doubt that his excreta had got access to the well water. In this case it was estimated that, after all possibility of further pollution had come to an end, no less than 1,861,000 gallons of water had been pumped from the well during the fortnight in question; and Dr. Buchanan, in dealing, in his Annual Report for 1881, with the question of the amount of specific pollution which might suffice to render a potable water dangerous, showed that in this case the water could have contained no such amount as one grain of excremental matter per gallon, and he brought forward considerations tending to prove that an amount of specifically polluting matter so infinitesimal in quantity as to be altogether beyond detection by chemical analysis was fully potent for mischief.' In fact, as Dr. Buchanan tersely remarks, 'the chemist can, in brief, tell us of impurity and hazard, but not of purity and safety.' Other inquiries which

¹ *Report of the Medical Officer to the Local Government Board for the Year 1887.*

have resulted in supporting this conclusion have since been made. Among them may be mentioned a most interesting inquiry into an enteric fever epidemic at Houghton-le-Spring, which was reported upon by Dr. Page.¹ In this case the causal relation of the public water-supply to the outbreak was clearly shown, and the strongest evidence was forthcoming that the water in question was being polluted by filth which had travelled three-quarters of a mile through a fissure in the magnesian limestone. A sample of this water was at the time pronounced by an analyst to be 'a good water for drinking.' Comparatively recently, an outbreak of enteric fever in the Tees Valley has been clearly shown by Dr. Barry to have been distributed along with public water-supply pronounced by an expert analyst to be free from contamination. Clearly, then, although the analyst's evidence of pollution must always be gratefully received, his assurances of the purity of a suspected water must not be allowed to override other considerations tending to incriminate the water in question.

It has also to be remembered that enteric fever may be due to the use of contaminated water in the preparation of various 'drinks,' ices, &c. Thus, in 1882 Mr. G. H. Fosbroke traced an extensive outbreak of enteric fever to the consumption of lemonade, ices, and spirits which had been prepared or diluted with water from a sewage-polluted well.²

The probability of enteric fever being disseminated by the agency of milk was first pointed out by Dr. Michael Taylor in 1858.³ In 1870 Dr. Ballard demonstrated the association of an extensive outbreak of enteric fever at Islington with the consumption of milk from a particular dairy, at which it was ascertained that a contaminated water was used for cleansing the dairy utensils. Three years later a widespread epidemic in Marylebone and other parts of the West-end of London was found by Mr. Netten Radcliffe and Mr. Power to be similarly distributed with a certain milk service; and here there was not only strong presumption that a contaminated water was used for dairy purposes, but that such water was actually contaminated by the excreta from a person suffering from enteric fever.⁴

Other outbreaks have since been traced to infected milk-supplies. In some of such instances the source of infection has been found in the use of contaminated water for the purpose of diluting the milk or cleansing the utensils, or in the presence of a person suffering from the disease at the particular dairy. But in an outbreak of enteric fever at Eagley and Bolton, traced, in 1876, beyond doubt to a particular milk service by Mr. W. H. Power, with the assistance of Mr. Robinson and Mr. Sergeant, the medical officers of health to the districts concerned, the question was raised as to the possibility of the milk obtaining infective quality from an ailment of the cow, as in the case of scarlet fever and diphtheria.

Since that date at least one outbreak has occurred in this country where, after minute inquiry, contamination of the implicated milk after it had left the cow appeared highly unlikely, and Dr. J. F. Allen, the Medical Officer of Health to Pietermaritzburg, has expressed his conviction, based upon his experience in Africa, that the disease may be caused in man by the milk of cows suffering from a similar malady.

¹ *Annual Report of the Medical Officer to the Local Government Board for the Year 1889.*

² See also a recent Report to the London County Council, by Dr. George Turner, on 'An Outbreak of Enteric Fever in the South-East of London.'

³ *Edinburgh Medical Journal*, May 1858.

⁴ *Report of the Medical Officer to the Privy Council and Local Government Board*, 1874.

Relation to Malaria.—The question whether or not enteric fever and malaria stand in any special relationship to one another has been extensively discussed. Boudin and others have maintained that a mutual antagonism exists between them, and that where malaria is common, there enteric fever is rare, or absent. This would seem to have been so in some localities; but it does not by any means universally hold good, and the more probable explanation of the phenomena as observed would seem to be that the rise of civilisation, by leading to the cultivation of the land and crude attempts at drainage, has tended to banish malaria, giving rise at the same time to the aggregation together of the people with—under the circumstances of the then defective sanitary arrangements—all its attendant pollution of air, water, and soil. To this, no doubt, was the increase of enteric fever due, and not to the disappearance of malaria. The question, moreover, is complicated by difficulty of diagnosis, and it is impossible to say to what extent the alleged disappearance of the one disease and appearance of the other may not in certain localities have been overstated. In some cases, during the earlier period of observation, enteric fever may, though present, have been mistaken for malaria; while, on the other hand, some of the later cases of supposed enteric fever may have been but an altered type of malaria.

De novo Origin of Enteric Fever.—In the foregoing pages it has been assumed that every case of enteric fever is derived from an antecedent case, human or animal, of like kind. The question as to the possibility of a *de novo* origin of enteric fever has, however, been keenly contested, and in a certain sense it must be regarded as still an open one.

The impartial inquirer can hardly fail to be struck by the weight of authority which has ranged itself on the side of the view that this disease may, and from time to time does, arise independently of an antecedent case. On the other hand, however, it has been maintained that the dependence of enteric fever on a specific poison implies that every case of that disease must be connected with an antecedent case of the same disease; that this must be as true of enteric fever as of small-pox; and that on this circumstance, in fact, depends its *specificity*. But this argument rests upon the assumption that there can be no degrees in *specificity*, no progressive development of specific quality, and this seems to lie at the root of the whole matter. It would be impossible, for instance, at present to allege that enteric fever never grows, as it were, out of simpler forms of diarrhoea.

The question must, indeed, be patiently worked out by the light of observation, neither the one theory nor the other being allowed to divert the attention of the inquirer from a faithful record of the actual facts. Every effort must, in all instances, be made to trace a causal connection between isolated cases of enteric fever and previous cases of that disease; while, on the other hand, failure after sincere endeavour to trace such a connection must be carefully noted, along with any observations suggesting other possible origin of the malady.

CHOLERA

Synon. : *Asiatic Cholera* ; *Indian Cholera* ; *Spasmodic Cholera* ; *Malignant Cholera*. Fr. *Choléra Asiatique* ; Ger. *Asiatische Cholera* ; It. *Colera Asiatico*.

The connected history of epidemic cholera dates back only to the early years of the present century. Nevertheless it would appear, as Dr. J. M. Cunningham remarks, that cholera is 'no new enemy of mankind,' but has been known in India 'from the earliest times of which there is any record.'

Mr. Macnamara quotes a passage which is highly suggestive of cholera from the Sanscrit writer, Susruta, who lived before the Christian era, and he also considers that 'Hippocrates, Galen, and Whang-shoo-ho are witnesses to the existence of cholera in their day, both in Europe and China ; and their evidence is supported by a series of Grecian, Roman, and Arabian authors, bearing record to the fact of the presence of the disease in the various countries in which they lived.'

The first notice of cholera in India by a European writer appears to be that of Gaspar Correa, a Portuguese, and refers to an extensive and fatal outbreak in the army of the sovereign of Calicut in the year 1508. Later in the sixteenth century outbreaks are mentioned by Portuguese and Dutch writers as having occurred at Goa. In the seventeenth century there is evidence of the presence of cholera at Batavia, Goa, and 'in London during the autumn of 1676, when Sydenham states that the cholera morbus was raging.'² Caution must, however, be observed in concluding that the early accounts of cholera on extra-Indian soil refer to true malignant Asiatic cholera.

Towards the close of the eighteenth century there were records, by Dr. Paisley and others, of epidemics of cholera in different parts of India. A limited number of cases also occurred in 1808-9, and scattered outbreaks from 1811 to 1814.

But the more important history of cholera dates from the year 1817. At that time commenced what proved to be the first recorded pandemic diffusion of the disease. Beginning in 1817 with a widespread prevalence in Bengal, cholera during the two following years extended throughout India and commenced to spread abroad. Travelling first (1819-20) in a southerly and easterly direction to Ceylon, Mauritius, Burma, China, Siam, Penang, Singapore, Sumatra, Borneo, and Java, it somewhat later spread in a north-westerly direction, through Arabia and Persia, and up the Caspian to Astrakhan (1823). It died out, however, as the cold weather came on.

In 1826 began a second and far more extensive pandemic. Starting from Bengal, the disease again spread (1829-30) to Russia by two routes, the one *via* Cabul, Balkh, Bokhara, and Khiva (caravan routes) to Orenburg ; the other by Teheran and the shores of the Caspian Sea to Astrakhan, and thence up the Volga to Saratov and Kazan. In the meantime cholera was also spreading through Arabia, whence it advanced to Suez, Cairo, and up and down the Nile to Thebes and Alexandria respectively. From Russia it gradually extended over Europe and the greater part of America. It reached England in July 1831, having apparently been brought to the Medway by

¹ *A Treatise on Asiatic Cholera*, by C. Macnamara, p. 8.

² Macnamara, *op cit.*, p. 6.

ships from Riga. In October of the same year it broke out at Sunderland, as a result, it was believed, of importation by ships from Hamburg. It appeared at Newcastle and Gateshead in November, and at Haddington, in Scotland, in December.

In the following year, 1832, the disease was more extensively prevalent in Great Britain, many large towns in England, Ireland, and Scotland being invaded. To Canada the disease was apparently carried by Irish emigrants in June 1832, and rapidly spread throughout that country and also the United States. This pandemic did not entirely cease till the winter of 1837-38. It then died out 'at every point in the immense territory it had overrun in the period from 1826 to 1837. For the next ten years the soil of Europe, Africa, and America was completely free from it' (Hirsch).

The third great pandemic dates from the year 1846. Cholera, which assumed a wide epidemicity in India during the years 1840-41, extending also to Further India, the Philippines and China, appeared at Cabul in 1842. Thence, contrary to its custom, it extended from west to east through Peshawur, Lahore, and the North-West Provinces. In 1844 it reappeared at Cabul, spreading to Herat, Samarcand, and Bokhara in the same year. In 1846 it extended westwards to Asterabad and Teheran, and southwards to the shores of the Persian Gulf, whence it took a north-westerly direction along the valley of the Tigris, *via* Bagdad, to Diarbekir. In the same year the whole of the Arabian peninsula also became invaded, either from the Persian Gulf, or perhaps independently from Bombay *via* Aden. By 1847 cholera had spread from Persia to the shores of the Caspian Sea, whence it extended in a westerly direction along the coast of the Black Sea to Constantinople, and in a northerly direction to Astrakhan. At Orenburg, in the basin of the Ural, 'it joined hands with another invasion coming from Bokhara' (Hirsch). It now spread rapidly through European Russia, travelling up the Volga to Moscow and the shores of the Baltic. It also advanced from the Sea of Azov northwards up the river Don, and in a westerly direction to Odessa. During the winter the disease abated, but it reappeared over the whole of Russia and Poland in the spring of 1848. By the end of this year it had died out in Russia, but in the meantime it had invaded Germany, the Netherlands, Belgium, Great Britain, and America. As regards Great Britain, the first case heard of occurred in the person of a seaman named Harnold, who arrived in London on September 18 or 19 in a steamer from Hamburg, where cholera was prevalent prior to the departure of the vessel. This man died at Horselydown on September 22. The next case heard of was a man who slept in the same room with Harnold. During the first week in October some twenty-six cases, mostly fatal, occurred in London. Of these, eighteen were either on or close to the river Thames. About the same time (October 4, 1848) cholera appeared in Edinburgh. There the first cases were among some pilots who had been engaged in bringing in to Leith a ship from Cronstadt, where the disease was known to have been present in the summer. Also, in October cholera appeared in Hull and Sunderland, and again its appearance had been immediately preceded by the arrival of ships from Hamburg. The first known case in Ireland (December 2) was that of a man who had recently arrived in Belfast from Edinburgh, which city, as has been seen, was already invaded. To America the disease clearly seems to have been carried by German emigrants, who arrived almost simultaneously in two cholera-stricken ships, the 'New York' and the 'Swanton,' at Staten Island and New Orleans respectively.

During the winter of 1848-49 cholera apparently abated somewhat, but in the spring of 1849 it broke out again with increased vigour throughout the

greater part of Europe and America. In England the disease raged from April to December, 119 places being invaded. The mortality was greatest at Hull, where it reached the rate of 241 per 10,000 of the population. During this year France also was severely attacked. The disease appeared in Paris in March, and by the end of June 15,000 deaths had occurred. In 1850 Europe generally suffered little from cholera. The disease was, however, epidemic in Sweden, and also on the African seaboard of the Mediterranean, Egypt and Malta being heavily invaded. It was also widely diffused over America. During the next two years there was a marked remission in cholera prevalence in Europe, though scattered epidemics occurred from time to time, especially among the German Baltic ports. England remained entirely free during this period. But the following year (1853) saw an extensive resuscitation of the disease in Europe, and it became again widely diffused over a great part of the Continent. In the summer of this year it appeared in London, apparently as a result of importation from Germany, and later in the year it broke out at Newcastle, Shields, and Liverpool, also, it would seem, through importation from German ports. Later in the year it was epidemic in Manchester, Edinburgh, Glasgow, and Dundee. In 1854 it was very severely epidemic in Great Britain, and extensively prevalent over Europe generally, and America—in the latter case in consequence partly of fresh importations from Europe. For the following six years (1855–60) cholera continued from time to time more or less prevalent in Europe. Localised outbreaks occurred in England in 1855 and 1857. In 1859 cholera was imported into London, Hull, and Shields from Hamburg, but failed to spread. By the end of the year 1860, however, cholera had apparently died out in Europe. ‘Regarded as a whole,’ says Hirsch, ‘this pandemic shows numerous fluctuations of intensity, the maxima falling in the years 1849–50 and 1853–55. In none of the intervening years was the disease altogether extinguished on extra-Indian soil, and there is no reason for attributing the fresh outbreak of 1853 in Europe, Africa, and America, to a new importation of the morbid poison from its native habitat. All the facts tell rather in favour of a continuous reproduction of the poison in extra-Indian countries, and that power of reproduction was exhausted only after the lapse of more than ten years.’

The fourth great pandemic began in the year 1863. As regards its invasion of Europe, this pandemic differed conspicuously from those already described, and ‘is remarkable as marking a new epoch in the history of cholera, viz. its introduction by way of the Red Sea, wholly or in great part.’¹

Starting from the basin of the Ganges, the disease, during the years 1863–65, became widely epidemic in Bengal, the North-West Provinces, Bombay, the southern parts of the Deccan, and Ceylon. It was also prevalent in China and Japan. In 1865 it was carried by ships to South Arabia (Hadramaut). It next broke out among the pilgrims assembled at Mecca (May 1865), by whom it was carried to many places, among them Suez (June). From Egypt the disease, during the next few weeks, spread to Malta, Marseilles, Ancona, Valencia, Constantinople, and other places. From these centres it extended through France, Italy, Spain, Turkey, Roumania, and Southern Russia. England was invaded in August of this year (1865), though only on a small scale, a few cases occurring at Southampton, and a localised outbreak (apparently connected with the Southampton cases) at Theydon Bois, in Essex. To Southampton the disease seems to have been brought from Alexandria. In the Western Hemisphere,

¹ ‘Retrospect of the Successive Epidemics of Cholera in Europe and America’ (E. F. Willoughby, M.D., D.P.H., *Epidem. Trans.*, N.S., vol. x. 1890–91).

the disease appeared in October 1865 at Guadeloupe shortly after the arrival of two ships from Marseilles and Bordeaux. Limited outbreaks also occurred in 1865 in Austria and Germany, and a more severe one in Belgium (Luxembourg).

In the following year (1866) the disease became extensively prevalent in Belgium, the Netherlands, Sweden, England, France, Italy, Turkey, and Russia. Scattered cases also appeared in Denmark, Norway, and Finland. It now, too, appeared in and became diffused over parts of North and South America. In England the disease was, during this year, imported at various places, notably Bristol and Liverpool, from Rotterdam; Southampton, by the P. and O. steamer 'Poonah,' coming from Alexandria *via* Malta and Gibraltar; Goole from Antwerp; and Shields from Hamburg. To North America cholera seems to have been in the first instance carried by Irish and German emigrants from Liverpool.

Although cholera in Europe and America now began to decline, some years elapsed before it disappeared. Thus, it was more or less present in Russia each year from 1867 until 1874, and in Germany in each of the same years, with the exception of 1869-70. In Switzerland it was present in 1867; in France in the same year, and also in 1878; in Austria in 1867, 1872, and 1878; Sweden in 1871 and 1878; South America in 1867-8, and North America (re-imported) in 1878.

The years of lowest epidemicity during this period were 1869-70, after which, up to 1878, the disease again became more prevalent. It is maintained by some that this renewed prevalence was a result of a fresh importation of the disease from India, and there would appear grounds for thinking that such was in part, at all events, the case. It seems certain that cholera became widely epidemic in North-West India after the great Hurdwar bathing fair in 1867, and that thence it spread through Persia into Russia in 1869 and 1870; so that the subsiding European epidemic would seem at least to have been reinforced by this fresh importation. Hirsch, however, takes the opposite view, and regards the renewed prevalence in Europe at this period as a resuscitation of the temporarily abated epidemic. By the end of 1874 cholera had disappeared from Europe, and did not return for a period of almost ten years.

In June 1884, however, cholera broke out at Toulon, possibly, it would seem, as a result of importation by French troops from Cochin China or Tonkin.¹ At the end of the month the disease had appeared in Marseilles,² and later in the year it spread over a large part of France. More than 880 deaths occurred in Toulon, and nearly 2,000 at Marseilles. As early as the end of June, cholera had appeared in Paris, but the serious prevalence of the disease in that capital did not take place until November, during which month there occurred there 988 cholera deaths. Altogether, during the year 1884 some 5,000 deaths from cholera occurred in France. In the meantime the disease had also appeared both in Italy and Spain, notwithstanding the extensive quarantine precautions which were taken to prevent its importation to those countries. In Italy, it seems first to have broken out at Spezia. Before the end of the year 858 Italian communes had been more or less invaded, and, according to the Official Report of the Minister of the Interior,

¹ For the detailed history of cholera in Europe during this and other recent years, see the Reports and Papers on Cholera submitted by the Medical Officer to the Local Government Board as a supplement to the Board's *Fifteenth Annual Report*; also the various Annual Reports of the Medical Officer to the Local Government Board.

² There appears some evidence that cholera was present at Marseilles in the year 1888. See footnote to p. 9 of the *Annual Report* for that year of the Medical Officer to the Local Government Board.

as many as 27,080 attacks and 14,299 deaths had resulted. The disease was most severe at Naples, in which city there were 7,086 deaths. It is notable that Rome, with its excellent water-supply, almost entirely escaped, only thirteen cases and six deaths being recorded for the whole province. In Spain the epidemic was not, during 1884, of a very extensive character. In all there were 274 cholera deaths in that country, 215 of which occurred in the province of Alicante.

During this year (1884) cases, apparently of cholera, were three times brought in ships to England, but no spread of the disease occurred. Germany, also, practically escaped.

By the late autumn or early winter the disease had died down in France, Italy, and Spain. But it reappeared in each of those countries in 1885. This time Spain was heavily attacked. The earliest recorded cases occurred in the province of Valencia during the month of February, but the disease did not become widely diffused until June. It then increased through the months of July and August, beginning to decline in September, but it had not died out at the end of the year. During the period February 5 to December 31, 1885, forty-six provinces had been invaded, the total recorded cases numbering 388,685, and the deaths, 119,620. In Italy the disease became epidemic in a number of provinces in August, and before the end of the year 3,459 deaths were recorded, 2,959 of which occurred at Palermo. As regards France, cholera was, during 1885, most fatal in Marseilles and Toulon, at which places 1,089 and 814 fatal cases occurred respectively, the main mortality occurring in the month of August. Later in the year, however, cholera appeared in the province of Finistère, and some hundreds of deaths occurred there, notably at Brest.

Several suspicious cases of diarrhoea were brought by different ships to England during 1885, but this country again remained free from epidemic cholera.

By the spring of 1886 cholera had practically disappeared from France and Spain. In Italy, however, it was again prevalent, especially on the Adriatic seaboard, and more than 6,000 cholera deaths were recorded. Austria-Hungary now also became somewhat severely invaded, especially at its Adriatic ports, the deaths numbering over 2,000. The disease still lingered in the kingdom of Italy in 1887, mainly in the island of Sicily; but by the end of that year the European epidemic which had begun in 1884 came to an end.

Europe now remained free from cholera until 1890, when the disease suddenly reappeared at Valencia. At this time cholera was steadily advancing through Persia and along the Euphrates Valley, but there seemed no sufficient grounds for connecting the Valencia outbreak with that circumstance, and it was suggested, on the other hand, that the reappearance of the disease in Spain was due to excavations in ground infected in the previous epidemic by cholera evacuations.

In the meantime (1890) cholera continued to advance in Persia, Turkey in Asia, Syria, and Arabia, reaching Tabrez, Diarbekir, Aleppo, and Mecca.

About June 1891 it again broke out at Aleppo and the surrounding villages, appearing later at Damascus, Antioch, and other places. In Mecca also it reappeared, causing upwards of 11,000 deaths. From Mecca it was carried by pilgrims to Medina and Jeddah.

Towards the end of 1891 cholera, which had apparently extended from the neighbourhood of Peshawur, broke out in Cabul. After a temporary abatement during the months of January and February 1892, it reappeared there in March. In the same month it was also severely prevalent at Herat,

and two months later at Meshed. Later still, by some two months or more, it reached Teheran. But in the meantime (the beginning of June) it had broken out in Askabad, whence it quickly spread along the route of the Trans-Caspian Railway and across the Caspian Sea to Baku. From Baku it spread westward to Tiflis, being carried northward, at the same time, by steamboat traffic up the Caspian Sea to Astrakhan, at the mouth of the Volga, where it appeared on June 30. By July 6 it had broken out at Saratov, 500 miles up the Volga, and a little later at Kostroma. By the first week in August a number of the Central Russian provinces were invaded. By August 16 the disease was established in St. Petersburg, and by the 28rd the invasion of Hamburg was officially announced. To Hamburg the disease was probably brought by Russian emigrants *en route* for England and America. Later on cholera appeared at Antwerp, Amsterdam, Rotterdam, and in Galicia, cases also occurring in Brussels, Berlin, and other places.

But in addition to the cholera above referred to, which undoubtedly came direct from the East, there was also throughout the summer (1892) a sustained and extensive prevalence of cholera in France. The disease appeared in the suburbs of Paris at the very commencement of April, the city itself becoming invaded a week or two later. Subsequently cases appeared at Havre, Rouen, and other places. Although this French cholera did not exhibit a tendency to very rapid spread, its fatality was considerable, and there are strong grounds for regarding it as true Asiatic cholera. Its early appearance, however, and the absence of evidence of its direct importation from the East, make it possible, as Mr. Macnamara suggests, that it was a revival of the European cholera of 1864-87.

The mortality from cholera in Europe during 1892 was very great. In European Russia to November 30, 132,700 deaths occurred. In Hamburg, according to official returns, the cases in the first nine weeks of the outbreak numbered 17,989, and the deaths, 8,261. As regards France, the deaths from April to September 14 in Paris and the suburbs, in the Department of the Seine, amounted to over 1,400.

The experience of England during the epidemic was decidedly encouraging. Two days after Hamburg had been declared infected, three cases of cholera from that city arrived at the port of London in the s.s. 'Gemma,' and by the middle of October some twenty-nine undoubted cases had been brought to this country. But in no instance, so far as is known, did the disease extend beyond the imported cases.

The points to note with regard to the cholera pandemic of 1892 are: (1) That the disease again travelled along main lines of communication, and, in many instances, in conspicuous association with the movements of persons along those lines. In this connection it is noteworthy that within a month 'of the recognition of cholera at a town on the Trans-Caspian Railway it (cholera) had penetrated to the heart of Russia in Europe, the transit from Central Asia having taken as many days as, before the creation of railways and steamboat lines, it took months.'¹ (2) That cholera has again exhibited its tendency to flourish where unwholesome conditions abound, and that as regards Hamburg, and Paris and its suburbs, at least, the victims have for the most part been persons consuming contaminated water. (3) That attempts to stay the march of the disease by measures of quarantine have again, in some instances, signally failed, while, on the other hand, the more practicable and reasonable system of medical inspection adopted in this country, although it does not claim to be an absolute protection against the

¹ 'The Route of Asiatic Cholera in 1892,' by Dawson Williams, M.D. (*British Medical Journal*, Sept. 17, 1892).

importation of the disease, has nevertheless been attended with success in that respect.

Before leaving the subject of the history of cholera, reference must be made to one or two circumstances brought out by that history. In the first place, the favourite, and possibly original, home of the disease would seem to be India, and especially Lower Bengal—a region from which cholera is practically never absent, and from which it has again and again extended in epidemic or pandemic diffusion over wide areas of the earth's surface. In such epidemic or pandemic excursions cholera has tended especially to advance along main lines of human communication, such as caravan routes, navigable rivers, and the lines of sea traffic. Its appearance in previously uninhabited localities has often been found to have been closely preceded by the arrival of persons from infected districts; and lastly, extensive diffusions of the disease have at different times followed upon the congregation and subsequent dispersion of pilgrims, as at the great Hurdwar Fair, in India, and at Meschid and Mecca. It will have been observed from the historical sketch above given that, in its marches from India to the West, cholera has followed certain well-defined routes, which have been described as the Central Asiatic, the North Persian, the Persian Gulf, and the Red Sea routes. The Central Asiatic route advances through Cabul, Balkh, Bokhara, and Khiva, to Orenburg in European Russia; the North Persian, *via* Cabul, Herat, Meschid, Astrabad, and Teheran, and thence up the Caspian Sea to Astrakhan, and through Tabrez to Erzeroum, Trebizond, and along the Black Sea; the Persian Gulf route passes up the shores of the Gulf and north-westwards, along the river Tigris to Baghdad and Diarbekir, and the Euphrates to Aleppo. The Red Sea route, first followed in 1865, passes from India by sea to South Arabia, and thence along the shores of the Red Sea to Suez, Cairo, and the Mediterranean.

As regards geographical distribution little need be said. At one or another time cholera has extended, as we have already seen, widely over the earth's surface. In its marches, however, it has, so far, spared certain localities. Thus, it has never yet invaded Australia, the East Coast of Africa south of Delagoa Bay, the islands of the Pacific Ocean, the islands of St. Helena and Ascension, Iceland, the Farøe Islands, the Hebrides, the Orkney Isles, Lapland, and some other places (Hirsch). Even in invaded continents certain places have enjoyed a marked degree of immunity. Exemptions of this sort are probably due, either to the relatively little communication between the places in question and the continent of India, or to the enjoyment by such places of special sanitary advantages, more particularly pure and wholesome water-supplies.

Mortality and Fatality.—The mortality from cholera is often enormous. As regards this country, the deaths resulting from the 1831–32 epidemic were estimated at over 80,000. They were not, however, accurately known, as registration had not then been introduced. In the next invasion of Great Britain (1848–49) the mortality was very great. Thus, in the year 1849, when the epidemic was at its height, the cholera deaths registered in England and Wales amounted to 53,293, which was equivalent to a rate of 8,088 per million of the population. There was also during this year a notable increase in the 'diarrhoea' mortality. It is, however, satisfactory to observe that upon each of the two later invasions of this country (1853–54 and 1865–66) there was a marked decline in the mortality. In the year 1854 the cholera deaths were 20,097, giving a rate of 1,080 per million against 3,088 of the year 1849; and in the year 1866 the deaths still further fell to 14,878, or 672 per million. Since the latter year England has remained free

from epidemic cholera. In each English invasion the prevalence and mortality of the disease have been far greater in the second than the first year of the epidemic.

The fatality of cholera is also very high, usually ranging from 80 to 50 per cent. of the attacks, and it may be even higher. In 1892 it seems to have been about 45 per cent. both for the whole of Russia and for Hamburg. The fatality of cholera is said to be greater at the beginning than during the later stages of an outbreak.

Influence of Climate and Season.—That Asiatic cholera is largely influenced by climate there seems no room for doubt, for although it is true that the disease has shown its ability to diffuse itself widely under various conditions as to climate, it has not shown an equal ability to permanently establish itself under such diverse climatic conditions. On the contrary, in temperate climates it tends to die out in the course of a few seasons. Moreover, that a certain degree of heat favours the activity of the poison is sufficiently evidenced by the fact that in Europe the disease has generally attained its greatest prevalence in the months from June to August, dying down during the winter, often only to reappear in the following summer. The fact of there having been certain exceptions to this order of events does not alter the bearing of the usual experience. 'As a general rule,' says Macnamara, 'it may be stated that cholera will not extend during the cold of a European winter, or even of our Punjab cold season.'

As regards rainfall, it would seem that a moderate degree of rain favours, and is probably essential to, cholera prevalence, though excessive wet arrests the spread of the disease. Further information upon this subject, however, will be found at page 348, vol. i. of this work.

Influence of Race.—There is a general consensus of opinion among authorities to the effect that the negro race is especially liable both to attack and death by cholera. This has been particularly noted on the East Coast of Africa, at Mauritius, Réunion, and other places. But as to the relative susceptibility of other races little seems to be certainly known.

Of Sex.—Judging by the experience of the three cholera epidemics which have occurred in England during registration times, the mortality at all ages taken together is rather greater among males than females, the male mortality being the higher up to the age of fifteen years and after that of sixty-five.

The fatality (or case mortality) is greater for females than males in the age period 0-5 and during the age periods 10-15 and 15-25; at all other age periods it is greater for males.

And of Age.—As regards age apart from sex, the actual number of deaths is much greater during the age period 0-5 than at any subsequent age period, and of the years included in the period 0-5, it is greatest in the second year. If the mortality at different age periods is considered in relation to the numbers living at those periods, it appears that it diminishes from 80.1 per 10,000 living to 12.9 and 7.0 respectively during the first three lustres of life (0-5, 5-10, and 10-15). It then remains almost stationary, though increasing slightly, for the next ten years, after which it increases steadily with each succeeding decennial period until it reaches 43.6 for the period 75-85.¹

The case mortality is also greatest in children and old persons.

Cause and Modes of Dissemination.—The ultimate dependence of cholera upon microphytic life-processes is here provisionally assumed. The evidence with respect to the bacteriology of cholera is, however, dealt with

¹ See *Report on the Cholera Epidemic in England*, Dr. W. Farr (Supplement to the *Twenty-ninth Annual Report of the Registrar-General*).

at page 175, *ante*. Among the circumstances affecting the development and diffusion of cholera, conditions of locality are of great importance. On a large scale this is seen in the constant presence of the disease in Bengal, its so-called 'endemic centre.' On a smaller scale it is seen in the frequently observed fact that certain localities, even particular parts of towns, have been severely invaded in successive epidemics, while other more or less circumscribed localities have repeatedly escaped serious invasion, notwithstanding repeated importations of the poison.¹

It is doubtless true as regards European experience, that in the instances in which particular localities have again and again been heavily attacked, a partial, and in some cases, probably, a complete explanation of the fact is to be found in the existence at such places of polluted water-supplies or other unwholesome conditions. But, in a general way, there is much evidence of the influence of other factors, such as elevation; nature and character of the soil, together with its natural purity or organic contamination; moisture and temperature of the soil, and variations in the level of the ground-water. These matters, however, are discussed in the first volume of this work (p. 389).

That sanitary defects are conducive to cholera prevalence and mortality is unquestionable. Such defects no doubt operate indirectly by inducing a lowered standard of health with diminished power of resistance to infection, but their main danger lies in the specific contamination of air, soil, food, and especially water, to which they give rise. Mr. Macnamara speaks of the general agreement of Anglo-Indian authors 'that cholera, when extending over a country, often settled on the inhabitants of low-lying, ill-drained, and overcrowded localities.'² Sir John Simon, commenting in his Ninth Annual Report to the Privy Council upon the experience of the cholera epidemic which England was then (1866) passing through, remarked: '... The diffusion of cholera among us depends entirely upon the numberless filthy facilities which are let exist, and specially in our larger towns, for the fouling of earth and air and water, and thus secondarily for the infection of man, with whatever contagium may be contained in the miscellaneous outflowings of the population. Excrement-sodden earth, excrement-reeking air, excrement-tainted water, these are for us the causes of cholera.'

But the conditions so far referred to are conditions tending to promote the activity and diffusion of the cholera organism when it is present in the locality. It has, however, already been seen that the conspicuous feature in the behaviour of cholera during the present century has been its liability at frequent intervals to escape from its endemic centre, and sweep over more or less wide areas of the earth's surface. Various theories of 'cholera waves' and the like have been advanced to explain these extensive marches of the disease, but, the more closely the matter is studied, the more evident does it appear that the actual migration of the disease is accomplished by human agency.

Not only does a survey of cholera pandemics exhibit their tendency to advance along the lines of human communication, but a detailed study of particular outbreaks sufficiently often discloses the fact of the importation of the disease by infected persons or their infected clothing.

Doubtless the variations in the amount of cholera prevalence in India from time to time, and the periodical diffusions of the disease from that country, indicate the operation in particular years at the endemic centre, and, perhaps more or less throughout the march of the disease, of meteorological

¹ See Hirsch, *op. cit.*, vol. i. p. 439.

² Article on 'Cholera,' Quain's *Dictionary of Medicine*.

logical or other conditions which are favourable or the reverse to the multiplication of the cholera organism or to the promotion of its pathogenicity; but the evidence goes to show that the actual transport of the organism is effected by human agency.

The mode by which the disease is communicated, however, differs notably from that of small-pox, typhus, and other typical infectious diseases, and more closely resembles that of enteric fever. It is not, as with typhus, those who are in attendance upon the sick who are especially singled out for attack. Such persons do not, as a rule, appear to suffer in greater proportion than the other inhabitants of the infected district. On the other hand, there is abundant evidence that cholera may be communicated by drinking water contaminated with the bowel discharges of persons suffering from the disease. With respect to this evidence the reader must be referred to the article on 'Water' in the first volume of this work (p. 267).¹

It is not, however, maintained that water-carriage constitutes, even in this country, the only means of the propagation of cholera. All that is claimed is that experience has proved that contaminated water-supplies have played a conspicuous part in the dissemination of the disease. On the other hand, the behaviour of cholera seems to require for its explanation a theory of the ability of the cholera organism to carry on certain of its life phases in the soil—a view which is now becoming generally held in this country with regard also to the organisms of enteric fever and diarrhoea. It is probably also true that the cholera organism is capable, under certain circumstances, of escaping from its habitat in the soil and infecting human beings, either directly or by fastening on to food. This almost certainly occurs in the case of the closely allied malady, diarrhoea.

It has been again and again observed that in England cholera has attained its widest diffusion during the second year of its appearance in the country; and in so far as the later diffusions were connected with the earlier appearances of the disease, it can hardly be doubted that, during the interval between the two, the cholera organism, although reduced to a relatively latent condition as regards pathogenic manifestation, must have continued its existence—presumably in the soil.

We are indebted to Mr. Power for the following hypotheses formulated by him in 1888-84 in provisional explanation of the general behaviour of cholera, and especially of the character of English cholera epidemics above referred to:—

CHOLERA HYPOTHESES, 1888

1. The cause of cholera is a living organism.
2. This organism has phases in its life-history, during some of which it has not, and in others it has, the power of producing cholera. Let these respectively be called 'non-malignant' phases and 'malignant' phases. In all phases the organism has the power of multiplying itself.
3. In most parts of the world the organism can exist in phases of both sorts. But only within certain geographical limits—its 'customary' area—has it the faculty of passing from non-malignant into malignant phase.
4. Alike in its non-malignant and in its malignant phase the organism is liable to be conveyed—in the body of a human host or otherwise—beyond its customary area, and be deposited here and there throughout the world.
5. Such conveyance of the organism in its non-malignant phase can never be demonstrable—i.e. by the appearance of cholera—inasmuch as *ex hypothesi* 3 it cannot, when existing outside its customary area, pass into its malignant phase.

¹ See also Dr. Snow, *On the Mode of Communication of Cholera*; Mr. Macnamara's *Treatise on Asiatic Cholera*; and various reports in the Eighth and Ninth Reports of the Medical Officer to the Privy Council (1885 and 1886).

6. But such convection of the organism when it is in its malignant phase can result in cholera, first, among the population immediately receiving it; secondly, among populations receiving it from that first population.

7. The resulting cholera in such case will appear only when local and climatic conditions are favourable, and the absence of cholera cannot, therefore, alone be evidence of the non-convection of the organism in its malignant phase.

8. The total of the local and climatic conditions required for the appearance of cholera in places outside its customary area, to which the cholera organism has been conveyed, is never continuous, but always recurrent or cyclical.

9. At a time when this total of conditions is present, the conveyed organism, being in its malignant phase, produces cholera. It produces cholera, however, only in quantity proportioned to its own quantity, or to the quantity of its immediate descendants produced during the maintenance of the requisite conditions. [Wherefore the local outbreaks of the disease are apt to be of small dimensions when they appear for the first time after the convection of cholera to a place.]

10. At a time subsequent to the original introduction into a place of the cholera organism in its malignant phase, the total of local and climatic conditions requisite for the appearance of cholera may be absent; and in that case the cholera organism, though in its malignant phase, can for a while await the reappearance of the required conditions. On their reappearance, cholera will appear as in § 9.

11. This waiting cannot be indefinitely prolonged, consistently with maintenance of the malignant phase among the descendants of the imported organism. If prolonged, the required local and climatic conditions can appear, but no cholera will appear.

12. After the appearance of cholera in a place, and when the local and climatic conditions required by the organism in its malignant phase are passing away, the descendants of the original organism will be, for a while, in existence, and they will be, in greater or less number, endowed with the same quality of malignancy as their ancestors.

13. From these immediate descendants, malignant and non-malignant, a succession of generations can be produced, the generation always tending towards the non-malignant phase of the organism.

14. Upon recurrence of the requisite local and climatic conditions, such organisms as have assumed the non-malignant phase cannot produce cholera.

15. Upon such recurrence, the organisms which continue to live in their malignant phase may or may not have become more numerous than in earlier generations. Often they have become more numerous. [Wherefore local outbreaks of cholera following upon a slight original outbreak (§ 9) are apt to be of larger dimensions than the original outbreak.]

16. The tendency of cholera organisms in their malignant phase, when existing outside their customary area, to die or to exhibit the non-malignant phase in their descendants, overcomes in the long run the tendency to multiply in any malignant phase. [Wherefore, unless exceptional conditions be present, the tendency of cholera is to disappear from localities outside its customary area.]

In connection with this subject, the remarks of Dr. Sims Woodhead upon cholera in his paper, already quoted, are of considerable interest. He points out that the cholera organism, when grown anaerobically, gains increased virulence, but largely loses its power of resistance to germicidal agents. Conversely, when grown aerobically it largely loses its poison-forming function, but gains in power of resistance. Its cultivation in the bodies of human hosts, therefore, while augmenting its virulence, does not tend to conserve that section of a given crop which has taken to colonise in the human subject. On the other hand, its aerobic existence outside the body, while diminishing its ability for immediate harm to human beings, increases its ability of maintaining itself (other essential conditions being present), and, consequently, its prospective danger to man when the season most favourable for its migration from the soil shall come round. In this way, also, Dr. Woodhead explains the fact that cholera displays but little tendency to spread immediately from person to person, notwithstanding that there is ample evidence of its dissemination by fomites, such as infected body-linen.

With respect to the subject of the spread of cholera, several other matters require mention. Most observers consider that the virus is given off

by the discharges from the sick, especially, according to some authorities, when those discharges are undergoing decomposition. To the recipient the virus would seem most usually to gain access by the alimentary canal, though it is not unlikely that it may be inhaled. The poison, as already seen, is without doubt in the main transported by human agency, but it is probably also, to some extent, air-borne, though only for short distances. An interesting case of the spread of cholera by milk has been recorded by Dr. Simpson, the Medical Officer of Health for Calcutta. In this case the milk was admittedly diluted with water contaminated with choleraic discharges.¹ Great and continued fatigue and alcoholic excesses are said to predispose to cholera.

Period of Incubation.—The period of incubation of cholera is usually quite short—from a few hours to three days; but, according to the late Dr. Parkes, it may be prolonged for ten or even twenty days.

Protection.—Cholera affords but a very slight degree of immunity, if any, against a subsequent attack.

Relation to other Diseases.—Cholera exhibits certain striking points of similarity to the epidemic diarrhoea of this country. In his report upon the latter disease Dr. Ballard remarks: 'The kinship of "diarrhoea" to malignant cholera is seen principally in the fact that both are clinically diarrhoeal diseases in which the abundant watery discharges from the bowels are more or less rapidly succeeded by collapse, in which there is some similarity (in diarrhoeal cases about to become fatal), in the *facies*, the sunken eyes, the shrinking of the bulk of the body, and algidity in certain cases. In both there is free desquamation of the intestinal epithelium; in both the kidneys are early implicated; and in both this condition may issue in uræmia and its results. Communicability through the medium of the morbid evacuations, although reputedly a character of malignant cholera, does not appear to be a character uniformly attaching to the disease; nor is non-communicability through the same medium a character invariably attaching to the epidemic diarrhoeal malady.' Indeed, it would appear that, regarded from the clinical view point, such differences as there are between the two maladies are largely differences in degree of malignancy.

Etiologically, too, these diseases exhibit many points of likeness. Both appear to be associated with filth, and especially with excremental filth. Both seem to be very similarly influenced by season, and especially by heat. The physical conditions of soil favourable to the one disease appear to be precisely those which are favourable to the other, there being in each case much evidence to show that the prevalence of the disease is greater or less according to the permeability of the soil, and its consequent ability to harbour organic refuse.² As regards the degree of moisture of the soil favourable to the prevalence of these two diseases, there is again similarity—a moderate degree of moisture being favourable, and excessive wetness unfavourable, to both.

Lastly, while Dr. Ballard has shown that a close relationship subsists between the temperature of the soil and diarrhoea prevalence, a very similar relationship between the temperature of the soil and the spread of cholera seems to have been observed by Delbrück, and also by Pfeiffer, though their observations have not as yet been corroborated.³

¹ *Practitioner*, vol. xxxix. p. 144.

² Compare especially the evidence adduced by Ballard with respect to diarrhoea (Supplement to the Report to the Local Government Board, 1887), and Hirsch (*op. cit.*, vol. i. p. 452-3) for cholera.

³ See Hirsch, *op. cit.*, vol. i. p. 461.

From what has been said, it will have been seen that, both clinically and etiologically, diarrhoea and cholera have many points in common. It is, of course, generally held that they are absolutely distinct diseases; but there would seem at least some grounds for the surmise that cholera may after all be but an Asiatic variety of a disease known elsewhere as 'diarrhoea' and cholera nostras—a variety, that is to say, which has been reared during the course of time under conditions of environment especially suitable for the fostering of its malignancy. The more pronounced communicability of cholera, as compared with diarrhoea, is no sufficient objection to this view, for not only is it a fact that diarrhoea in England is also at times communicable, but the heightened malignancy which is postulated of cholera by the above hypothesis might reasonably be expected to carry with it an increase of communicability.

As an objection to the thesis here suggested, it might be urged that, according to experience in this country, contaminated water plays a far more conspicuous part in the dissemination of cholera than in that of epidemic diarrhoea. But with regard to this it has to be remembered, that while on the one hand epidemic diarrhoea in England does sometimes appear to be associated with contaminated water-supplies, on the other hand, it is by no means certain that in India the contamination of drinking-water is responsible for the same proportionate share in the diffusion of cholera as would appear to have been the case with respect to the English epidemics of that disease; indeed, if such were the case it would be difficult to understand the attitude taken up by many Anglo-Indian authorities with respect to this matter.

May it not rather be with cholera in India, as it would seem to be with diarrhoea in England, that in localities in which the disease is conspicuously endemic, the soil is so charged with the necessary microzyme [which, being indigenous to the locality, is capable of maintaining itself continuously and of 'cropping' year by year] that the disease, although doubtless even there disseminated from time to time by water, is also largely, and perhaps more largely, disseminated by direct emanation from the soil—that in India, in other words, the spread of cholera is an affair more after the kind of that of diarrhoea in England? But that when the cholera microzyme is carried into other lands, and more especially into countries within the temperate zone, where the conditions are less favourable to its rapid multiplication in the soil, its opportunities for infecting man, particularly during the first season of its importation, are more largely dependent upon those chance occasions upon which it is conveyed to him in infected articles of clothing or of commerce, contaminated water-supplies, and the like? Such a view might, perhaps, afford a basis for reconciliation between the Anglo-Indian and British schools with respect to the etiology of cholera.

In closing these remarks upon cholera, one note of warning may be sounded. It must not for a moment be concluded that, because England has escaped cholera since 1866, she will enjoy a permanent immunity from that disease. It is true, of course, that of late years very considerable improvement in the sanitary condition of the towns has taken place, and, other things being equal, such improvement may doubtless be relied upon as meaning an equivalent diminution in the liability of cholera to spread. Nevertheless, there are still many localities in this country in which, owing to the supineness, in the past, of the Sanitary Authorities presiding over them, cholera would most undoubtedly cause havoc if it should unhappily gain access to them. Moreover, we cannot count upon 'other things being equal.' There is cholera and cholera, and the chance of its obtaining a foothold with

us must doubtless largely depend upon the amount and the type of the cholera sent to our shores. To illustrate the point, it may be mentioned that any person who, in view of our prolonged immunity from influenza, had inferred our permanent freedom from that malady, would during the last three years have had a somewhat rude awakening. Of course there are vast differences between influenza and cholera, but the biological factor has to be reckoned with as regards both, though no doubt it counts for less in respect to the spread of the latter than the former disease.

Neither must any abatement of cholera in Europe during the winter of 1892-93 be taken as meaning the end of our danger. On the contrary, reference to the behaviour of European cholera in the past gives abundant grounds for anticipating that it will almost certainly reappear upon the Continent in the spring, and attain, probably, a far wider prevalence there than it has done during the summer of 1892. If such should unfortunately turn out to be the case, we shall have to defend ourselves against imported cholera from a number of different ports, and for a period of many months. And it must be remembered, then, that no system of quarantine or of medical inspection can be safely relied upon to keep out the disease, and that the only true safeguard is to be found in pure water-supplies, efficient methods of excrement and refuse disposal, and wholesome conditions of life generally.

EPIDEMIC DIARRHŒA

Synon. : *Choleraic Diarrhœa* ; *English Cholera* ; *Cholera Nostras* ; *Infantile Cholera* ; *Muco-enteritis* ; *Gastro-enteritis* ; *Dysenteric Diarrhœa* ; *Alvus soluta* ; *Defluxio*. Fr. *Diarrhée*, *Cours de ventre* ; Ger. *Durchfall* ; It. *Diarrea*.

Diarrhœa, in the sense of a mere flowing from the bowels, may be simply a physiological process—the natural reaction of the healthy bowel against obnoxious contents. Again, it may be due to nervous influences, or be symptomatic of various morbid conditions of one or other of the internal organs ; and sometimes it is compensatory, or ‘vicarious.’ But there are now abundant grounds for the view that the diarrhœa which so conspicuously swells the mortality returns is of quite another sort, and, indeed, belongs essentially to the category of epidemic diseases. This has for some years been recognised by the Registrar-General, who includes diarrhœa among his ‘Principal Zymotic Diseases.’ The Royal College of Physicians also classifies ‘Epidemic Diarrhœa’ along with ‘Specific Febrile Diseases.’

This view of the nature of diarrhœa was based in the first instance upon the broad analogies between diarrhœa, regarded in a comprehensive manner, and other epidemic diseases. Thus, diarrhœa affects large numbers of persons at the same time and place ; it displays a very decided affinity for certain populous localities (indeed, so marked is this circumstance as to have led the Registrar-General to frequently refer to certain towns as diarrhœal towns) ; and it is influenced in a remarkable degree by season.¹

But the more recent researches of Drs. Ballard and Klein have now established the title of diarrhœa to be regarded as an epidemic disease. Apart from

¹ The seasonal rise in the diarrhœa mortality, inasmuch as it occurs during the height of the summer, has often been attributed to the eating of fruit. This, however, can hardly be the true explanation, since the mortality from summer diarrhœa falls mainly upon the children under one year of age. Such children do not, as Dr. Longstaff remarks, eat extensively of fruit, and this cause could therefore only be thought of as

the more detailed points of agreement between it and other epidemic diseases, in the matter of epidemiological features, which may be gathered from the summary, about to follow, of Dr. Ballard's Report,¹ these observers have conclusively shown that diarrhoea is marked out, both by its symptoms and pathology, as a general disease, of which the diarrhoea is but one of its several manifestations. In addition to certain changes in the alimentary canal which might have been anticipated, decided changes were invariably found after death in other organs, notably the kidneys, liver, and lungs. Indeed, Dr. Ballard remarks that 'in the bodies examined there were marked pathological changes, not only in the intestines, but in all the viscera; and not alone in the viscera of protracted cases, but in those of infants the total duration of whose illness had not exceeded twelve or fourteen hours.'

In studying the symptomatology of diarrhoea, Dr. Ballard and Mr. Power personally inquired into 840 fatal cases at Leicester during the summers of 1881 and 1882.

The 'leading phenomena' of the disease Dr. Ballard describes as 'diarrhoea, vomiting, convulsive phenomena; a bodily temperature at certain periods above, at other periods below what is normal; reduction in quantity or actual suppression of urine, embarrassed breathing, and, where looked for, commonly physical indications of pulmonary hyperæmia or inflammation, pallor of surface of the body, loss of bulk and flesh, and exhaustion, with its various well-known clinical features. I must add, that occasionally there is jaundice. Now and then a (fugitive) rash has been observed on the body.'

Dr. Ballard's more detailed remarks upon these symptoms should be studied in his report. It may, however, here be mentioned that, while stating that diarrhoea is most frequently 'the predominant symptom,' he says: 'I may here state my strong suspicion, almost my belief, that the malady usually characterised by diarrhoea may run its course from first to last, and even to death, without any remarkable diarrhoea at all. In other cases, although diarrhoea occurs, it is by no means the prominent symptom of the disorder; it may be comparatively of trifling amount or of short duration.' As regards vomiting, he points out that 'in only 48 out of 826 fatal cases occurring in Leicester of which I have notes (and in which the presence or absence of this symptom is mentioned) was vomiting absent altogether.'

The vomiting usually occurs coincidently with the diarrhoea, but 'occasionally it stands alone, perhaps for a whole day, as the prominent feature of the attack.' 'As a rule, the shorter and sharper the illness, the earlier in its course does this symptom appear.' Convulsions were present at some period or other of the attack in all but a small percentage of these fatal cases. They were mostly regular 'fits,' but in about a fifth of the cases they consisted of one or other of the well-known minor convulsive phenomena of children. With respect to the convulsions, Dr. Ballard remarks that 'there is reason to regard them not only as one of the most important phenomena of the malady, but, when occurring, as they mostly do occur, late in the illness, as indicating (and probably then as often due to) an uræmic condi-

operating in a wholesale way, 'through the maternal organism, which is possible, though not in a high degree probable.' Another objection to the theory is, that in the rural districts, where the eating of fruit might be expected to be at a maximum, the diarrhoea mortality is at a minimum. Similarly, the increased summer mortality has been attributed solely to atmospheric heat. It was, however, some time ago pointed out by Sir George Buchanan that a high temperature during the second quarter of the year does not give rise to the typical annual epidemic, and, as will be seen later, it has now been shown by Dr. Ballard that atmospheric heat alone does not suffice as a cause of the increased mortality.

¹ Supplement in continuation of the Report of the Medical Officer of the Local Government Board for 1887.

tion, the result of the kidney affection invariably present in these cases. The *comatose condition* in which the patient often dies appears also as a rule due to the same condition of the blood; but in some cases evidences of intra-cranial inflammation have been observed.'

In referring to the temperature charts published with his report, Dr. Ballard says: 'One important fact which they indicate, so far as my inquiry is concerned, is the "algide" character of the developed malady. At the commencement there appears to be some little febrile disturbance; but sooner or later, in the cases about to become fatal, the temperature falls more or less below the normal range, being lower in the morning than the evening, and even then it mostly fails to attain a normal standard. Towards the end of a fatal case the temperature is apt to rise. . . . In several cases of very short duration, where the child is rapidly prostrated with deluging watery stools, the temperature falls rapidly, and the child may die in collapse.'

In touching upon the subject of symptoms, we have gone somewhat beyond the scope and intention of these papers, but it seemed necessary to do so in the present instance, as an essential part of the argument for the right of diarrhœa to rank as a general disease.

With respect to the history and geographical description of diarrhœa little need be said. The malady is clearly described by Hippocrates, and seems to have been more or less present in all times and in all places. It has been pointed out by Sir George Buchanan, however, that, in its conspicuous epidemic form, diarrhœa appears, so far as this country is concerned, to be a comparatively modern phenomenon—at least, judging from the old Bills of Mortality.

Mortality.—The English mortality from 'diarrhœa and dysentery' year by year from the commencement of registration will be found in the following table, compiled from the Registrar-General's Annual Reports:—

TABLE XII.—*England and Wales.—Deaths from Diarrhœa and Dysentery.*

Year	Deaths	Rates per million living	Year	Deaths	Rates per million living
1888	8,109	208	1866	24,608	1,164
1889	8,099	200	1866	18,266	858
1840	4,097	260	1867	20,818	960
1841	3,755	286	1868	30,929	1,405
1842	6,002	372	1869	20,775	935
1848	—	—	1870	26,126	1,161
1844	—	—	1871	24,987	1,094
1845	—	—	1872	23,084	995
1846	—	—	1873	22,514	962
1847	14,842	868	1874	21,888	923
1848	13,696	787	1875	24,729	1,028
1849	20,881	1,189	1876	22,417	917
1850	18,504	760	1877	15,282	619
1851	16,918	941	1878	25,103	1,008
1852	20,378	1,117	1879	11,468	452
1853	16,068	874	1880	30,185	1,171
1854	21,995	1,181	1881	14,536	558
1855	14,207	754	1882	17,185	653
1856	15,150	798	1883	15,988	598
1857	22,887	1,189	1884	26,412	978
1858	15,331	787	1885	18,398	492
1859	19,710	1,001	1886	24,748	899
1860	10,858	546	1887	20,242	727
1861	20,162	1,002	1888	12,839	455
1862	12,156	597	1889	18,434	648
1863	15,994	775	1890	17,429	606
1864	17,482	882	1891	18,658	469

In connection with fatal diarrhoea, Dr. Ballard's report deals with two important and interesting matters: 1. The duration of the malady in fatal cases; 2. The influence exerted upon the duration of the fatal illness by previous healthiness or weakness.

1. It appears that the malady may be fatal in a few hours, or may linger for three, four, or even six or eight weeks. Of the 840 fatal cases observed at Leicester in the epidemic periods of the years 1881 and 1882, about one-half were fatal in less than a week; about one-quarter in between one and two weeks; between an eighth and ninth before the end of the third week; and about 15 per cent. at some time later than this. A further study of these fatal cases shows that the 'ferocity' of the malady, as judged by the shortness of the illness, increases with progress of the epidemic—at least, it was so at Leicester in the years 1881 and 1882. The increase, however, was not altogether a steady increase, but seemed inclined to occur in waves, and there were differences in the increase of the two years.

2. The previous health of 882 out of the 840 Leicester cases was ascertained. Of these 882, 141, or 42·5 per cent., were recorded healthy, and 191, or 57·5 per cent., either as weakly from birth or for a longer or shorter time prior to their fatal diarrhoeal illness. It would therefore seem that '*infantile diarrhoeal mortality is in part dependent upon previous general health.*' In connection with age in these cases, the figures showed that the influence of previous weakly condition on mortality was most conspicuous under nine months of age. It was also found that previous weakness had '*much to do with the speedy collapse of the most rapidly fatal cases and with the cases which have a duration of forty-eight hours and under four days, as also with the fatal cases of the most prolonged duration—i.e. three weeks and upwards.*'

But when the proportion of weakly to healthy children fatally attacked at different periods of the epidemic seasons of 1881 and 1882 came to be studied, it was found that the weakly were by no means specially attacked during the early periods. On the contrary, the evidence went rather to indicate that 'weakly children require a longer exposure to the epidemic cause, whatever that may be, than healthy children.'

Influence of Sex.—The incidence of the disease appears to be greater upon children of the male sex at all ages from birth upwards. The mortality is greater for males in infancy and old age, but usually rather greater for females from the second or third to about the fiftieth year. At all ages together it is greater for males, doubtless owing to the larger number of males living during the early period of life.

Influence of Age.—As regards age in relation to attack, Dr. Ballard finds that 'the actual incidence upon age (by which term wherever it is used is signified relation to the population living at the several ages) is chiefly upon children under five years of age, and of these most upon those under two years.

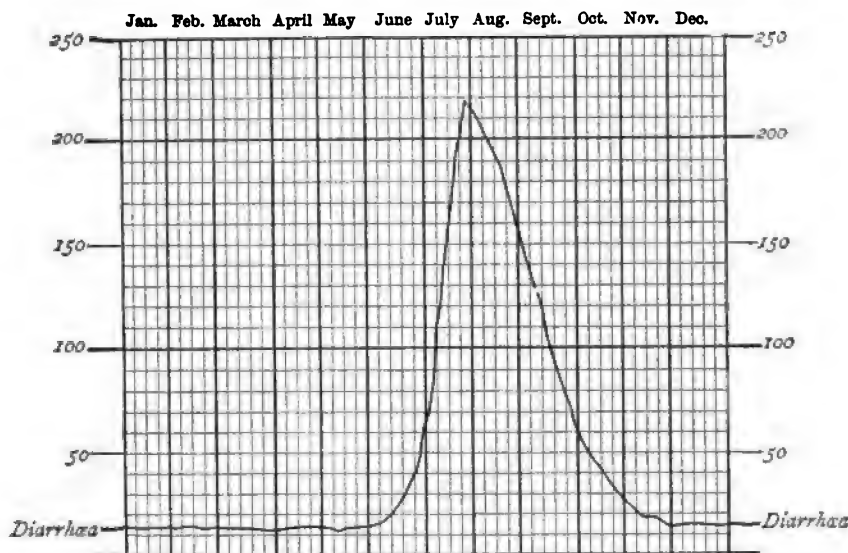
'After five years of age the actual incidence lessens remarkably, and is least between the ages of five and twenty-five years, after which it gradually increases.

'Among children under five years of age the incidence is comparatively small upon those under three months, after which age it apparently increases; beginning to lessen somewhere between the ages 1–2 years.' The incidence upon age, however, varies in different years, and it appears that 'in years of greater general prevalence the increased prevalence is more obvious, on the whole, upon those over five years of age than upon those under five years.'

The large majority of the diarrhoea deaths occurs during the first year of

life, the second three months of that year having the highest mortality. After the second year the deaths are few, and gradually diminish until the twentieth year, when they again steadily rise throughout the remainder of life.

Influence of Season.—The influence of season on London diarrhoea mortality is shown in the curve here given, which is taken from the Registrar-General in his Annual Summary for 1890. The curve represents the actual average number of deaths each week, and is based on the diarrhoea deaths



registered in London during the fifty years, 1841 to 1890. It appears from the curve that the conspicuous rise in the London mortality corresponds in point of time with the conspicuous rise in prevalence as shown in a case-table given by Dr. Ballard, and the maximum is attained in the same four-weeks period.

Cause and Dissemination.—The provisional results of Dr. Ballard's inquiry into the causation of diarrhoea may be summarised as follows :—

A. GENERAL CONDITIONS

Atmospheric Temperature.—A high atmospheric temperature conduces to diarrhoeal mortality, but it operates *indirectly*, and is not the main cause.

Temperature of the Earth.—This is a far more important condition. Dr. Ballard 'made for London and many other towns in the kingdom a large number of charts, showing week by week for many years the earth temperature at a depth of one foot from the surface and at a depth of four feet from the surface, each chart showing also the diarrhoeal mortality of the corresponding weeks.'

The general results shown by these charts are stated as follows :—

'a. The summer rise of diarrhoeal mortality does not commence until the mean temperature recorded by the 4-foot earth thermometer has attained somewhere about 58° Fahr., no matter what may have been the temperature previously attained by the atmosphere or recorded by the 1-foot earth thermometer.

'b. The maximum diarrhoeal mortality of the year is usually observed in the week in which the temperature recorded by the 4-foot earth thermometer attains its mean weekly maximum.

'c. The decline of the diarrhoeal mortality is in this connection not less instructive, perhaps more so, than its rise. It coincides with the decline of the temperature recorded by the 4-foot earth thermometer, which temperature *declines* very much more slowly than the atmospheric temperature, or than that recorded by the 1-foot earth thermometer; so that the epidemic mortality may continue (although declining) long after the last-mentioned temperatures have fallen greatly, and may extend some way into the fourth quarter of the year.

'd. I do not wish it to be inferred that the atmospheric temperature, and the temperature of the more superficial layers of the earth, exert no influence on diarrhoea. Their influence, however, is little, if at all, apparent until the temperature recorded by the 4-foot earth thermometer has risen as stated above. Then their influence is apparent, but it is a subsidiary one.'

Rainfall exerts an influence upon diarrhoeal mortality, but probably an indirect one, through its influence on the temperature of the soil.

Air-movement also influences diarrhoeal mortality, wind lessening, and calm, in the diarrhoeal season, promoting it.

B. CONDITIONS OF LOCALITY

Elevation above sea-level has some influence, but by no means a controlling one. Indeed, it seems to influence diarrhoeal mortality 'only in so far as it affects infant mortality from all causes together.'

Soil seems to have a decided influence, and this is, apparently, largely a matter of porosity. Of whatever nature the soil, the more loose and porous it is, the more conducive to diarrhoeal mortality. Hence, sand and mould most favour diarrhoea. Where the houses are built upon solid rock the diarrhoea mortality is low. Organic fouling of the soil, whether excremental or vegetable, is distinctly favourable to a high diarrhoeal mortality. Persons, therefore, living upon soil polluted by leaking sewers, drains, or cesspools, or upon 'made soil,' or the site of market-gardens, are apt to suffer from a high diarrhoeal mortality. Even persons living upon rock are liable to suffer more from diarrhoea if the rock is much fissured than if such is not the case, owing apparently to the opportunities for the accumulation of filth in the fissures.

Both excessive wetness and excessive dryness of soil seem to lessen diarrhoeal mortality, but a moderate dampness of soil favours it—i.e. 'an amount of habitual dampness which is decided, although not sufficient to preclude the free admission of air between the constituent physical elements of the soil.'

Density of population favours, and the reverse condition disfavors, diarrhoeal mortality. This is seen by a general comparison of the 'diarrhoeal' towns of various sizes, and by a comparison of that of the more and less crowded parts of the same towns. The influence of aggregation, although observable upon infant mortality from other causes, is most noticeable with respect to diarrhoea. It is, however, probably less direct than in the case of measles or scarlet fever.

Density of buildings (whether dwelling-houses or others) upon area, as in factory towns, appears to favour diarrhoeal mortality, and 'it is probable that this difference of density of buildings upon area is one of the circumstances which have to do with the difference of diarrhoeal mortality between large and small towns.'

Restrictions of, and impediments to, the free circulation of air, whether about or within dwellings, promote diarrhoeal mortality. As an instance of the latter case, 'back-to-back' houses are particularly objectionable.

Domestic darkness and general dirtiness within dwellings, as well as filthy accumulations of domestic refuse in privies, ashpits, dustbins, &c., are conducive to diarrhoeal mortality.

Sewer and cesspool emanations, 'especially in a concentrated form and suddenly let loose, may occasion attacks of fatal diarrhoea.' They may, probably, also cause a diarrhoeal epidemic in a non-diarrhoeal season, and hence in all likelihood operate in a similar fashion in the diarrhoea season.

Mere coal smoke, and emanations from chemical works, seem without special influence.

Undefined polluted conditions of drinking-water at times seem to cause epidemic diarrhoea, but there is at present no evidence that water-pollution plays a prominent part in the causation of the annual infantile diarrhoea mortality of this country.

C. CONDITIONS RELATING TO POPULATION

Social Position.—Diarrhoea mortality is notoriously greatest among the lower classes. This, however, is the case, and even to a greater extent, with mortality from other causes.

Food, no doubt, is concerned with the prevalence of epidemic diarrhoea, though probably not, as generally supposed, by reason of its causing ordinary dyspepsia, but owing to its contamination with some substance, 'which substance is by itself an efficient cause of the malady.'

a. As regards the influence of the mode of feeding young infants. This was especially studied for Dr. Ballard by Dr. Hope, the Assistant Medical Officer of Health for Liverpool, the general result of whose inquiries closely coincide with previous medical opinion on the subject. They are expressed by Dr. Ballard as follows:—

'1. That infants fed solely from the breast are remarkably exempt from fatal diarrhoea, even among the low-class Irish, the degree of exemption being exactly the same among the Irish as among the English and other races in his city.

'2. That infants fed in whatever way with artificial food, to the exclusion of breast-milk, are those which suffer most heavily from fatal diarrhoea.

'3. That children fed partially at the breast, and partially with other kinds of food, suffer to a considerable extent from fatal diarrhoea, but very much less than those who are brought up altogether by hand.

'4. As respects the use of "the bottle," that it is decidedly more dangerous than artificial feeding without the use of the bottle.'

b. Food stored in dark, ill-ventilated places, and exposed to telluric or other unwholesome emanations, probably becomes liable to cause diarrhoea.

Maternal neglect conduces to infant mortality from diarrhoea. Thus, the mortality among illegitimate infants is greater than that among the legitimate from all causes, but the increase is rather greater in the case of diarrhoeal mortality than of that from other causes. This, however, is only observable in years of low epidemicity—'the presumably less potent or less abundant specific cause (in such years) operates fatally more easily on the illegitimate than on the better cared-for class of infants.' But in highly epidemic years the illegitimate seem to be the earliest to suffer.

The occupation of females from home, as conducing to neglect and artificial feeding of infants, promotes diarrhoeal mortality.

The propositions provisionally formulated by Dr. Ballard with respect to diarrhoea causation will be found on page 866, vol. i., and therefore need not be repeated here.

Diarrhoea is sometimes highly infectious, the infection apparently being associated with the bowel discharges of the patients. An account, by Dr. R. Bruce Low, of four particularly interesting and instructive outbreaks of communicable diarrhoea of this sort will be found in the appendix to Dr. Ballard's report.

Relation to other Diseases.—The question as to the kinship of diarrhoea and cholera has been considered in the section on the latter disease.

The analogies between diarrhoea and scarlet fever are interesting, and deserving of careful attention and study. They are thus referred to by Dr. Ballard:—

‘As in scarlet fever, so in “diarrhoea,” the condition of the kidneys is an essential element in the disorder, and the uræmia observable in the one disease finds its analogue in the other. From this view-point the intestinal lesions in diarrhoea may be but the analogue of the skin eruption in scarlatina. That the one disease is notoriously communicable from one person to another, while the other is usually regarded as non-communicable, goes for little as an argument against such analogy, since, on the one hand, very many outbreaks of scarlatina (namely, those of milk origin) seem to have hardly any such communicable quality, and, on the other hand, communicability is a quality not unknown among cases of epidemic diarrhoea. In both diseases it is from the cutaneous or quasi-cutaneous (mucous) surface chiefly implicated that the contagious principle gets abroad—viz. the skin and throat surfaces in scarlatina, and the intestinal mucous surface in “diarrhoea.”’

The question of a possible relationship between diarrhoea and enteric fever needs also to be kept in view.

Period of Incubation.—Judging from Dr. Bruce Low's cases of communicable diarrhoea, the period of incubation appears to be very short—on an average, ten to twelve hours.

MALARIAL DISEASE

Synon.: *Ague*; *Intermittent Fever*; *Remittent Fever*; *Bilious Remittent Fever*; *Paludal Fever*; *Miasmatic Fever*. Fr. *Fièvre Aiguë*, *Fièvre Paludéenne*, *Fièvre Rémittente*; Ger. *Wechselfieber*, *Bösartiges endemisches Fieber*; Ital. *Febbre Intermittente*, *Febbre Remittente*.

Malarial disease, being intimately associated with conditions of soil, has already been dealt with as regards its etiology and the measures necessary for its prevention, by Dr. Copeman, in the first volume of this work. As regards bacteriology, it has also been dealt with by Dr. Klein. There are, however, still certain points of interest which may be referred to here.

It will be observed, of course, that the synonyms given above do not all apply to clinically identical manifestations, and it is regarded by some as not unlikely that bacteriology may ultimately establish ‘specific’ distinctions between the conditions represented by certain of them, such as intermittent and remittent fever. Indeed, functional and even slight morphological differences are already said to have been observed among the microbes which

appear to stand in causal relation to the various forms of malarial fever. It must, however, be remembered that such differences, instead of indicating 'specific' distinctions among the micro-organisms in question, may but represent different phases, normal or developmental, in the life-cycle of one and the same species of organism; and that such is actually the case seems to be the inference suggested by epidemiological evidence. The several forms of malarial disease are, therefore, here regarded as varieties of the same malady, differing mainly in degree of malignancy.

As regards history, clear record of malarial illness is found in the medical writings of antiquity, but the fuller accounts of the disease seem to date from the sixteenth century. In its distribution malarial disease still covers an enormous area of the world's surface, though it is far more prevalent and intense in the tropical and sub-tropical than in the temperate countries; and it does not, apparently, extend at all beyond the limits of 68° N. and 57° S. 'Covering a broad zone on both sides of the equator, the malarial diseases reach their maximum of frequency in tropical and sub-tropical regions. They continue to be endemic for some distance into the temperate zone, with diminishing severity and frequency towards the higher latitudes. . . .'¹

Space does not permit of a detailed account of the distribution of malaria being given here, and for particulars on this point the reader must be referred to Hirsch and other authorities. The main features of its present distribution, however, seem to be as follow:—It is widely prevalent, and in a virulent form, in tropical Africa, especially on the west coast. It is also prevalent in Algiers and in parts of Egypt, as the Nile valley. In Asia it is conspicuously present throughout India, Ceylon, China, Afghanistan, Persia, Arabia, and Syria; in the Western Hemisphere, the West Indies, Brazil, Peru, the coast of the Gulf of Mexico, and the southern and central parts of the United States. In Europe it is most severely prevalent in Italy, but occurs to a greater or less extent in every country except the Farøe Islands and Iceland. Great Britain in the present day suffers but little, though the disease still lingers in the counties on the east coast—notably in the fen districts of Lincolnshire, Norfolk, Huntingdon, and Cambridge; but even in these localities it has of late almost disappeared. In Ireland it is but very slightly present, and Scotland is almost, if not entirely, free from it.

Although, as has been said, malaria is most prevalent and most malignant in tropical and sub-tropical countries, yet among such countries certain districts are far more malarious than others, while some enjoy a complete exemption in this respect. As regards India, for instance, the Presidency of Madras suffers much less than those of Bengal and Bombay. On the West Coast of Africa, according to Hirsch, malaria becomes less severe from Cape Lopez southwards, and this exemption 'becomes more and more marked the nearer we approach the Cape of Good Hope, which itself enjoys, along with St. Helena, an almost complete immunity from the endemic fever.' For instances of other important immunities, New Zealand and Tasmania are said to be completely, and Australia almost completely, exempt.

As might be expected of a malady presumably caused by micro-organisms that are largely saprophytic, malarial disease is, perhaps, more than almost any other disease of the class under consideration, entitled to be described as 'endemic.' Yet it is a point of great interest that even this disease has at times exhibited decided epidemicity, extending to localities in which it is not usually met with. This circumstance raises the question whether in such epidemic extension there is actual transport of the malarial organism beyond its ordinary endemic areas, or whether the exceptional appearance of the disease

¹ Hirsch, *op. cit.*, i. 197.

in localities not ordinarily prone to it may not be consequent upon the acquirement of pathogenicity there by organisms which are usually benign—as a result, of course, of exceptional conditions, meteorological or other. Or a third hypothesis might be suggested, to the effect that the exceptional conditions referred to operate indirectly by increasing the susceptibility of individuals, and thus rendering them vulnerable to attack by organisms, indigenous to the neighbourhood, but not usually pathogenic. The fact, however, that some districts, which up to a certain time have enjoyed immunity from malarial disease, such as Réunion and Mauritius, have subsequently become endemic centres of that malady, might be quoted as suggestive of the transportation of the organism. It seems to have been shown by Dr. Salisbury that the malaria organism may be transported from place to place in soil; and Sawyer records that while living in a malarious part of Illinois he ‘visited a friend at Milton, Mass., and fell ill of intermittent fever. The lady of the house, who interested herself greatly on the patient’s account, and who had never seen a case of ague before, had a slight aguish attack of fever and chill on the fifth day, with gastric disturbance; but she set aside the idea that she could possibly be ill of ague, as the disease was quite unknown in Milton, or occurred only now and then in imported cases. However, she had a more severe attack the day after, and on the ninth day the first pronounced paroxysm of ague occurred, and with that all doubt as to the nature of the disease vanished.’¹ This case is at least suggestive of the conveyance of malaria in clothing, or even of direct infection from case to case. And the fact that malarial disease did not become prevalent in Réunion until some three years after it had been epidemic in Mauritius, is consistent with its having been imported there by persons or things from the latter place. The large majority of observers have, of course, declared against the spread of malarial disease by infection, and it may, no doubt, be concluded that such a mode of dissemination is at least not the rule; but it is by no means certain that it never occurs. In studying the subject in future, opportunity should be taken of observing the behaviour of the disease in this respect when it occurs in epidemic form, especially beyond the limits of its endemic area, as it would probably be under such circumstances that its infectiousness, if it exists at all, would most show itself.

As regards the distribution of the various forms of malarial disease, the more severe kinds—e.g. the remittents—are most common in those tropical and sub-tropical districts in which malarial disease generally is most conspicuously endemic, the disease in temperate climates being more commonly of the less severe intermittent type. And the same seems for the most part to hold good as regards the sub-varieties of intermittent fever, the severer forms with the short intermissions, such as the quotidian, forming a larger proportion of the cases in warm than colder countries, and *vice versa*. With respect to variations in type from time to time, it appears, according to Hirsch, that ‘at the beginning of the epidemic, or the rise of the endemic, intermittent fevers are observed almost exclusively; that in the subsequent progress the cases of severe sickness become more and more numerous, predominating at the height of the epidemic, again becoming relatively fewer as the amount of sickness decreases; while only intermittent forms are observed at the close.’

This is highly suggestive of the progressive development of infectiousness, already referred to in connection with certain other epidemic diseases, and is very similar to the rise and fall of malignancy described by Dr. Whitelegge as characteristic of the true developmental cycles of measles and scarlet fever.

¹ *Boston Medical and Surgical Journ.*, Dec. 1867, p. 538. Quoted by Hirsch.

In several other respects, also, the phenomena of malarial disease are of special interest as regards their possible bearing upon analogous phenomena in connection with other diseases. The chronic character, for instance, of malarial disease, as evidenced by the so-called malarial 'cachexia,' and the fact that persons who have once suffered from intermittent fever are liable to recurrent attacks years after they have left a malarious neighbourhood, seems to have its analogue in the cases occasionally met with of chronic and recurrent diphtheria. The facts, too, with regard to protection or acquired immunity in malarial disease are of the highest interest, and deserving of special study in connection with the subject of protection generally. For here we have a disease in which repeated attacks are common, and, indeed, in which one attack may in a sense be said to increase liability to another, and yet, paradoxical as it may appear, there is evidence that the disease does, to some extent, at least, protect against itself. The answer to this seeming contradiction may possibly be, that while one attack increases liability to another attack, i.e. a recrudescence, it nevertheless protects against a *re-infection*. The negro race suffers decidedly less from malaria than most, and probably all, other races of mankind, but that their immunity is due to prolonged exposure to the poison is suggested by the alleged fact that negroes after long residence in non-malarious districts have, upon subsequent exposure to malaria, been readily attacked. As regards Europeans also, continued residence in malarious countries seems to confer protection against the more severe forms of the disease. On the other hand, it is the severe remittent form that, in unhealthy districts, frequently attacks new-comers. Lastly, while it is notorious that repeated attacks of the milder intermittent form of the disease are frequent, second attacks of the more severe remittent form, according to Maclean, less often occur, and persons who have suffered from remittent fever shortly after their arrival in a malarious country 'are seldom exposed to second attacks' [of remittent fever].

In the above facts we may, perhaps, see illustration of the establishment and repeated renewal of protection by small doses of the virus, and also evidence of relation between the permanency of protection and the dose or quality of the virus.

As regards sex- and age-influence, males appear to suffer more frequently than females, but this may be due to increased opportunities for infection. No age is exempt, but attacks are least frequent among the very young and the old. Sir Thomas Watson, however, quotes a case from Dr. Russell's 'History of Aleppo,' in which a 'woman had tertian ague, which attacked her, of course, every other day, but on the alternate days, when she was well and free, she felt the child [*in utero*] shake; so that they both had tertian ague, only their paroxysms happened on alternate days.'

Although in localities in which malaria is endemic the disease occurs in any season of the year, its prevalence yet appears everywhere to be largely regulated by season, though the particular time of year in which it is most common varies in different climates. Thus, in temperate regions, according to Hirsch, it seems to have two maxima, one in the spring, and another in the autumn, the latter one being the more important. In the warmer climates, where malaria is more rife, the maximum is in the late summer and early autumn, the spring maximum of the temperate climate practically disappearing; lastly, in the most malarious districts in the tropics the maximum prevalence is during and towards the close of the rainy season.

Malaria may, without doubt, be conveyed by air-currents, but for what distances is uncertain. Parkes considered that the distance is seldom greater

than one or two miles, and that it was to be doubted whether 'belief in transference of malaria by air-currents for ten, twenty, or even 100 miles is correct.' In any case, its conveyance by air is, to a large extent, arrested by belts of trees and sheets of water. On the other hand, 'when favoured by ravines and currents of heated air, it can scale mountains to a height which appears to differ in different climates, varying from four or five hundred to two or three thousand feet' (Maclean: Quain's 'Dictionary of Medicine').

The evidence with regard to the spread of malaria by water is conflicting. There is, no doubt, a very strong belief in this mode of its transmission among the inhabitants of malarious districts, and there certainly would seem to be some evidence in support of the view. Probably the strongest case is that recorded by Boudin. In this case 120 soldiers embarked in the 'Argo' for transport from Bona, in Algiers, to Marseilles. During the voyage 111 of them, thirteen of whom died, suffered from different forms of malarial fever. Two other vessels, carrying between them 680 soldiers, also from Bona, and arriving at Marseilles the same day as the 'Argo,' had no cases of illness at all, and the only ascertainable difference of circumstance between the troops in these ships and those in the 'Argo' was the difference of drinking-water. The latter were exceptionally supplied with water, which was said to have an unpleasant smell and taste, from a marsh near Bona; those on the other ship were supplied with good water. Finally, the nine soldiers on the 'Argo' who escaped were said to have purchased wholesome water from the crew of that vessel.¹

The incubation period of malaria would seem, according to the evidence, to vary considerably, but more extensive data are required. Fagge gives it as usually from six to twenty days, but quotes cases recorded by Maclean and Hertz, of Amsterdam, which seem to show that it may be as short as a few hours.

ERYSIPELAS

Synon.: *St. Anthony's Fire*; *The Rose*. Fr. *Erysipèle*; Ger. *Erysipelas*, *Rothlauf*; It. *Risipola*.

History and Geographical Distribution.—The term erysipelas, as applied to spreading inflammations of the skin accompanied by fever, is found in the earliest medical records; and, indeed, the distinction between erysipelas occurring in association with wounds and erysipelas (regarded as) occurring independently of wounds, which has survived to the present day, dates back to the writings of Hippocrates.

From time to time, however, opinions have differed widely as to the actual processes which should be embraced by the term erysipelas; whether, for instance, it should include erythema, and certain inflammatory conditions of the mucous linings of the throat, lungs, and pelvic viscera; whether, also, it is to embrace the suppurative inflammations of the skin and subcutaneous cellular tissue, known in this country as 'phlegmonous erysipelas'—a point contested by Volkmann and other German authorities.

Unfortunately, the precise knowledge required for definitely setting at rest some of these questions is still wanting, as will appear later on.

As regards geographical distribution, erysipelas is tolerably uniform in its distribution throughout the temperate zone of both hemispheres. It also

¹ For further evidence on the subject see vol. i. p. 272 of this work.

occurs with tolerable frequency in cold countries, as Iceland, the Farøe Islands, and Greenland, and is not uncommon in warm sub-tropical latitudes. It seems, however, to be of decidedly less frequent occurrence in the tropics, though there are accounts of it at Réunion; and in the traumatic form, at least, it is met with in India.

Mortality.—In the following table will be found the number of deaths recorded year by year in England and Wales as due to erysipelas for the years 1888 to 1891, exclusive of the four years, 1848 to 1846, for which period the causes of death were not abstracted by the Registrar-General.

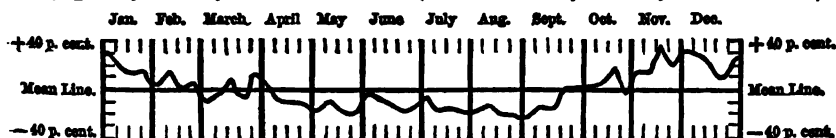
TABLE XIII.

ENGLAND AND WALES							
Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period	Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period
1888	1,203	81	76	1866	1,665	78	82
1889	1,140	75		1867	1,446	69	
1890	1,217	79		1868	1,948	88	
1891	1,189	78		1869	1,854	88	
1892	1,111	70		1870	2,120	94	
1893	—	—	106	1871	2,216	97	106
1894	—	—		1872	1,771	76	
1895	—	—		1873	2,027	87	
1896	—	—		1874	8,858	142	
1897	2,050	—		1875	8,018	126	
1898	2,618	—	111	1876	2,327	95	81
1899	2,808	—		1877	2,077	84	
1900	2,206	124		1878	1,870	75	
1901	1,998	118		1879	1,824	72	
1902	2,075	116		1880	2,014	78	88
1903	1,812	100	97	1881	2,314	89	
1904	1,987	105		1882	2,435	92	
1905	2,245	122		1883	2,166	81	
1906	2,125	113		1884	2,145	79	
1907	1,573	88	89	1885	1,996	78	54
1908	2,026	105		1886	1,523	55	
1909	1,954	100		1887	1,853	67	
1910	1,685	85		1888	1,635	59	
1911	1,542	78		1889	1,209	48	
1912	1,523	76	89	1890	1,375	48	48
1913	1,920	94		1891	1,263	48	
1914	2,104	102					
1915	1,963	95					

Influence of Season.—Just as it has been seen that erysipelas is apparently more abundant in the colder countries than in the tropics, so it is also decidedly more common, or at least more fatal, in the colder than in the warmer seasons of the year; and this seems to be true for all countries in which careful observations on the point have been made.

As regards London this result is illustrated in the mortality curve given below (Buchan and Mitchell. 'Scottish Meteorological Journal,' 1874-75).

Erysipelas, for all Ages and both Sexes.—(London Deaths for Thirty Years, 1845-74).



With respect to the influence of particular kinds of weather on erysipelas mortality, it will be seen by referring to Dr. Longstaff's plate (page 856) that the mortality from erysipelas, like that of scarlet fever, seems broadly to bear an inverse ratio to the rainfall. Cold east winds have often been stated to be conducive to erysipelas.

Influence of Race, Sex, and Age.—It has been alleged that negroes are immune against erysipelas; but later observations show that such is not the case, though perhaps they suffer less frequently than other races. With them there is no visible rash, but fever, swelling (which pits on pressure), and subsequent desquamation of cuticle. In the brown races the erysipelatous skin assumes a copper colour.

As regards sex, it is usually said that erysipelas is more common among women than men. However it may be with respect to attack, it appears from the table given below that during the ten years 1881-90 the deaths at all ages from erysipelas in England and Wales was greater among males.

TABLE XIV.

Period	Deaths at all ages			Deaths at subjoined ages								
	Male	Female	Total	0-3 Months	3-6 Months	6-12 Months	Total under 1 year	1-	2-	3-	4-	Total under 5 years
Ten years' 1881-1890	9,773	8,878	18,651	M. } 455	136	71	2,690	152	60	30	32	2,964
				F. } 368	181	102	2,729	174	76	45	35	3,069
					823	173	5,419	326	136	75	67	6,023
Deaths at subjoined ages—continued												
	5-	10-	15-	20-	25-	35-	45-	55-	65-	75-	85 and upwards	
M. } 120	120	149	180	478	824	1,105	1,383	1,447	851	152		
F. } 97	112	189	301	462	667	855	1,046	1,199	844	177		
	317	323	318	381	940	1,481	1,960	2,429	2,646	1,695	329	

The table also gives the age-distribution of the erysipelas deaths during the same years, and it will thus be observed that for the period in question practically 80 per cent. of all the deaths occurred during the first year of life. The deaths under one year were not split up by the Registrar-General into the three sub-periods shown in the table until 1888, but according to the figures for the subsequent years the mortality is very decidedly highest during the first three months of life.

Cause and Mode of Dissemination.—That the ultimate cause of erysipelas is a micro-organism is now sufficiently clear (see p. 98). In many instances this micro-organism unquestionably gains access to the system of the recipient through a wound in the skin, but whether this is always so is uncertain. It has been maintained, indeed, that the microbe of erysipelas is capable of obtaining entrance through the uninjured skin or mucous membranes. The time-honoured distinction between traumatic and idiopathic erysipelas is, of course, based upon the latter supposition. The distinction in question has, however, in recent years received far less acceptance than formerly, and the

¹ All the figures in this table refer to the ten years 1881-90, except those in the age groups 0-3, 3-6 and 6-12 months, which refer only to the years 1888-90.

view has been gaining ground that, even in the case of so-called idiopathic erysipelas, the virus enters the system through some insignificant and usually unobserved breach of surface. In support of this thesis it was pointed out by Trousseau that 'idiopathic' erysipelas may often be traced to a minute breach of continuity in the skin or mucous membrane at the angle of the eye or mouth, or to a slight eczema of the nose; and it is a matter of common experience that this form of erysipelas usually starts from some such point of junction between the skin and mucous membrane, where cracks, fissures, and eczema are peculiarly liable to occur. König succeeded in tracing fifteen out of twenty-nine cases of erysipelas of the head and face to injury of the affected parts, and in many of the remaining cases there was so much swelling at the time of admission to hospital that no satisfactory examination could be made.¹ On the whole, therefore, it seems probable that a wound of some kind, however trivial, is always present.

Erysipelas is certainly infectious, but is less uniformly so than many of the epidemic diseases, such as small-pox, typhus, and measles. The necessity for a wound (if such necessity exists) would doubtless, in part, explain the commonly smaller infectiousness exhibited by the disease among the general public, but even in the surgical wards of hospitals erysipelas often fails to spread. Its prevalence, however, in different hospitals and in the same hospitals at different times has varied largely. To some extent this is very likely due to differences from time to time in the infectiveness of the disease—i.e. to actual variations in the pathogenicity of the streptococcus. An interesting series of cases brought before the Paris Academy, in 1864, by Dr. Blin, and apparently indicating clearly an unusually high degree of infectiveness, are thus recorded by Fagge:—'One of the surgeons at the Lariboisière Hospital had under his care two patients suffering from erysipelas when he was himself seized with it. A medical friend from Guise visited him, and fell ill after returning to that place, where no other case of the disease then existed. That gentleman's servant was attacked, and also a relative who came to see him, and who lived in the neighbourhood. The latter gave erysipelas to his wife, and three members of another family, who were repeatedly in contact with them during their illness, suffered in their turn. From this family the disease spread to two Sisters of Mercy, and they carried it to their home and gave it also to a medical man who attended them; and, lastly, it passed from him to his daughter.'

But other circumstances play an important, though indirect, part in determining the prevalence of erysipelas in particular localities, hospitals, and, as has been often observed, even in particular wards of hospitals. The most important of these circumstances are overcrowding and defective ventilation, filth accumulation, general want of cleanliness, and defective drainage arrangements. In whatever way these circumstances operate, whether by increasing susceptibility, or as affording nidus and pabulum outside the body for the erysipelas microphyte, there can be no doubt as to their operation. '... The influence of any epidemic [of erysipelas] is immensely increased by an unhealthy condition of a ward from overcrowding,' says Mr. Erichsen; and this has been the general experience.

An interesting illustration of the effect of filth accumulations in this respect is afforded by the case recorded by Mr. Thomson in the 'Medical Times and Gazette,' December 1856, and also by the late Mr. de Morgan in Holmes' 'System of Surgery.' It appears that the occupants of two beds, one on either side of a window in a large ward on the ground-floor of the

¹ Quoted from Fagge's *Principles and Practices of Medicine*.

Middlesex Hospital, were frequently, during a period of years, attacked by erysipelas. Inquiry elicited the fact that a large uncovered dustbin was situated just below the window in question. Suspicion attaching to this dustbin, it was carefully cleaned out and covered over, erysipelas thereupon ceasing to invade the beds in question. Two years later patients in these beds were again attacked with erysipelas, and examination showed that the dustbin was again in a foul condition and had been left open. It was therefore entirely removed, and the disease disappeared from the ward.

In the year 1874 an epidemic of erysipelas in the Radcliffe Infirmary, at Oxford, was associated by Mr. Netten Radcliffe¹ with a blocking of the drains by faecal matter and the admission of foul drain-air to the hospital. Dr. König,² of Rostock, traced the spread of erysipelas at the hospital there to defective cleansing of the cushions on the operating-table, which were covered with old bloodstains. On discarding these pillows the erysipelas at once ceased.

Among predisposing causes special to the individual, intemperance, want of proper food, and visceral disease are said to be important. It is also often said that liability to erysipelas is increased by a previous attack. Certainly the disease seems to afford little, if any, protection.

Relation to other Diseases.—The question as to what clinical conditions should, on the ground of etiological identity, be included under the term erysipelas is, as has been said, one which it is as yet impossible to answer definitely. Moreover, in the rational study of the origin and natural history of disease, consideration of conditions of causation should take account of possible gradations of genetic relationship between associated maladies, whether or not having similar manifestations. As regards erysipelas, the widely different views which have been, and still are, held upon these matters by different authorities show the subject to be one especially deserving of careful and close study.

And as to this it is here only intended to put forward, provisionally, a few considerations, suggested for the most part by clinical or epidemiological experiences.

With respect, first, to the question of relationship between erysipelas and erythema, it has to be noted that, as often employed, the latter term is one of very uncertain signification. While, on the one hand, there can be little doubt that many so-called cases of erythema are, in reality, mild cases of erysipelas, it is quite certain that there are other conditions of superficial dermatitis which are altogether etilogically distinct.

Next there is the question as to whether erysipelas is 'specific' to the extent of being entirely distinct from various other forms of blood-poisoning which, without inducing any notable spreading dermatitis, give rise to local suppurative adenitis or more remote abscess. Many authorities have answered this question in the affirmative, but the manner in which cases of the latter sort are, in practice, sometimes closely associated with cases apparently of true erysipelas appears to raise great doubt upon the point. In this connection it is important to observe that Fehleisen's erysipelas streptococcus is microscopically and in culture so like the *Streptococcus pyogenes* that some bacteriologists regard the two as identical *quod* species, and consider the one an attenuated form of the other (see page 94).

But the question under consideration is bound up with that of the relation of erysipelas to certain other diseases, and there are not a few additional epidemiological facts which go to support the thesis of genetic relationship

¹ *British Medical Journal*, 1875, vol. i. p. 651. *Report of Medical Officer, Local Government Board*, 1876, pp. 88-69.

² *Arch. der Heilkunde*, 1870, p. 23.

between erysipelas and septic processes as such. It may be mentioned, first, that in this country at least experience has established the interchangeability of *cutaneous* erysipelas and the varieties of the disease characterised by deeper, diffuse, and suppurative inflammation. Clinical observation, too, long since indicated a connection between erysipelas and puerperal fever. Attention seems first to have been drawn to this connection during the latter half of the last century, and since that time a large number of facts have been recorded which go to indicate a very close relationship between erysipelas and puerperal fever. Not only has puerperal fever been many times clearly traced to infection carried by medical men to the lying-in-chamber from cases of phlegmonous erysipelas in their ordinary practice, but nurses and others suffering from commencing cutaneous erysipelas of the head or face seem without doubt to have set up puerperal fever in 'labour' patients they chanced at the time to be attending. Interesting cases illustrating these points are given by Hirsch, but it must suffice here to say that Hirsch summarises the grounds upon which belief in the etiological connection of the two diseases in question is based as follows:—

'(1) *The coincidence in time and place of the two diseases in epidemic form, both in lying-in institutions and among the population at large.*

'(2) *The familiar fact that women in labour attended by doctors or midwives who were suffering themselves from erysipelas, or had come in contact with erysipelas patients, have taken puerperal fever.*

'(3) *The converse fact to (2), that doctors, midwives, nurses, and other individuals who come into close contact with puerperal fever patients suffer from erysipelas remarkably often; also, that the new-born infants of mothers with puerperal fever die of erysipelas in an unusually large ratio.*

'(4) *The fact, vouched for by many observers, that childbed fever itself has not unfrequently an erysipelatous character, if I may so speak; or, in other words, that the disease begins to develop from an erysipelas which mostly arises in the lacerated vaginal mucous membrane.'*

Further evidence of relationship between erysipelas and puerperal fever is found in the similarity of their seasonal incidence, and more strikingly, perhaps, in the closeness with which mortality rises and falls in different years, as shown in the diagram reproduced, by Dr. Longstaff's permission, from 'Studies on Statistics.' In his diagram the rise and fall above and below the mean in the mortality of a number of diseases is indicated for the twenty-six years, 1855–80. With reference to the close resemblance between the curves of erysipelas and puerperal fever, Dr. Longstaff remarked in his original paper, read before the Epidemiological Society in 1880: 'I confess that I find it difficult to avoid the conclusion that they are both due to one poison.' In republishing this paper in his 'Studies on Statistics' in 1890 he has in an appendix given, in another table, similar mortality curves for these two diseases, along with rheumatism, but separately for each of the eleven registration districts of England and Wales, and for the longer period of thirty-four years, 1855–88. In these curves for the different sections of the country the fluctuations are greater than in those for the country as a whole, and, as might be expected, the correspondence between the curves representing the mortality of the three diseases is not always so close as in those based upon the larger figures of England and Wales. Nevertheless, it is conspicuous, and, as Dr. Longstaff remarks, 'far closer than mere chance coincidence would account for;' and he adds, '... a relationship which holds good in the several parts of the country, as well as in the entire area, cannot be fortuitous.'

But in addition to the resemblance of the erysipelas and puerperal fever

curves to one another, there is a decided likeness between those curves and that of pyæmia.¹

In addition, there are broad points of agreement between all the curves in the diagram (representing collectively Dr. Longstaff's, 'scarlatinal group' of diseases), in that the elevations and depressions so well-marked in the scarlatina curve are more or less traceable in all the others. There is also a general agreement between the curves in exhibiting a sort of inverse relationship to the rainfall, which is exhibited at the top of the diagram on Sir George Buchanan's method.²

One highly interesting result brought out by Dr. Longstaff's inquiries is the close resemblance between the curve of 'rheumatism of the heart' and those of erysipelas and puerperal fever. Is this to be taken as indicating that rheumatism is, after all, a septic disease? The subject is well worth close investigation. In the meantime, it must be remembered that ulcerative endocarditis, the origin of which, according to Fagge, is 'far most frequently in rheumatism,' is apparently a septic process (see page 92).

Lastly, the question arises as to the relationship, if any, of erysipelas to diphtheria. The frequency with which congestion of the fauces accompanies facial erysipelas has long been known. Sir Thomas Watson laid great stress upon soreness of the throat as an early clinical feature, and Mr. Marcus Beck³ mentions redness and congestion of the fauces as always present in erysipelas of the head. In some cases, known as erysipelatous pharyngitis and laryngitis, the disease is mainly limited to the throat.

In this connection the extensive and very malignant epidemics of 'ery-

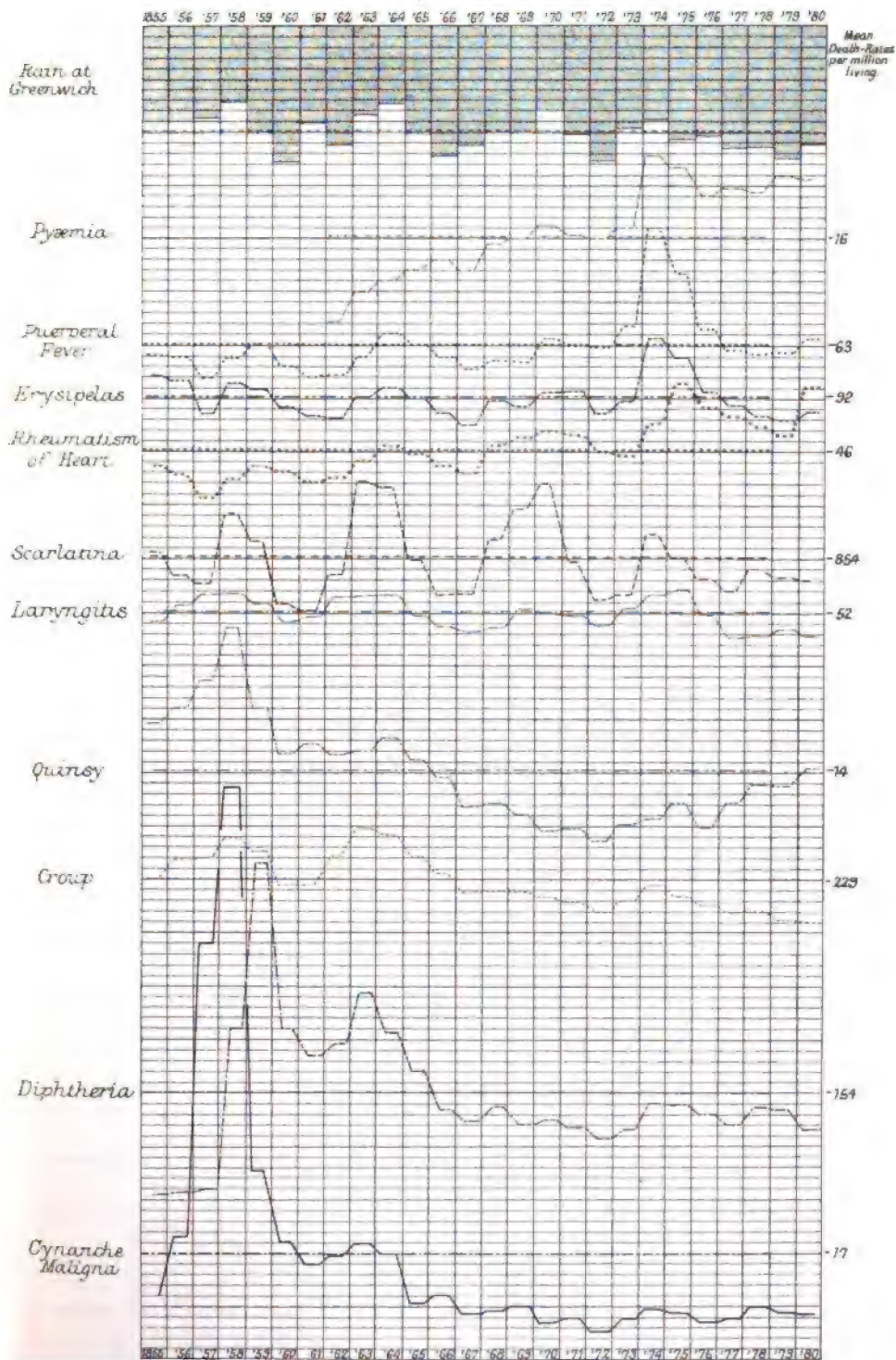
¹ The pyæmia deaths were not given by the Registrar-General until 1862, and, as Dr. Longstaff remarks with reference to the pyæmia curve, 'there can be little doubt that the steady rise from that year must be attributed to changes of nomenclature. . . .'

² It is only right to add that in the 1890 Appendix, above mentioned, Dr. Longstaff refers to the mortality returns for the further period of ten years, 1879-88, that had become available since the construction of his diagram, which originally embraced the period, 1855-78. In several respects it appears that the resemblances indicated among the curves in that diagram (Plate XLIV.) were not so closely maintained for the later period. The pyæmia curve, indeed, we learn has 'in recent years exhibited no distinct relationship to any of the others.' As regards puerperal fever and erysipelas, the curve for the former disease was above the average for nine of the ten years, and that of the latter was somewhat low, reaching the average only in 1882; but, 'nevertheless a close correspondence in the directions of the fluctuations of the two curves has been maintained.' The scarlet-fever curve has fallen so conspicuously that there is little discernible correspondence between it and other curves. The registered 'croup' mortality has somewhat declined, while that of diphtheria has steadily increased; but as regards their fluctuations these curves have continued to run similar courses; and the laryngitis curve has fairly corresponded with them. Lastly, there has not generally been the same close inverse relationship between these curves and the rain curve as had been previously observed, though in the very wet year of 1879 erysipelas, puerperal fever, and rheumatism of the heart were all at a minimum, while in the dry year of 1884 diphtheria was at a maximum. The discrepancies thus indicated between the results of these two periods were probably in part due to the operation of disturbing causes during the more recent period. Among such disturbing causes may be mentioned certain changes in the classification of deaths which were introduced by the Registrar-General in 1881; improvements in diagnosis and certification by medical practitioners, and increased attention to sanitary matters, which, with better sanitary administration, may have artificially diminished the mortality of some of the diseases in question, and so have tended to obscure natural similarities between them. No doubt also there were other less obvious disturbing causes in operation; but however that may be, the agreements between the curves which were maintained throughout the earlier and comparatively long period of twenty-six years are far too conspicuous to be ignored, and are certainly significant. It will be remembered, too, that as regards erysipelas and puerperal fever, and croup and diphtheria, the fluctuations in the curves have continued to run together.

³ Quain's *Dictionary of Medicine*.

ENGLAND AND WALES.

*Death rates from Erysipelas, Scarlatina & certain other diseases,
with rain at Greenwich.
All ages and both sexes.*



The broken horizontal lines indicate means for the twenty four years 1855-78.
The curves express the fluctuation per cent above or below the mean. Each
division of the vertical scale corresponding to 10%.
The figures at the side give the actual values of the Means of the Death-Rates



'erysipelas' which occurred in North America from about 1841 to 1864 are of especial interest. Throat symptoms constituted an early and conspicuous feature of the disease, and, according to Hirsch, in severe cases the mucous membrane was often found covered with ash-coloured sloughs.

Judging from the accounts of this epidemic, the phenomena of erysipelas and diphtheria seem to have been about equally mixed. Indeed, Volkmann is inclined to regard the disease as 'an affection very closely related to diphtheritis, perhaps even with pure diphtheritis of the throat.' Hirsch, on the other hand, explains that on clinical and etiological grounds he agrees with the American profession in regarding it as malignant erysipelas.¹

Period of Incubation.—The incubation period of erysipelas would seem to be short, and is given by most clinical observers as usually about four days or less. According to Murchison,² it may range from one to eight days, but is usually from one to three or four days. For inoculated erysipelas the incubation period seems to be, as a rule, much shorter. In Fehleisen's inoculations into rabbits with cultures of his erysipelas streptococcus the skin of the ear showed a deep blush after twenty-four hours. In his inoculations in the human subject, with cultures of this organism which were undertaken for the removal of sarcomatous growths, the period varied from fifteen to sixty-one hours.

PUERPERAL FEVER

[Synon.: *Childbed Fever*; *Puerperal Septicæmia*. Fr. *Fièvre puerpérale*; Ger. *Kindbettfieber*, *Puerperalfieber*.

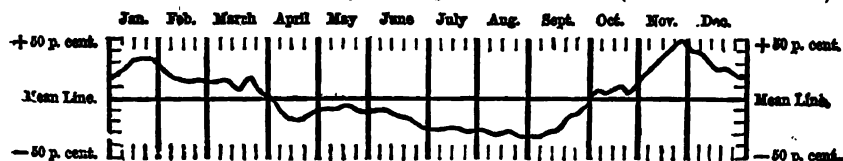
Puerperal fever has occurred in all ages, and seems to have been by no means uncommon in the days of Hippocrates. In more modern times, however, its prevalence was, no doubt, until recently much increased by the establishment and multiplication of maternity institutions. As regards distribution, it occurs in all climates, but, like erysipelas, less frequently in the tropical and sub-tropical than in the higher latitudes. In addition to this apparent preference for the higher latitudes, most observers are agreed that both its prevalence and mortality are greatest in the colder seasons of the year.

With respect to prevalence, Hirsch records that, of 195 epidemics 'for which the time of prevalence is accurately given,' there occurred:—

In winter	66	In autumn and winter	11
In spring	34	In summer	10
In winter and spring	25	In summer and autumn	7
In autumn	21	In spring and summer	5

As to mortality, the incidence upon the autumn and winter months is indicated for London in the following curve.

Puerperal Fever.—London Deaths for Thirty Years, 1845–74. (Buchan and Mitchell.)



¹ *Op. cit.*, vol. ii. p. 398.

² *Trans. Clin. Soc.*, vol. xi., London, 1878.

The deaths in England and Wales annually recorded by the Registrar-General as due to puerperal fever are given in the following table. But instead of the rate based upon the proportion of deaths to the general population, which is given in the corresponding tables for other diseases, a rate calculated on the annual number of births has been here substituted. Although this does not take account of still-birth confinements and miscarriages, it seems to be the closest indication obtainable as to the true increase, or the reverse, of mortality from this disease.

TABLE XV.

Year	England and Wales		Puerperal fever deaths to a thousand births	Year	England and Wales		Puerperal fever deaths to a thousand births
	Puerperal fever deaths	Births			Puerperal fever deaths	Births	
1847	784	539,965	1.5	1870	1,492	792,787	1.9
1848	1,365	563,059	2.4	1871	1,464	797,428	1.8
1849	1,165	578,159	2.0	1872	1,400	825,907	1.7
1850	1,113	593,422	1.9	1873	1,740	829,778	2.1
1851	1,009	615,865	1.6	1874	3,108	854,956	3.6
1852	972	624,012	1.6	1875	2,504	850,607	2.9
1853	795	612,391	1.3	1876	1,746	887,968	2.0
1854	954	634,405	1.5	1877	1,444	888,200	1.6
1855	1,079	635,043	1.7	1878	1,415	891,908	1.6
1856	1,067	657,453	1.6	1879	1,464	880,389	1.7
1857	838	663,071	1.3	1880	1,659	881,643	1.9
1858	1,068	655,481	1.6	1881	2,287	883,642	2.6
1859	1,288	689,881	1.8	1882	2,564	889,014	2.9
1860	987	684,048	1.4	1883	2,616	890,722	2.9
1861	886	696,406	1.3	1884	2,468	906,750	2.7
1862	940	712,684	1.3	1885	2,420	894,270	2.7
1863	1,155	727,417	1.6	1886	2,078	908,866	2.3
1864	1,484	740,275	2.0	1887	2,450	886,331	2.8
1865	1,333	748,069	1.8	1888	2,386	879,868	2.7
1866	1,197	753,870	1.5	1889	1,852	885,944	2.1
1867	1,066	768,349	1.4	1890	1,956	869,937	2.2
1868	1,196	786,858	1.5	1891	1,973	914,157	2.2
1869	1,181	773,381	1.5				

From this table it will be seen that puerperal fever attained its maximum recorded mortality during registration times in the year 1874. Nevertheless, it must be noted that, notwithstanding the great advance made in our knowledge of the etiology of this disease and our ascertained ability to control it by cleanliness, ventilation, and especially by antiseptic measures, the last ten years show a higher sustained mortality than any previous decade. This is no doubt largely due to better certification, and is, so far, apparent rather than real, more especially as in 1881 the Registrar-General adopted a system of writing to medical practitioners for further details in all cases returned simply as pyæmia, septicæmia, peritonitis, &c., in women of the child-bearing age—a system which has led to the inclusion under the head of puerperal fever of a number of deaths which would not otherwise have been so classified. If, however, all due allowance were made for improved certification, the period in question would at least not show the decline which we might reasonably have anticipated in the case of this essentially preventable disease.

This is the more remarkable since the application of modern knowledge (as indicated by generally improved hygienic conditions and the use of antiseptics) in the management of lying-in hospitals—institutions which in earlier years were prolific centres of puerperal fever—has led to a very marked decline in the puerperal fever mortality at such hospitals. According to

Dr. Cullingworth,¹ the mortality at the Vienna Lying-in Hospital (the largest in the world) was reduced by hygienic reforms from 28 per 1,000 in the years 1857-62 to 16 per 1,000 in 1863-80, and, subsequently to the introduction of antiseptics, has since fallen to 7 per 1,000. In the similar institution at Dresden the reduction has been from 50 per 1,000 in 1872 to 10 per 1,000 in 1886-87; in the New York hospital, from 60 per 1,000 in 1888 to 2 per 1,000 in 1885-86; and in the Boston hospital, from 55.5 per 1,000 in 1882 and 45.8 per 1,000 in 1883, to 16 per 1,000 in 1884, 6.4 in 1885, and none in 1886. At the Maternité, in Paris, the total death-rate was reduced by sanitary improvements from 98 per 1,000 in 1858-69 to 23 per 1,000, and, after the introduction of antiseptics, to 11 per 1,000. Equally satisfactory progress has been made in our own lying-in hospitals, from which puerperal fever has almost entirely disappeared. From this it seems to follow the sustained mortality from the disease in England must be due to its prevalence outside maternity institutions. Evidently, then, the conditions under which women are confined outside hospitals have not in an equal degree shared in the improvement undergone by the same conditions in hospital practice. Such improvement can only be brought about by a full appreciation of all that is known of the etiology of the disease, and an adequate sense of responsibility on the part of those brought into relation with the lying-in chamber. It must be remembered, as insisted upon by Semmelweiss, that puerperal fever is essentially a septic process, the virus gaining access to the body, in most cases, at all events, through the mucous surfaces of the utero-vaginal passages, to which it may readily be conveyed from case to case by the hands, instruments, and clothing of the attendants, or by sponges, bedding, and other articles. In one or other of the ways indicated puerperal fever has frequently been spread through a series of cases by medical practitioners, midwives, and nurses.

It is, however, not alone from puerperal fever products that the infection may be brought. There is ample evidence to show that it may come from various septic and decomposition sources altogether apart from the lying-in room. Many cases are on record which leave no room for doubt that puerperal fever, as such, has often had its origin in the attendance of women in labour by medical men who had shortly before conducted *post-mortem* examinations, or been in close attendance upon persons suffering from septic maladies.

The ability of erysipelas infection to give rise to puerperal fever has been elsewhere dealt with (p. 355), and there is reason also for thinking that scarlet fever and other infectious diseases may operate in a similar manner; but with regard to such diseases the evidence is not so strong as in the case of erysipelas.

Various other less direct causes play an important part in the origin, or at least the maintenance, of puerperal fever, the most important of which are overcrowding, insufficient ventilation, drainage defects, filth nuisances of all kinds, and want of cleanliness generally. There is clear evidence, for instance, that the past prevalence of the disease in lying-in hospitals, which has already been referred to, was largely due to these causes. It has frequently been recorded that outbreaks in such institutions occurred at times of unusual overcrowding, or were traced to want of ventilation and proper cleanliness; and we have already seen that marked diminution in mortality has followed upon improvement in these respects.

There is no evidence of association between puerperal fever and any particular telluric conditions.

¹ 'Introductory Address at St. Thomas's Hospital in 1888.' Reported in *Public Health*, 1888.

TUBERCULOSIS

Synon.: Fr. *Tuberculisatio*n, *Tuberculose*; Ger. *Tuberkelbildung*, *Tuberculose*.

Tuberculosis—that is, disease which is now known to be causally related to the operation of the so-called bacillus tuberculosis—occurs in the human subject in a variety of clinically different forms, the most notable of which are acute general tuberculosis, tubercular phthisis, tubercular meningitis, tubercular peritonitis, tabes mesenterica, scrofula, and lupus.

But although apparently all due to the same bacillus, the clinical differences between some of these several forms, say, for instance, between acute tuberculosis and lupus, are such as to suggest either corresponding functional differences between the bacilli causing them, or important differences in the tissue-condition of the persons affected. Tubercular disease is by no means limited to the human family. Several of the lower animals, particularly guineapigs and rabbits, as also fowls, have been shown by experiment to be susceptible to it, though in different degrees; and the bovine species suffer naturally, and in large numbers, from a form of tuberculosis known as 'the grapes.' Pigs also not uncommonly suffer from tubercular disease. With reference to the above matters, however, the reader must be referred to the article by Dr. Klein (p. 208, *ante*).

As regards the general history and geographical distribution of tubercular disease, it is certain that in some forms, as, for instance, pulmonary phthisis, it has occurred in all ages, and that it now occurs practically in all countries; but the data for forming even an approximate estimate of the relative prevalence in the past, at different times and places, of this and other forms of the malady are wanting.

In the present day the disease is far more common in some countries than others. Phthisis, for instance, is, taken generally, most prevalent in the countries within the temperate zone, and especially in the more populous parts of such countries. But that no particular temperature *per se* is a bar to its prevalence seems tolerably evident from the facts of its distribution. Thus, although many cold countries, such as Iceland and the Farøe Islands, apparently enjoy a marked relative immunity from phthisis, that malady is very common in North Greenland. On the other hand, as regards the countries with high atmospheric temperature, although phthisis is comparatively rare in Syria, Algiers, and other parts of the North Coast of Africa, it is common in certain parts of India, the islands of the Southern Pacific, and in Mauritius, Réunion, and Madagascar. A notable fact is, that in certain countries, especially Brazil, Australia, and the United States, the disease seems to have decidedly increased of late years.

Mortality.—That tubercular disease occasions an enormous mortality is a matter of common knowledge. It is impossible, however, to ascertain accurately the extent of that mortality, owing to the large element of uncertainty which attaches to the actual cause of many of the deaths, especially among children, ascribed to one or other form of tuberculosis. Hirsch, however, estimates that among civilised communities the average deaths from phthisis amount to one-seventh of the total mortality.

As regards England and Wales, Table XVI., compiled from the Registrar-General's Annual Reports, shows the number of deaths recorded year by year as due to phthisis, and the corresponding death-rates per million of the population. It is gratifying to observe from this table that

TABLE XVI.

PHTHISIS. ENGLAND AND WALES.							
Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period	Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period
1838	59,025	8,996	8,880	1866	55,714	2,602	2,448
1839	59,559	8,989		1867	55,042	2,589	
1840	59,923	8,997		1868	51,423	2,386	
1841	59,592	8,822		1869	52,270	2,352	
1842	59,391	8,746		1870	54,231	2,410	
1843	—	—	8,880	1871	53,876	2,342	2,316
1844	—	—		1872	52,589	2,271	
1845	—	—		1873	51,855	2,194	
1846	—	—		1874	49,879	2,081	
1847	53,817	—		1875	52,948	2,202	
1848	51,663	—	2,851	1876	51,775	2,119	2,040
1849	50,299	—		1877	51,353	2,079	
1850	46,618	2,824		1878	52,856	2,111	
1851	49,166	2,781		1879	51,272	2,021	
1852	50,594	2,826		1880	48,201	1,869	1,880
1853	54,918	3,084	2,603	1881	47,541	1,825	
1854	51,284	2,791		1882	48,715	1,850	
1855	52,290	2,822		1883	50,058	1,880	
1856	48,950	2,601		1884	49,325	1,827	
1857	50,106	2,627	2,564	1885	48,175	1,770	1,685
1858	50,442	2,591		1886	47,872	1,739	
1859	50,149	2,547		1887	44,935	1,615	
1860	51,024	2,564		1888	44,248	1,568	
1861	51,931	2,581		1889	44,738	1,573	
1862	50,962	2,502	2,564	1890	48,866	1,682	1,685
1863	51,072	2,476		1891	46,515	1,599	
1864	53,046	2,541					
1865	53,734	2,541					

during the period in question there has occurred both a relative and absolute diminution in the deaths so recorded. In part this is, no doubt, a matter of nomenclature, but there are independent grounds for thinking that an actual diminution in phthisis mortality has occurred.

Influence of Climate and Season.—Tubercular disease may occur in any climate, and although climatic conditions without doubt exercise a large influence over such malady, it seems probable that this influence has, owing to its having been confused with other factors, been somewhat exaggerated. It has already been pointed out that mere high or low atmospheric temperature is no bar to a considerable prevalence of the disease.

There appears, however, to be a general agreement among observers that, other things being equal, the disease is of a more severe and rapid type in tropical than in temperate climates.

But the fact which seems most clearly indicated by a study of the distribution of phthisis is, that a moist atmosphere with a considerable daily range of temperature is decidedly favourable to its prevalence. How far this is an influence operating directly upon the life-processes of the tubercle bacillus, and how far an influence operating indirectly, by conducing to general susceptibility or to a relaxed and unhealthy condition of the mucous membranes of the recipients, it is difficult to determine.

As regards the influence of season, in this country the phthisis mortality (London) has its annual maximum in March, April, and May, and its minimum in August, September, and October (Buchan and Mitchell).

Influence of Race, Sex, and Age.—No race is exempt from tubercular disease, but the Jews are said to enjoy a relative immunity from it. On the other hand, many coloured races suffer considerably from phthisis, and it has been especially observed that negroes upon changing their natural and primitive habits of life for the conditions associated with civilisation exhibit a very marked susceptibility to it, and this is the more conspicuous if such changed mode of life is accompanied by migration to a temperate climate.

The sex and age distribution of recorded 'phthisis' deaths in England and Wales during the years 1881-90 is shown in Table XVII., compiled from the Registrar-General's Annual Reports.

TABLE XVII.¹

Period	Deaths at all ages			Deaths at subjoined ages									
	Male	Female	Total	0-3 months	3-6 months	6-12 months	Total under 1 year	1-	2-	3-	4-	Total under 5 years	
1881-1890	246,455	237,513	478,968	M. / 170 F. / 103	397 347	556 528	3,985 3,312	2,788 2,728	1,357 1,318	892 941	722 896	9,754 9,195	
			Totals		372	544	1,084	7,247	5,516	2,675	1,833	1,678	18,949

Deaths at subjoined ages—continued												
	5-	10-	15-	20-	25-	35-	45-	55-	65-	75	85 and upwards	
M. /	4,131	5,157	17,595	27,522	59,126	53,952	38,810	21,766	7,578	1,015	42	
F. /	5,371	10,538	24,877	30,275	59,381	44,226	26,103	12,839	4,907	767	49	
	9,502	15,695	42,472	57,804	118,507	98,178	63,913	34,605	12,485	1,782	91	

It will be observed, as regards sex, that during the period in question, at all ages together, the deaths among males exceeded those among females. This also holds good for the first three years of life, and for the age period 35-45 and upwards. For the intervening periods the deaths among females exceeded those among males. It will be observed that in the age period 10-15 years the recorded mortality among females was double that among males.

As regards age, the deaths registered as due to 'phthisis' fell from the first to the fifth year. After the age period 5-10 they increased up to the age period 25-35, subsequently to which it steadily decreased.

Of the other forms of tubercular disease, it appears, according to Dr. Sims Woodhead's cases, that tubercular meningitis is most common between the third and eighth years of life, but that tubercular disease of the mesenteric glands is commonest at an earlier age, namely in the period from one to two and a half years, which, as will be seen later, may be significant from an etiological point of view.

Cause and Mode of Dissemination.—The discovery of the tubercle bacillus, although a matter of the highest importance and of very great scientific interest, by no means exhausts, or even nearly exhausts, the subject of the etiology of tubercular disease. It has already been pointed out, with respect to microbic diseases generally, that although a given microbe con-

¹ All the figures in this table refer to the ten years 1881-90, except those in the age groups 0-3, 3-6, and 6-12 months, which refer only to the years 1888-90.

stitutes the particulate cause, yet various other conditions are also requisite for the production of the phenomena which we recognise as disease. In the case of tuberculosis this is pre-eminently true, and the 'other conditions' must in practice be regarded as having an importance hardly second to the microbe itself.

It is a matter of common knowledge that tubercular disease is conspicuously prone to occur in different members of the same family, and this also through successive generations. Such might, of course, be regarded—especially in view of the microbic nature of the malady—as a consequence of direct or indirect infection, as ordinarily understood, from case to case; and there can be little doubt but that some instances of multiple attacks in the same family are to be thus accounted for—a matter, however, which will be referred to later. But in view of the great frequency with which this disease is seen to 'run in families' (even though the members of such families are separated, and living under different conditions), and of the slight tendency which it exhibits in general to spread by infection, it seems clear enough that heredity is responsible for a considerable share of its prevalence. Whether this influence of heredity is indirect, depending upon the transmission from parent to child of a special susceptibility, or whether it is direct, involving the actual transmission of the tubercle bacillus in the process of gestation, is a matter which, in the present state of knowledge, cannot be definitely answered; but it is quite possible that it operates now and again in both of the ways suggested. The subject has, however, already been dealt with by Dr. Klein in this volume.

Among other conditions influencing tuberculosis, topographical and telluric circumstances, such as elevation, dampness of soil, hold important place; but for this branch of the subject the reader must be referred to Dr. Copeman's article, vol. i. p. 856 of this work. Certain conditions, however, which belong to the class of hygienic, or, more correctly, unhygienic, conditions remain to be considered here. Foremost among these, as regards that common form of tubercular malady, pulmonary phthisis, is defective ventilation. As evidence of the influence of this factor we have, first, the circumstance that phthisis is most common in large cities, and especially in the most densely populated parts of such cities, where overcrowding and imperfect domestic ventilation are commonly at a maximum, and the free movement of air around dwellings at a minimum. Next we have the more definite and convincing evidence as to the close relationship between defective ventilation and phthisis mortality that is afforded by the past history of life in barracks, ships, prisons, and other institutions. Much important evidence on this point will be found in the Report of the Royal Commission on the Sanitary Condition of the Army which was published in 1868. Up to that time the barracks were overcrowded and insufficiently ventilated, and the phthisis mortality among troops was excessively high. Subsequently to the Report of the Commission the allowance of air-space was increased and the ventilation improved, with the result of a very marked diminution in the phthisis mortality.¹ Similar experience is afforded by the health-history of the Royal Navy, and by that of the occupants of

¹ The overcrowding had been greatest among the Foot Guards, and in that branch of the Service the phthisis mortality had been highest. During the ten years 1837-46 it was 11·9 per 1,000 of strength. For the seven years 1864-70 it had been reduced to 2·3. The mean of the phthisis mortality in the Household Cavalry, Cavalry of the Line, Foot Guards, and Infantry was, for the years 1837-46, 7·89 per 1,000 of strength. In the year 1888 the mortality from phthisis in the British Army at home was 1·2 (See Parkes's *Hygiene*, 8th edit., pp. 592-4).

prisons.¹ The same conclusion is also brought out in another way. In summarising the results of an exhaustive inquiry into the excessive mortality from lung disease in certain districts which had been undertaken for the Privy Council by Dr. Greenhow, Sir John Simon remarked :—'*In proportion as the male and female populations are severally attracted to indoor branches of industry, in such proportion, other things being equal, their respective death-rates by lung disease increased.* And there are medical reasons, which need not now be detailed, for assuming the augmented lung disease to be phthisis.'²

But the pernicious effects of defective ventilation which have been so far referred to are due chiefly to the accumulation in the air of the products of respiration (including, doubtless, the tubercle bacillus from phthisical persons); the exhalations from the body; and, to some extent, also to the products of imperfect combustion consequent on defective methods of heating and lighting. Experience, however, has shown that the loading of the atmosphere of mines, factories, and workshops with special kinds of dust produced in different trades is also a potent indirect cause of phthisis, and apparently of true tubercular phthisis.³ So obvious, indeed, was this result that the expression 'miner's,' 'weaver's,' and 'knife-grinder's' consumption long ago came into popular use. The subject was extensively investigated in the continuation of the inquiry by Dr. Greenhow, to which reference has already been made, and much light has since been thrown upon it by Traube, Zenker, Von Ins, Merkel, Theodore Williams, Watson Cheyne, and others. The dusts in question, it appears, upon being inspired, are taken up by the leucocytes and deposited throughout the lung substance, imparting to the lung in time their particular colour, as, for instance, in the case of the black lung of the miner and the red lung of the oxide-of-iron worker. But their ability for harm, *quâ* phthisis, appears to depend largely upon their hardness and angularity. It has long been known that, of the various dusty trades, those of the steel and stone workers are associated with particularly high mortality from lung disease. Coal-miners have, it is true, suffered heavily from phthisis, but it now seems probable that where this has occurred it has rather been due to the general effects of defective ventilation of the mines than to the inhalation of coal dust. When coal-mines are well ventilated, the phthisis mortality among the miners does not appear to be excessive, though the 'carbonisation' of their lungs still occurs. It has, indeed, been held by some observers (as Seltmann and Merkel) that coal dust *per se*, so far from conducing to phthisis, affords some protection against it. On the whole, it would seem that, as a factor in the causation of phthisis, inhaled dust operates in an entirely indirect manner, its operation depending upon its ability to give rise to a

¹ For evidence as to the high mortality from phthisis at Millbank Prison in former times, see Dr. Baly's paper, *Medico-Chir. Trans.*, 1845, xxviii. 118.

As regards foreign prisons, an interesting case is quoted by Parkes. In the defectively ventilated prison of Leopoldstadt, at Vienna, during the years 1834-47 there died of phthisis 51·4 per 1,000 of the prisoners, while in the well-ventilated House of Correction in the same city during the years 1850-54 only 7·9 per 1,000 died. The comparative length of sentences in the two cases was not given, but Parkes considered that 'no correction on this ground, if needed, could account for this discrepancy (Parkes's *Practical Hygiene*, 8th edit., p. 168).

² *Third Report of the Medical Officer of the Privy Council*, 1860, p. 30.

³ Space does not admit of a review of the evidence as to the relation to one another and to tubercular phthisis of the lung ailments associated with different trade processes. The subject is discussed by Pye Smith in Fagge's *Principles and Practice of Medicine*, 2nd edit., vol. ii. p. 239. It is sufficient here to say that the tubercle bacillus has been found in knife-grinder's and potter's phthisis.

catarrhal or mechanically injured condition of the mucous lining of the lungs, and so to favour the entrance and activity of the tubercle bacillus.

The relation of food to the propagation of tuberculosis is a subject of great importance. Deficient and defective food no doubt indirectly predispose to tubercular infection, by leading to impaired vitality. But it is as a possible carrier of the tubercle bacillus that food, in this connection, assumes its special importance. It has already been seen that cattle suffer in considerable numbers from tubercular malady, and although the identity of such malady with human tuberculosis may not be completely established, it is in a very high degree probable that the two are but different manifestations of an identical cause. If such is the case, the danger to man as regards cattle is a double one, for infection may doubtless occur both by the ingestion of milk and flesh. The frequency with which the young children of healthy parents suffer from *tabes mesenterica* is, as Dr. Sims Woodhead has pointed out, very strongly suggestive of infection by milk.

As regards meat, there are good grounds for believing that infection may result in man from the consumption of the imperfectly cooked flesh of animals that have died of tubercular disease, and the important question has arisen as to whether the whole of the flesh of such animals is unfit for human food, or only those parts in which tubercular lesions are manifest. The evidence at present afforded by laboratory experiments on this subject is somewhat conflicting, and the question cannot be said at present to have been definitely solved. The whole matter is now the subject of inquiry by a Royal Commission. Possibly the infectivity of the tissues generally of tuberculous animals, in contradistinction to that of those tissues which are the seat of evident tubercular changes, may depend upon the activity or otherwise of the tubercular ailment at the time of the animal's death. But further evidence on the matter is wanted.

Lastly, reference must be made to the so-called contagiousness of phthisis. This question has been forced to the front during recent years by Koch's demonstration of the microbic character of the malady; and there can hardly be any doubt that phthisis may be spread by infection from case to case, a view which has long been generally held in Italy, Spain, and Portugal, and was advocated by Galen, Morgagni, Dr. Wm. Budd, and others.

Cases in which the disease seems almost certainly to have been transmitted from husbands to wives have been recorded by Dr. Weber¹ and other observers, and the rapidity with which cases of acute phthisis are in practice occasionally seen to succeed one another in members of the same family, but of varying ages, are highly suggestive of infection. On the other hand, the extreme rarity with which the nurses in consumption hospitals are attacked, even though they are exposed to more or less continuous opportunities of infection, seems to indicate that the infective power of the disease is of a very low order, and it has, indeed, been held by some altogether to negative the idea of its infectiousness. The reconciliation of the apparently conflicting evidence is probably to be found in the vast importance, as regards tubercular disease, of the indirect causes, which has been insisted upon above. It is not enough that the tubercle bacillus should be taken into the lungs or alimentary canal—it must also there meet with a favourable soil if it is to give rise to tubercular disease. No amount of negative evidence, therefore, is valid as an objection to the theory of infectiousness. A broad argument for the infectiveness of phthisis is probably to be found in the fact of the increase of the disease during recent years in certain countries, such as Australia and the United States, which have been subject to considerable

¹ *Chin. Soc. Trans.* 1874.

immigration from infected countries, and, in the former case, to considerable immigration of persons affected with the disease. This is quite consistent with a theory of the importation of the disease, and its subsequent spread by infection.

Relation to other Diseases.—It has often been maintained that malarial disease is antagonistic to tuberculosis, but there seems now a considerable doubt on this point. It appears to be a fact, however, that phthisis is seldom seen in the subjects of gout, and, more curiously, it is true that mitral stenosis is almost never found in persons who die of phthisis.

CROUP

Synon.: *Cynanche Trachealis*, *Cynanche Stridula*. Fr. *Le Croup*; Ger. *Häufige Bräune*, *Der Croup*; It. *Laringitide Membranacea*.

The doctrine of croup as a disease *sui generis* seems to have been first especially insisted upon by Dr. Home, of Edinburgh (1765), in his 'Inquiry into the Nature, Causes, and Cure of the Croup.' In 1801, Dr. John Cheyne followed upon the same lines. But cases of what Hirsch considers must have been croup had been described as early as 1576 by Baillon in Paris. During the seventeenth and eighteenth centuries further cases, also regarded by Hirsch as croup, were referred to as 'catarrhus suffocativus' by different observers.¹ In 1821, however, Bretonneau maintained the identity of membranous croup and diphtheria, and thus initiated a controversy which has continued to the present day.

It is only proposed here to refer to a few points which must be taken into consideration in attempting to arrive at a conclusion as to the nature of 'croup,' and its relation to diphtheria.

It was admitted by Bretonneau, and has since been abundantly proved, that an inflammation of the laryngeal mucous membrane set up by irritants such as boiling water may result in an exudation of lymph and the formation of a false membrane indistinguishable to the eye from a diphtheritic membrane. There is, therefore, no *a priori* ground for asserting that a membranous laryngitis must of necessity be a diphtheritic process. Further, there would seem no reason to doubt that an inflammatory condition of the mucous lining of the larynx and trachea may be, and sometimes is, set up by non-diphtheritic causes, among which cold and damp doubtless play an important part, even if bacteriology should ultimately show it to be but an indirect part.

When, however, we come to consider the title of 'croup' to be regarded as a separate and specific disease, or when we consider the alleged identity of croup and diphtheria, other important considerations must be taken into account.

Table XVIII. shows the mortality recorded as due to 'croup' year by year since the commencement of registration.

On reference to the table, it will at once be observed that of late years the number of deaths returned as croup has undergone a decided diminution, both absolutely and relatively to the population. Now this is not the case with the diseases of the respiratory system, in the causation of which cold

¹ Among them, Blair, of London, in his *Observations in the Practice of Physic*, 1718.

and damp unquestionably play an important part; and it would seem, therefore, that the deaths ascribed to croup are by no means all of an ordinary catarrhal character.

TABLE XVIII.

Showing Year by Year the Deaths in England and Wales certified as Due to 'Croup,' and the corresponding Death-rates per Million Living.

ENGLAND							
Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period	Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period
1838	4,463	302	270 ¹	1866	5,168	241	208
1839	4,192	227		1867	4,887	202	
1840	4,336	282		1868	4,491	204	
1841	4,177	268		1869	4,478	202	
1842	4,457	—		1870	4,302	191	
1843	—	—	276	1871	4,116	181	184
1844	—	—		1872	3,640	157	
1845	—	—		1873	4,282	183	
1846	—	—		1874	5,010	211	
1847	3,338	—		1875	4,542	189	
1848	3,777	—	224	1876	4,204	172	154
1849	4,038	—		1877	3,910	158	
1850	4,322	243		1878	4,061	162	
1851	4,180	236		1879	3,574	141	
1852	4,058	227		1880	3,571	138	
1853	3,660	202	276	1881	3,594	138	163
1854	3,998	218		1882	4,609	175	
1855	4,419	239		1883	4,591	172	
1856	5,207	277		1884	4,748	176	
1857	5,279	277		1885	4,235	156	
1858	6,220	319	288	1886	3,685	134	126
1859	5,636	286		1887	3,979	143	
1860	4,380	220		1888	3,682	129	
1861	4,397	219		1889	3,241	114	
1862	5,667	278		1890	3,145	109	
1863	6,957	337	288	1891	2,638	91	126
1864	6,777	324					
1865	5,921	280					

On the assumption that 'croup' is a separate specific disease, the diminished mortality ascribed to it might, of course, be referred to sanitary improvements, or to a natural decline in the malady. Nevertheless, there are grounds for believing it to be due to improved diagnosis; for thinking, in fact, that some of the deaths which in former years would have been returned as 'croup' are now correctly returned as diphtheria.

For that many cases of diphtheria have been, and still are, ascribed to croup is well known. Inquiry very frequently shows that antecedent to, or concurrently with, an increase in the deaths from diphtheria there has also been an increase in the deaths returned as 'croup;' and it also brings to light instances of diphtheritic paralysis following upon attacks of so-called 'croup.' Moreover, Dr. Longstaff's curves, exhibiting the variations year by year (1855-80) in the mortality from different diseases, among them croup and diphtheria, show that 'since 1861 the curves of croup and of diphtheria have been so very similar as to suggest that, at the very least, there is great confusion in the diagnosis of the two diseases, and even to be a weighty argument in favour of those pathologists who maintain their identity.'²

¹ For four years only.

² *Studies in Statistics*, p. 316.

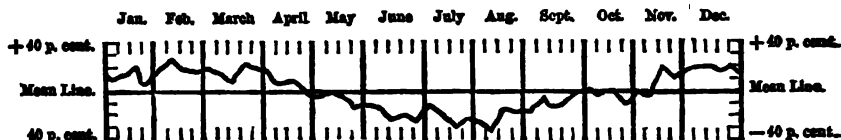


The facts thus indicated seem to make it clear that many of the 'croup' deaths are really due to diphtheria.

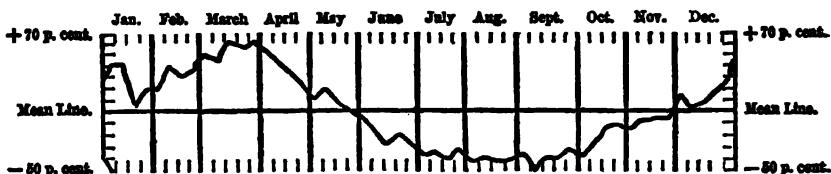
Here, again, however, it must not be concluded that the whole problem is solved. Dr. B. A. Whitelegge has pointed out that the seasonal curve of croup approximates more closely to the laryngitis curve than to that of diphtheria (*see curves below*); that 'croup is more fatal to males than females upon the whole, the mortality among females being lower up to fifteen years of age. The highest mortality in both sexes occurs in the second year of life. In all these respects "croup" is allied to laryngitis rather than diphtheria.'¹

SEASONAL MORTALITY²

Croup, for all Ages and both Sexes.—London Deaths for Thirty Years, 1845-74 .



Laryngitis, for all Ages and both Sexes.—London Deaths for Thirty Years, 1845-74.



The provisional conclusions, then, to which the above considerations seem to point are:—

1. That a considerable number of cases of so-called 'croup' are, without doubt, cases of diphtheria.
2. That this, however, is not true of all such cases.
3. But that those which remain, not being of diphtheritic origin, are probably cases of laryngitis, or tracheitis, of a kind more closely allied etiologically to bronchitis.

PNEUMONIA

Synon.: *Inflammation of the Lungs, Peripneumony. Fr. Pneumonie; Ger. Lungenentzündung; Ital. Pneumonitide.*

In considering pneumonia from the point of view of epidemic disease we are, for the present day, mainly concerned with the form known as acute lobar pneumonia, occurring as a so-called idiopathic affection. It must be pre-mised, however, that this restriction is provisional only, for, as will be seen

¹ *Hygiene and Public Health*, by B. A. Whitelegge, M.D., B.Sc., D.P.H., 2nd edit., p. 301.

² These curves are reproduced, by permission of Dr. Buchan and Sir Arthur Mitchell, from their papers already referred to.

later on, it is not improbable that, in dealing with the subject from this narrowed view-point, we may be including under a common name two or more maladies which, though superficially identical, are due to different causes; while, at the same time, we may be separating from one another, on the ground of clinical or apparent etiological difference, manifestations due to one and the same cause.

Acute lobar pneumonia was for a long time regarded as a non-specific inflammation, dependent usually upon exposure to cold and damp, the fever and general symptoms being considered as secondary to the local inflammatory action. Of late years, however, this view has largely lost ground, and it has become more and more recognised that in its sudden onset, its rapidly attained pyrexia, the definite course of its symptoms, its relatively constant duration, and its sudden defervescence, pneumonia rather exhibits the characters of a specific fever, with inflammation of the lung as its most prominent local manifestation. This latter thesis, moreover, as contrasted with the older one, derives some indirect support from the fact that pneumonia in its distribution in space, as well as in its relation to season and certain particular kinds of weather, exhibits some points of contrast to bronchitis—a disease also, and upon stronger evidence, held to depend upon cold and damp. Then, again, the existence of a specific¹ fever characterised by pneumonia, or, perhaps, of several kinds of such fever, is rendered probable by the frequency with which pneumonia has been observed to occur in epidemics, and by the fact that it is certainly at times infectious. Lastly, bacteriology is leading to the same general conclusion, and bids fair to put the doctrine of pneumonia upon a more satisfactory basis.²

Pneumonia may be traced in the earliest medical writings, though it is only since the introduction of the stethoscope that a definite distinction has been drawn between it and other diseases of the chest. It seems likely that in former times pneumonia was more prone to occur in epidemic form than is the case in the present day. At all events, there are not a few records of its wide prevalence in the sixteenth, seventeenth, and eighteenth centuries in Italy, Germany, France, and especially in Switzerland, where it has long been known as 'Alpenstich.' The earliest records of epidemic pneumonia in England and North America seem to date from the eighteenth century.

During the present century an extensive pandemic diffusion of pneumonia occurred in North America from the years 1812 to 1825, as well as various other epidemics in the same country, both during the earlier and more recent years of the century. In 1858–55 pneumonia was epidemic at Julianahab, in South Greenland. As regards Europe, a number of epidemics have, during the present century, been recorded for Italy, France, Germany, Switzerland, and Great Britain, a few in Norway and Denmark, and one in Iceland (1868). Speaking generally, however, the European epidemics of the present century, as compared with those of previous centuries, have, according to Hirsch, been mostly far apart and confined within narrow limits.³ Two epidemics are recorded for India (Punjab), in 1875 and 1892. The most recent epidemics of importance in this country occurred at Middlesbrough in 1888, and at Scotter (Lincolnshire) in 1890.⁴

¹ The word *specific* is, of course, used here only in the relative sense which is consistent with the views expressed in the introductory section.

² For the bacteriological evidence so far available see p. 123, *ante*.

³ For fuller information as regards epidemic pneumonia see Hirsch, *ibid.* vol. iii. p. 125.

⁴ See Reports by Drs. Ballard and Parsons respectively, in the *Annual Reports of the Medical Officer to the Local Government Board for 1888 and 1890*.

Pneumonia, like bronchitis, occurs more or less extensively over nearly the whole world, though certain localities, notably Lower Egypt and Tunis, seem to enjoy a marked relative immunity from it. As already mentioned, however, there is not the close correspondence between the distribution of pneumonia and bronchitis which would be expected upon the hypothesis of their being due to one and the same cause, such as exposure to cold and damp. Thus, the prevalence of bronchitis appears to increase towards the northern latitudes—Iceland, the Farøe Islands, Sweden and Norway, being among its principal seats. This is not the case with pneumonia, which does not seem to be particularly prevalent in the countries named; occurring there, indeed, according to Hirsch, only to a moderate extent. Particular countries in other latitudes might also be cited in which the distribution of these diseases does not closely correspond. As regards England, it has been pointed out by Dr. Longstaff¹ (for the period 1876–80) that pneumonia was comparatively much more fatal than bronchitis in some registration counties, whereas in other registration counties the opposite was the case.

Nevertheless, there can be no doubt that the amount of both pneumonia and bronchitis is largely influenced by cold, but such influence is *greater in the case of bronchitis than in that of pneumonia*. This is brought out, among other ways, by a study of the seasonal mortality of the two diseases. As regards both, the London weekly deaths are above the mean from November to April, and below it from May to October. The pneumonia deaths, however, rise above the mean about a week earlier (last week in October), and fall below it about a fortnight later (first week in May), than the bronchitis deaths. The absolute maximum of pneumonia deaths is in December, that of bronchitis deaths in January. The absolute minimum of both is in August. But the rise and fall in the deaths from both these diseases, above and below the mean, which correspond broadly in point of time to the colder and warmer seasons of the year, are more conspicuous in the case of bronchitis than in that of pneumonia.

But in addition to the general agreement (though subject to differences of detail) exhibited by the seasonal, or week-to-week, mortality of bronchitis and pneumonia, there is a decided general agreement between the extent of mortality of these diseases in different years, and this also appears to have a relation to temperature. Dr. Longstaff, in the paper to which reference has just been made, has compared the curves indicating the mortality of these diseases from year to year (London deaths, 1850–82) with one another, and also with a curve representing the number of cold days at Greenwich in each year. The result obtained is that the year-to-year mortality curves of bronchitis and pneumonia correspond closely in direction and both bear a general resemblance to the 'cold-day' curve; but here again, as in the seasonal curves, the fluctuations are greater in the bronchitis curve than in that of pneumonia.

The general result of the considerations above put forward, independently of the results of bacteriological research, is that, although cold is doubtless an important indirect or predisposing cause of pneumonia, it is not the ultimate cause.

Further analytical study of the influence exerted upon bronchitis and pneumonia mortality by cold shows that this influence is dependent, not alone upon the mere degree of coldness of the atmosphere, but largely also upon sudden and frequent fluctuations of temperature. This seems to be brought out clearly by the observations of many observers, such as Huss for

¹ 'Phthisis, Bronchitis, and Pneumonia,' by G. B. Longstaff, M.A., M.D. (*Epidem. Soc. Trans.*, 1883, vol. ii. N.S.). Also reprinted in *Studies in Statistics*.

Sweden, Eschbaum for Bonn, Schramm, and also Klinger, for Bavaria, Chansaux for Paris, and Gordon for India. Several Continental observers have ascribed an important influence in particular epidemics to relative absence of rain and lowness of the subsoil water. At Middlesbrough, Dr. Ballard noted that the epidemic made progress during the weeks of drought, but was apparently held in check by abundant rain.

It has already been suggested that the cases commonly grouped together under the head of lobar pneumonia may be instances of two or more different diseases. Bacteriological evidence, so far, is consistent with this view. In the Middlesbrough cases, for instance, Dr. Klein failed to identify either the micrococcus of Friedländer or the diplococcus of Fränkel and Weichselbaum, but he found a short and distinctive bacillus, which apparently stood in causal relation to the malady. For epidemiological suggestions on the point we have the fact of the differences in type of pneumonia from time to time, and the differences in its infectiousness. As regards type, it is to be noted that pneumonia occurring in epidemic form has very commonly exhibited a typhoid character, with somewhat prominent gastric and intestinal symptoms, and a high mortality. This might, perhaps, indicate a class distinction between the pneumonia of such epidemics and that which occurs sporadically. Again, suggestion of the spread of the disease by direct infection is frequently met with in the literature of pneumonia, and there are particular cases on record which seem to show undoubtedly that the malady does at times spread by infection. Of such particular instances may be mentioned some cases which occurred in 1876, and were reported by Dr. Patchett in the '*Lancet*' for 1882, and a series of cases in 1879, reported by Dr. F. H. Daly in the '*Lancet*' for November 12, 1881. Also cases recorded by Mr. Wynter Blyth in his '*Manual of Public Health*,' page 415, and by Dr. Ballard in his Middlesbrough report.

The variations, then, in the property of infectiousness apparently attaching to pneumonia at different times and places are, like the variations in type, consistent with the view of there being different kinds of pneumonia. Differences in the age incidence, which will be referred to later, may also possibly bear a like interpretation, and the subject is well deserving of very careful inquiry. It must, however, be remembered in such inquiry that variations in type, infectiousness, and age incidence, do not necessarily imply specific distinctions in the ailments exhibiting them. They may all be thought of as possibly representing differences in the virulence of one and the same contagium vivum. That this may be so as regards differences of type is matter of common knowledge; and there are reasons for thinking that it may likewise be so as regards infectiousness and age incidence. It is possible therefore that ordinary sporadic lobar pneumonia is capable, under conditions especially favourable to the pathogenicity of its contagium vivum, of passing from time to time into an epidemic form of a typhoid character, with a high fatality and an appreciable degree of infectiousness.

No race is exempt from lobar pneumonia, but many coloured races, particularly negroes, are especially susceptible to it. As regards sex, the mortality at all ages together, according to Dr. Longstaff, is greater for males than females, almost in the proportion of three to two. The disparity is most marked at ages 35-65, when males suffer more than females, in the proportion of two to one. It must not, however, be concluded that, case for case, pneumonia is more *fatal* to males than females. As a general rule the reverse is the fact.¹ According to Huss's statistics, the relative

¹ See article on 'Pneumonia,' by the late Dr. Wilson Fox, in Reynolds's *System of Medicine*, 1st edit. p. 689.

fatality among the two sexes, given an equal number of attacks for each, is males ten, females fourteen. It would thus appear that more males die of pneumonia than females because many more are attacked—that, in other words, the liability to attack is much greater in males, but the liability to death if attacked is greater in females.

With respect to age, the recorded mortality is highest at the extremes of life, being, however, about three times as great in the first year of life as in old age. It falls very rapidly through the first three years, and more gradually to the thirteenth year, when it attains its minimum. Subsequently it rises steadily throughout the remainder of life. The fatality, or case mortality, in relation to age, differs from the mortality in that it is greatest in advanced life. Except, perhaps, for the first year or two of life, it is low during childhood and adolescence, but after about the thirtieth year it increases steadily with advancing years.

Of the general fatality of pneumonia at all ages together it is difficult to speak definitely, since this varies considerably in different times and places. Among Huss's cases (2,618 in number) it was 10 per cent. of the attacks. By other observers it has been found to range considerably above and below this.

The Middlesbrough epidemic pneumonia differed from that of ordinary experience, among other things, in having an especially high fatality (21 per cent.); in that its fatality at all ages together was greater among males than females; and that its incidence upon age, as judged by the number of deaths, in relation to the numbers living at each age period, was greater upon the middle and higher ages than is usual.

Among predisposing causes of pneumonia, other than those which have been referred to, must be mentioned unwholesome conditions of life generally. The records of pneumonia epidemics seem to leave no doubt that overcrowding, defective ventilation, emanations from sewers, filth accumulations, and the like, have had important influence upon the prevalence and type of the disease. As evidence of this we have, as Hirsch says, 'not only the epidemic outbreak and prevalence of pneumonia in confined and circumscribed buildings—barracks, prisons, and such-like—where the factor in question has been peculiarly noticeable at the time of the outbreak and so long as it lasted, but also the fact that in a number of epidemics which have spread over whole villages, those streets or houses have suffered most that were principally exposed to the particular harmful influences.'

In the epidemic at Middlesbrough, Dr. Ballard found good reason for attributing considerable influence to defective drainage as an agency in the incidence of the disease.

As regards influences special to the individual, fatigue and debility predispose both to attack and death. Persons whose constitutions are damaged by the abuse of alcohol are well known to be particularly liable to die of pneumonia.

The not uncommon occurrence of pneumonia in association with other diseases, notably enteric fever, malaria, and influenza, raises the question as to the relationship of the pneumonia in such cases to the diseases in question. Is it, for example, that in certain instances, and owing to some relatively unusual conditions, the enteric fever virus, instead of selecting the intestines for its chief local operations, attacks also, and perhaps most conspicuously, the lungs? Or, again, is it that the diseases in question predispose to independent attack by pneumonia? As a third conceivable hypothesis, it might be suggested that the cases under consideration are examples of hybridism. It is impossible at present to find definite answers to these questions. Perhaps the phenomena referred to may in some cases be due to one, and in some to another, of the causes suggested.

A question allied to those above raised presents itself in connection with the particular outbreaks of pneumonia in Middlesbrough and Scotter. They were apparently outbreaks of one and the same disease, and a disease, moreover, which in certain particulars differed from pneumonia as ordinarily met with. Now the fact, speaking broadly, that these unusual epidemics of pneumonia had some sort of time relation to the pandemic diffusion of influenza throughout the world might suggest a relationship between the two diseases—that the pneumonia of Middlesbrough and Scotter was, in fact, an expression of influenza. Against this view it might be urged that the pneumonia at Middlesbrough was epidemic more than a year before the European prevalence of influenza occurred; but with respect to this there is the fact that, as early as February 1887 an epidemic of an unknown infectious disease had occurred at Northallerton, in Yorkshire, and had been considered by the late Dr. Page, who investigated it for the Local Government Board, as allied to, if not actually identical with, epidemic influenza. Another objection to the thesis, however, is found in the fact that the pneumonia which occurred at Middlesbrough in 1888, although unusually abundant in quantity, seems in some respects, at least, to have resembled in character the pneumonia met with in that particular locality in previous years. On the other hand, it must be noted that in the incidence of its mortality upon age the Middlesbrough pneumonia exhibited a greater resemblance to influenza than to the usual pneumonia of this country; and lastly, it is somewhat suggestive to note that in two out of five cases of croupous pneumonia *following influenza*, Dr. Klein¹ has found 'in large numbers, almost in pure culture,' the particular micro-organism which he identified as the cause of the Middlesbrough pneumonia.

YELLOW FEVER

Synon. : Fr. *Fièvre jaune*; Ger. *Gelbes Fieber*; It. *Febbre gialla*.

History and Geographical Distribution.—Yellow fever is endemic only in certain comparatively limited areas of the earth's surface, whence it from time to time extends in epidemic and even pandemic fashion—thus, as in many other respects (though on a different scale), strikingly resembling cholera. Its two principal endemic centres are: (1) The coast of the Gulf of Mexico and the West India Islands; and (2), a limited portion of the West Coast of Africa, notably Sierra Leone. Whether the disease originated independently at these two centres, or whether, as seems more probable, both *a priori* and in view of the recorded behaviour of the disease in more modern times, at one only of them, being subsequently carried to the other, it is impossible to decide definitely from the historical data available. The difficulties of an inquiry into the point are, moreover, increased by the fact that the severe forms of malarial sickness have very frequently been mistaken for yellow fever. And although there is suggestion that yellow fever was prevalent among the natives on the shores of the Gulf of Mexico prior to the discovery of America, it is impossible to affirm this with confidence. But on the whole, having in view the fact that the first *reliable* accounts of the disease in Africa are of a considerably later date than those in the West

¹ Page 128, *ante*.

Indies, it seems likely, as Hirsch says, that the West Coast of Africa 'was infected from the Antilles, afterwards becoming an endemic focus when the disease got naturalised,' and that in the West Indies we have the headquarters and original home of the disease. Nevertheless, yellow fever apparently existed on African soil before the first African epidemic of which we have record, since this epidemic, which occurred at St. Louis (Senegal), is said to have been traced to importation from Sierra Leone.

In any case, the first unequivocal accounts of yellow fever in any country come from the West Indies towards the middle of the seventeenth century, when the disease was very fatal in Guadeloupe, appearing about the same time at Barbadoes and Cuba. The first known epidemic at Vera Cruz, the principal seat of the disease on the Mexican coast, seems to have occurred in the year 1699. On the West Coast of Africa, the first recorded outbreak was that already referred to at St. Louis, and occurred in 1778. Subsequently to the dates given the disease has been very frequently prevalent on the Mexican coast and the West India Islands, and on the West Coast of Africa. The particular localities in those endemic centres at which the most frequent prevalences have occurred are as follow:—*Mexican Coast*—Vera Cruz and Tampico; *West Indies*—Martinique, San Domingo, Jamaica, Guadeloupe, Antigua, Cuba, Santa Cruz, and St. Thomas; *West Coast of Africa*—Sierra Leone and Senegambia. During and since the eighteenth century yellow fever has also frequently extended up the east coast of North America, the highest points reached being apparently Halifax (44° 39' North) and Quebec (46° 50' North). It first appeared in New York in 1693, and has since been more or less prevalent there on some twenty-one occasions, the last of which was in 1870.

The places in the United States, in addition to New York, which have suffered the most frequent visitations are New Orleans, Philadelphia, Charleston, Mobile, Pensacola, Norfolk, and Baltimore. In New Orleans, judging from the mortality table given by Dr. Sternberg,¹ the disease was almost continuously present from 1817 to 1858. But with respect to yellow fever in the United States Dr. Sternberg remarks that, 'although it has occasionally prevailed as an epidemic in every one of our seaport cities as far north as Boston, and in the Mississippi Valley as far north as St. Louis, it has never established itself as an epidemic [? endemic] disease within the limits of the United States.'

The most important epidemics in the United States were the following:—1793, in Philadelphia (deaths 4,040, said to have equalled 10 per cent. of the population); 1797, in Philadelphia (deaths 1,800); 1798, Philadelphia and other places, including New York (deaths in Philadelphia, 3,645, said to have been 80 per cent. of attacks; in New York, 2,080); 1853, States of Florida, Alabama, Louisiana, Mississippi, Arkansas, and Texas (deaths in New Orleans, Louisiana, 7,970); 1867, States of Texas and Louisiana (deaths in New Orleans, 8,098; in Galveston, Texas, 1,150); 1878, States of Florida, Alabama, Mississippi, Louisiana, and Texas; 1878, 'the last and most extended epidemic' in the United States (Sternberg), invaded 182 towns, chiefly in Louisiana, Tennessee, Alabama, and Mississippi (deaths 15,984, attacks 74,000). Several of these outbreaks were clearly traced to importation from one or other of the endemic centres of yellow fever, and there can be little doubt that they were, in fact, all due to this cause.

As regards South America, Hirsch considers that prior to the year 1850 there are only records of two undoubted epidemics, both of which occurred

¹ *Report on the Etiology and Prevention of Yellow Fever*, by George M. Sternberg, Lieut.-Col. and Surgeon, U.S. Army, p. 44.

at Guayaquil, the first in 1740, and the second in 1842, and both of which are said to have been due to importation. In October 1849, however, yellow fever was imported into Bahia, and soon spread to Rio Janeiro, Pernambuco, and other places. Since that time Brazil has, apparently, seldom been free from the disease, and the question arises whether it has not become truly endemic there. In 1854 the disease was carried to Peru, and in the next few years it spread to many towns on the coast. Chili has so far been exempt (Hirsch).

In addition to its distribution in America and Africa, yellow fever has in not a few instances been imported into Europe, but except in the Iberian peninsula it has never attained any large epidemic proportions. During the several epidemics which occurred in Spain in the eighteenth century, the disease was for the most part limited to Cadiz, but in the years 1800–1804, when it again prevailed in Cadiz, it spread largely over Andalusia, extending to the seaboard of Murcia, Valencia, and Catalonia. In 1810 it broke out once more in Cadiz, Cartagena, and Gibraltar, and 'in the two following years it appeared at the same places anew, and from them it again spread through several of the coast towns of Granada, Murcia, and Valencia.' From 1819 to 1821 it was a third time epidemic in the provinces of Andalusia, Murcia, and Catalonia. Since 1821 it has four times (1823, 1828, 1870, and 1878) been imported into Spain, but has not attained any great prevalence. In Portugal there was an epidemic in 1723, and another in 1856 (120 cases, 53 deaths—Hirsch), besides several other importations which did not result in any notable spread of the disease. In 1804 and 1821 the disease was carried from Barcelona to Majorca. In 1804 it was also imported into Leghorn from Cadiz. Cases have at different times been brought to several other European ports, but except as regards Brest (1856), Saint-Nazaire (1861), and Swansea (1865), no epidemic has followed. In Swansea, where the disease was clearly brought by a vessel from Cuba, some twenty-six cases, fourteen of which ended fatally, occurred among the residents of the locality,¹ but, it should be noted, exclusively among persons who either resided in, or were taken by their business relations into, the neighbourhood of the infected vessel.

The absolute limits of yellow fever distribution, according to present experience, are—for the Western Hemisphere, 84° 54' south latitude (Montevideo) and 46° 50' north (Quebec); for the Eastern Hemisphere, 8° 48' south (Ascension) and 51° 37' north (Swansea). In epidemic form, however, its northern limits have been 48° 4' (Portsmouth, N. Hampshire), and 48° 34' (Leghorn) in the Western and Eastern Hemispheres respectively.

Influence of Climate and Season.—Yellow fever is undoubtedly, to a large extent, controlled by climate and season. This is evident from its geographical distribution, and from the fact of its being most prevalent in the hot season of the year. Nevertheless, although heat is necessary for the development of an epidemic, the disease, when once well established, does sometimes persist in spite of cool weather, though it is invariably arrested by frost. But while it is always arrested by frost, its cause is not necessarily thereby destroyed, but may, like that of cholera, survive the winter, and give rise to a fresh epidemic on the return of hot weather. In the case of infected ships, too, it has on different occasions been observed that although the disease has died out as the higher latitudes have been reached, it has reappeared on the return of the same ships to warmer climates.

With regard to the influence of humidity, the evidence for different places

¹ See Sir George Buchanan's Report: *Eighth Report of the Medical Officer to the Privy Council*, 1865. The twenty-six cases include six doubtful ones.

is somewhat conflicting. According to Hirsch, a high degree of atmospheric moisture is generally favourable to yellow-fever prevalence. It has been stated by some observers, however, that in particular localities (the coast of Guiana and Guadeloupe) dry weather favours the prevalence of the disease.

Fatality.—The fatality of yellow fever varies largely in different epidemics. This is due to several causes, and in part, no doubt, to variations in the malignancy of the virus. It depends, however, also in part upon race, as will be seen immediately, and particularly upon the degree of resistance primarily acquired, as a result of acclimatisation, by those attacked. Among unacclimatised adults, the fatality ranges from 20 to 60, or even 80, per cent.

Influence of Race.—No race is entirely exempt from yellow fever, but there is no doubt that negroes are decidedly less susceptible than the white races. They are less liable both to attack and to death in the event of attack. It is further said that, broadly, the susceptibility of different people is inversely as the temperature of their native climate.

Influence of Sex.—Both the attacks and deaths are more numerous among males than females. This is, no doubt, largely owing to the greater exposure of males and to their habits—especially over-indulgence in alcohol. Whether, apart from these factors, males are actually more liable to attack and death by yellow fever than females is doubtful.

Influence of Age.—In localities in which the disease is endemic the majority of observed cases occur among persons in middle life, attacks among children and old persons being said to be relatively few. In its bearing upon the age incidence of the disease this circumstance is, however, perhaps somewhat misleading. During epidemics in endemic localities visitors, who have not yet become acclimatised, form a large proportion of the cases, and such visitors no doubt consist largely of adult males. And further, it appears that in localities in which yellow fever is not actually endemic, but occurs in occasional epidemics, large numbers of children are attacked—for, unlike the children in endemic areas, they are not acclimatised. Even in New Orleans, it seems that during epidemic periods children die in large numbers. In Table XIX.¹ Dr. Bemiss has shown the age distribution of 905 cases which occurred in that city during the epidemic of 1878. The age distribution of the deaths and the fatality are also shown.

TABLE XIX.

Age	Cases	Deaths	Per cent.
Under 5 years of age	206	26	12·67
From 5 to 10 years of age	233	20	8·61
From 10 to 20 years of age	183	9	4·9
From 20 to 40 years of age	232	39	16·7
From 40 to 60 years of age	47	6	12·7
From 60 to 80 years of age	4	2	50·0

Protection.—One attack of yellow fever usually confers immunity, though second attacks, apparently, sometimes occur. But apart from any recognised attack of the disease, there is no doubt that a large degree of immunity may be acquired by long residence in a yellow-fever country. With respect to this Dr. Sternberg remarks: 'It is a remarkable fact that the population of a large city like Havana, or Rio Janeiro, in which yellow fever has been endemic for a series of years, enjoys such a degree of immunity from the effects of the deadly poison that there is no interruption of business or pleasure at a time when strangers in the city are falling sick on every side.

¹ Taken from Dr. Sternberg's Report.

The development of an *epidemic* in these cities depends [? partly] upon the presence of susceptible strangers in sufficient number to furnish a series of cases considered large enough to justify the use of the word. . . . Under exceptional circumstances, however, epidemics are developed in these endemic foci of the disease, in which those who, by birth or long residence, were supposed to be acclimatised furnish a certain quota to the general mortality.' This is of interest as illustrating the fact that the infectivity of a given disease is not a fixed quantity, and that, therefore, a degree of immunity sufficient to be proof against infection during one epidemic may not prevent attack during another epidemic of the same disease. Hirsch, also, remarks that the 'peculiar immunity [of the negro] proves "insufficient" in severe epidemics of yellow fever.'

But it is certain that those habitually residing in yellow fever localities enjoy a large relative immunity, and the question arises whether such immunity is transmissible by heredity, or whether it is entirely acquired by each individual for himself. Many authorities have advocated the hereditary theory, and it seems possible that immunity is transmissible. On the other hand, it is more likely, as Dr. Sternberg says, that 'the creole child owes his immunity not to his parents, but to individual acclimatisation, and not unfrequently, to say the least, to a mild, unrecognised attack of yellow fever.' In support of this view Dr. Sternberg quotes Dr. Dowler to the effect that 'many creole children had, during the epidemic of 1858, a fever, a slight fever, yellow fever if you please, known as such rather by the co-existence of the epidemic than from any severe symptoms among these children, a slight fever never yet described, having generally but one paroxysm, lasting from six hours to one, two, or three days, scarcely ever requiring medication. That a few of these cases acquired an alarming violence, and even proved fatal, is most true, most deplorable.' Lastly, it is clear that the immunity due to 'acclimatisation' may be largely lost by residence outside the yellow fever zone, and this seems true also of the pronounced immunity of the (African born) negro race. Further, the immunity gained by residence in a particular yellow fever locality appears to be, to some extent at least, special to that locality. Thus, it has been pointed out by Humboldt and others that the natives of Vera Cruz, who remain exempt while at home, are liable to die of yellow fever if they migrate to Havana, or some other place in which yellow fever is endemic. According to Cornuel¹ also, if two bodies of troops stationed at different points in the Antilles exchange garrisons, yellow fever is apt to break out among both, though other troops who have remained in the same garrisons remain unaffected.

The most efficient and permanent immunity is, without doubt, that which results from a well-marked attack of yellow fever, though even this immunity has been alleged by some observers to be lost by long absence from endemic localities.

Cause and Mode of Dissemination.—Judging both from its clinical features and epidemiological behaviour, there are strong grounds for believing yellow fever to be a microbic disease, but as yet the particular micro-organism upon which it may be presumed to depend has apparently not been identified. Several observers, notably Dr. Domingos Freire, of Brazil, and Dr. Carmona y Valle, of Mexico, have described different micro-organisms which they regard as standing in causal relation to the disease; but Dr. Sternberg, who has specially studied the matter on behalf of the United States Government, and whose report has been already referred to, believes that he has been able to exclude, in a definite manner, each of these several micro-organisms.

¹ *Annal. Marit.* 1844, ii. 739.

It has already been seen that yellow fever is endemic only in certain localities, and the evidence seems to show conclusively that when it has occurred elsewhere its occurrence has been due to importation. It is true, no doubt, that in particular instances the disease, having been imported into previously uninfected places, has, notwithstanding that it has died down during the cold weather, survived the winter, to reappear, independently of any fresh importation, the following year. And, further, where the conditions are favourable, the importation of the disease may perhaps lead to the establishment of a fresh endemic centre; this has possibly occurred during the latter half of the present century in Brazil. But apart from such resuscitations and the establishment of new endemic foci—themselves, in the first instance, referable to importation—the occurrence of the disease elsewhere than in the Gulf of Mexico, the West Indies, and the Guinea Coast has practically, with few exceptions, been definitely traced to the arrival of persons or things from one or other of the localities last named. There is thus, so far, no evidence of the latter-day *de novo* origin of the disease, even in the senses indicated in the introductory section to this article.

And if the behaviour of the disease is studied in more detail, the same general result is brought out. 'An analysis of any yellow fever epidemic,' says Hirsch, 'shows certain groups of cases so arranged as to constitute separate foci of disease, sometimes in single houses, sometimes in blocks of houses, or, again, in streets or groups of streets; so that each new case of disease, as it occurs, may be traced to infection of the individual within any one such focus. That case may, in its turn, become the centre of a new focus. . . .' And in this way the extensions of yellow fever, once it has been introduced into non-endemic localities, may frequently be traced back to the original introduction.

There can be little doubt, then, that in a sense at least yellow fever is a communicable disease—a conclusion now accepted by almost all observers, including a board of American experts who investigated the epidemic of 1878, and who reported that 'the most frequent agency in the dissemination of yellow fever from place to place is found in yellow fever patients. . . .'

But strong as is the evidence upon which this conclusion is based, evidence equally strong is forthcoming to show that the communicability of yellow fever differs widely from that of small-pox, typhus, and other typically infectious maladies, and that in certain important respects yellow fever more resembles, in the matter of communicability, enteric fever and cholera. It is a matter of very general experience, for instance, that those in close attendance upon the sick do not specially contract the disease. 'It is well known to the people of the City of Mexico that a visit to the sea-coast city of Vera Cruz during the epidemic season is likely to result in an attack of yellow fever. It is also well established that those who fall sick with the disease after their return to the City of Mexico never communicate it to others who are closely associated with them as attendants, &c. . . . This is also the experience of the physicians in charge of hospitals—e.g. the Charity Hospital of New Orleans. So long as the hospital and its vicinity remain uninfected, cases do not originate in the hospital, although yellow-fever patients may be admitted to the wards with unacclimatised persons suffering with other diseases, and be cared for by susceptible attendants.'¹

Similar testimony is given by Lawson for Sierra Leone. As regards the epidemic at Swansea in 1865, Sir George Buchanan points out in his report that 'persons [who had been] exposed to the fever-producing influences about the docks lay sick of yellow fever in various parts of the town . . .

¹ Sternberg, *op. cit.*, p. 57.

yet in no single instance out of all these did any person (whose business did not lead them to the infected neighbourhood of the docks) get yellow fever or any disease at all simulating it.'

Considering all the facts, then, it appears clear that the yellow fever patient does not usually at any rate directly infect others, but that he nevertheless gives off, in some way or other, probably with his discharges, the virus of the disease, and that this, if it should meet with suitable conditions, is capable of infecting the particular locality, and of thus, indirectly, giving rise to the disease in other persons. Outside the body the microphyte no doubt finds a habitat in the soil, and it has been observed by Thomas, Barton, and others, that breaking up the soil for the purpose of cutting canals, &c., in yellow fever countries has apparently given rise to outbreaks of the disease. It is also notable that the virus of yellow fever displays a special ability to establish itself in ships and dwellings. Epidemics on shipboard occupy a conspicuous place in the history of the disease; and that such epidemics have been due to the fact of the ships themselves becoming infected, and not to the direct transmission of the disease from case to case, is evident from the circumstances of the epidemics in question. Such epidemics, for instance, have frequently been limited to particular parts of the ship, as a single cabin, or one side of the vessel; and it has often been noticed that while the seamen have suffered, the officers and passengers have escaped. As regards the tenacity with which the disease clings to dwellings, Guyon, as a result of Lisbon experience, remarked of the infected house: '*whether there are still sick persons in it, or whether there are no longer any, that house will become a centre to reproduce the disease in the strangers who enter it; and what is true of a whole house applies also to a part of it.*'¹ Many observers, according to Hirsch, have reported to the same effect from North and South America, the West Indies, and Mexico.

It has been seen that the cause of yellow fever may be conveyed from place to place by the sick. But it may, too, be transported by fomites. In not a few instances an outbreak of the disease has followed the arrival of ships which, although coming from infected places, have apparently had no actual sickness on board, either at the time of their arrival or during the course of the voyage. Similarly, outbreaks in towns have followed the arrival of apparently healthy people from infected localities. A notable instance of this occurred at Madrid in 1878, when the persons first attacked were those who were closely associated with some soldiers who are said to have been themselves in good health, but who had recently come from Cuba, bringing with them their baggage.

There are, however, certain apparently conflicting facts with respect to the spread of yellow fever by the agency of infected persons and things which require a further hypothesis for their reconciliation. On the one hand, it is maintained that a single imported case may act as a focus for the spread of the disease, and that infected baggage from the yellow fever zone may also set up the disease elsewhere. On the other hand, it is said that 'even the most intimate kinds of contact, such as the healthy and the sick sleeping in one bed, the attendance of physicians and nurses upon the sick, the use of the uncleansed linen, clothes, or beds of yellow-fever patients, *post-mortem* examinations of their bodies, and the like, . . .'² have usually failed to spread the disease. In respect to this seeming paradox it has to be remembered that it is probably not every case of yellow fever that is capable of reproducing its kind. Moreover, it may be that the yellow fever microphyte is infective in certain phases and not in others, and particularly that, as has

¹ Hirsch, *op. cit.*, vol. i. p. 876.

² *Ibid.*, *op. cit.*, vol. i. p. 872.

been often suggested with regard to cholera, it is not infective immediately after leaving the patient, but requires time to undergo some functional modification outside the human body. This is rendered probable from the experience that the 'first cases of local origin in an epidemic do not, as a rule, occur until some time has elapsed after the arrival of the infected ship or fomites or sick person responsible for the introduction of the "germ."'¹

In addition to the conveyance of the yellow fever germ by persons and things, instances of the spread of the disease from one ship to another, or from the shore to the ship, or the ship to the shore, have often been brought forward as evidence of the carriage of the germ for considerable distances by the wind. But it is by no means clear that in the instances in question indirect human agency has been sufficiently excluded. For the present, therefore, the question must be left open, the probability, based upon the general behaviour of the disease, being against the transport of the virus to any distance by winds.

There is at present no evidence that yellow fever is spread by infected water or milk. The absence of evidence on these points, however, must not be taken as excluding the agencies in question, and, judging from the analogy of enteric fever and cholera, there is, as Parkes observes, '*a priori* probability that the cause is swallowed also in this case, and that it may possibly enter with the drinking-water.'

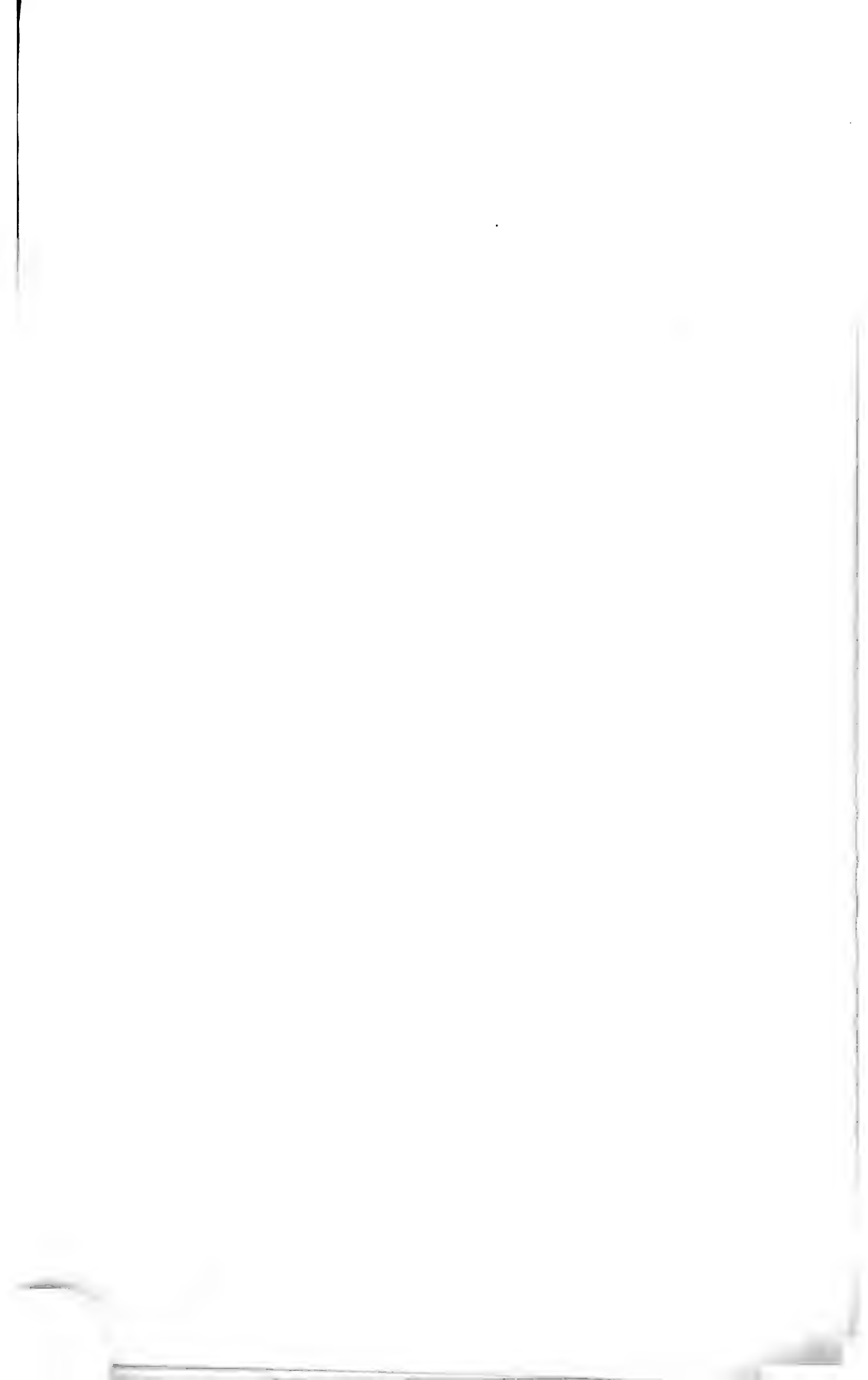
As regards the general conditions which favour the prevalence of yellow fever, heat no doubt occupies a foremost place. This, however, has already been considered. The influence of humidity has also been referred to. Another factor of importance is proximity to the sea. Yellow fever is essentially a disease of coast districts and the banks of great navigable rivers, particularly the former, and it seems only when the disease acquires a high epidemicity that it penetrates inland, its tendency to spread inland, for instance, being apparently much less than that of cholera. Altitude is also a factor which influences adversely yellow fever prevalence. In certain particular instances it is true that it has occurred in localities having considerable elevation. Thus, it has once or twice prevailed at Camp Jacob, in Guadeloupe (elevation 1,800 feet), and at Newcastle, in Jamaica (4,000 feet). But this is exceptional, and as a general rule it is a disease of the low-lying country. It does not appear, however, that it is especially prone to occur in malarious districts, as has so frequently been alleged—an allegation probably due to the frequency with which the severe forms of malarial disease have been mistaken for yellow fever. The latter disease has, in fact, often prevailed extensively in neighbourhoods relatively free from malaria; while, on the other hand, there are malarious districts in the yellow fever zone which are almost entirely free from yellow fever. Extremely strong arguments against the malarial nature of yellow fever are also found in the fact that yellow fever is almost entirely a disease of the towns, and that it occurs freely on board ship. On the whole, it would not appear that the disease is largely, if at all, influenced by the particular kind of soil. Insanitary conditions, however, seem to be of real importance. The 'places where the causes of the disease principally prevail are,' according to Bone, 'the vicinity of foul drains, the banks and channels of rivers which are dry at certain periods, the leeward openings of gullies, and crowded and ill-ventilated rooms, and ships with foul holds.' And there is a mass of evidence to the same effect from other observers.

Among the predisposing causes special to the individual, excessive fatigue, undue exposure to the sun, mental depression, and the debility following upon a debauch, are probably among the most important. As regards the

¹ Sternberg, *op. cit.*, p. 58.

last, Dr. Sternberg remarks that 'sailors who go on shore at an infected port for "a little spree" very commonly turn up in the hospital, or are taken sick after they come on board ship. . . .' Constipation and plethora have been also mentioned as predisposing conditions.

Period of Incubation.—This is usually short, ranging between twenty-four hours and four or five days. According to some observers, however, it may extend to fourteen days, or, exceptionally, even to five or six weeks. Such exceptionally long periods of incubation seem, from general experience, to be improbable, and it is more likely that they are to be explained by infection through the agency of fomites at a considerably later date than had been supposed.

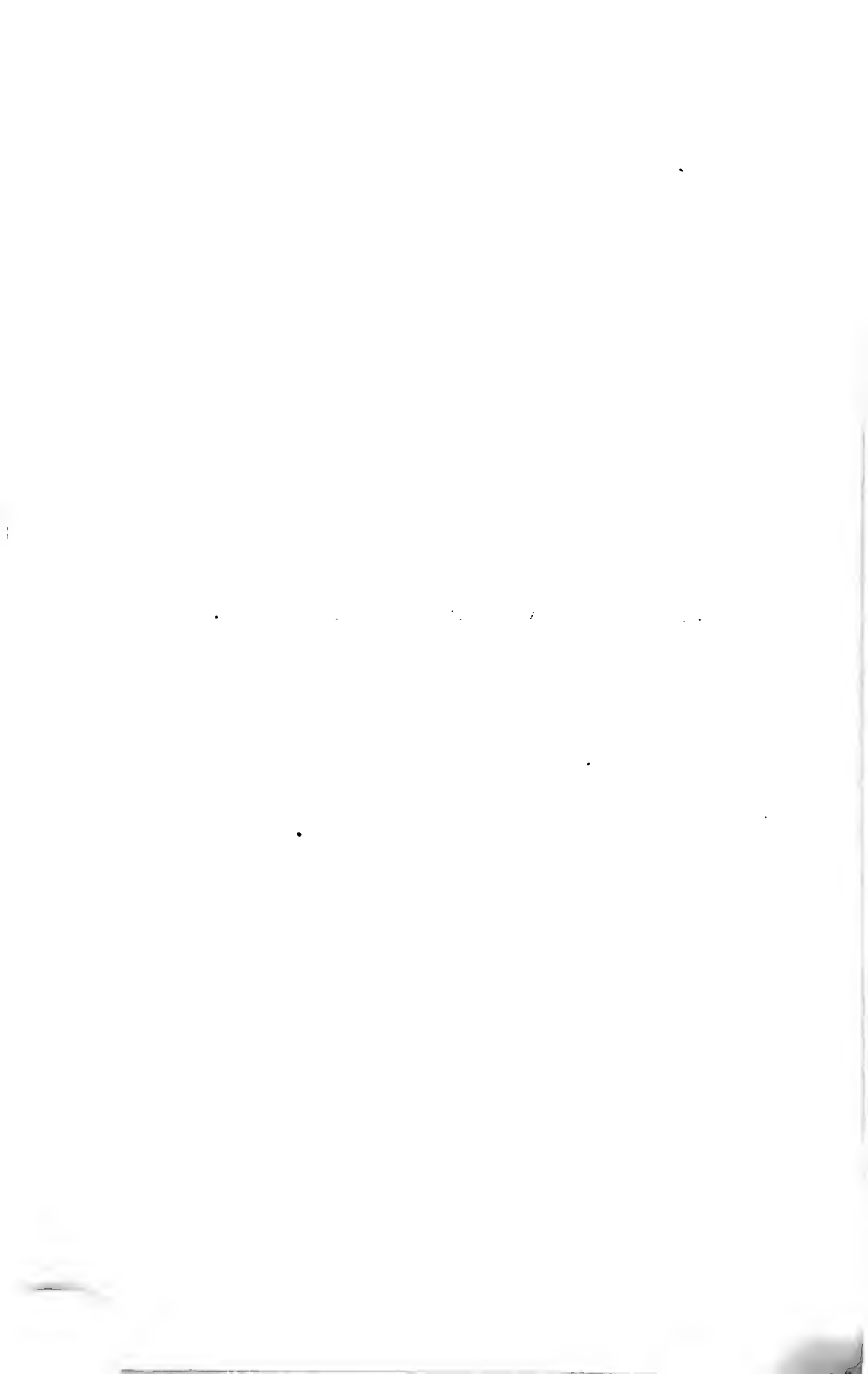


SMALL-POX AND VACCINATION

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HISTORY OF SMALL-POX

Few medical subjects have been the cause of more learned investigation and controversy than the history, and especially the antiquity, of small-pox.

The earliest records of its existence are to be found in Hindostan and in China. Holwell¹ says that a special deity was invoked by the Brahmins to ward off small-pox, and that the form of service used on these occasions, as also during the performance of inoculation, is prescribed in the Atharva Veda very many centuries before the Christian era. Next in order of antiquity, small-pox is stated to have been unknown in China² until the dynasty of Tsche-u, which began in the twelfth century B.C., and extended to the third century B.C. Inoculation is said to have been invented in China about 590 A.D.

In Arabia, the earliest known writer on the subject is Ahron, who practised in Alexandria in the time of the Emperor Héraclius (610–641 A.D.). Messue the Elder, another Arabian physician who wrote on small-pox, died about 857 A.D. But for what is known of the writings of Ahron, Messue, and others we are indebted to Rhazes, physician to the hospital at Bagdad, who died about 928 or 930 A.D. His treatise on small-pox and measles is well known, and has been frequently translated. In his own time the disease was evidently not new, and it is hardly conceivable that, had either Ahron, or Messue, or the other writers quoted by Rhazes, spoken of it as of recent introduction in their day, he would have failed to note so interesting a fact. The great prevalence of the disease is shown by the theory of its causation propounded by Rhazes, the object of the theory being to explain 'why hardly anyone escapes' attack.

In Greece and Rome.—Whether or not the disease had been known to the ancient Greeks and Romans centuries previous to Ahron's time has been very keenly disputed by medical writers, from Rhazes in the tenth century to Willan in the nineteenth.³ The chief cause of the remarkable divergences of opinion which have prevailed is undoubtedly to be found in the fact that the ancient writers looked on the principal epidemic diseases mainly as varying manifestations of one and the same fundamental cause. The Greek *loimos*, and the Latin *pestis*, or *pestilentia*, covered everything with which they were acquainted in this connection. When Rhazes was translated into Greek, small-pox was rendered loimic disease (*περὶ λοιμικῆς*), and when into Latin, the corresponding term was 'pestis.' The discussion as to Greece and Rome is, on the whole, of so speculative a nature that one cannot avoid falling in with the opinion of Greenhill⁴ that, in spite of all that has been urged by Willan, the presence of this disease in these countries 'cannot be proved,' neither can it be disproved.

¹ *Account of the Manner of Inoculating for the Small-pox in the East Indies*, 1767.

² *Mémoires concernant l'histoire, les sciences, &c., des Chinois*, par les missionnaires de Pékin, tom. iv. p. 392. Quoted in Moore's *History of the Small-pox*, pp. 22–3, 1815.

³ Willan's *Inquiry into the Antiquity of the Small-pox, Measles, and Scarlet Fever*. Edited by Ashby Smith. London, 1821.

⁴ The most convenient translation of Rhazes' work is the Sydenham Society's, by Dr. Greenhill, 1848.

Willan is more successful where, leaving aside the interpretation of particular words, he is able to find references to the general appearances and effects of what he holds to have been small-pox. The early Christians, inspired by their religion, mingled without fear with the victims of pestilence, and in nursing obtained a knowledge of the forms of epidemic disease. Eusebius describes a disorder which contributed to the epidemic that prevailed in Syria in 802 A.D., and it is not easy to get over such facts as that it was characterised by a dangerous eruption, which, unlike the true plague, spread over the whole body; which also affected the eyes, and often resulted in loss of sight; which had the effect of protecting against a second attack of the same disorder; and whose eruption was, according to a later writer, accompanied by a very offensive smell.

Gregory of Tours also makes a detailed statement relating to the latter part of the sixth century: 'De Lue quæ cum vesicis fuit.' Willan translates Gregory's narrative thus (p. 91):—'Last year the State of Tours was desolated by a severe pestilential sickness (Lue valetudinaria). Such was the nature of the infirmity (languor) that a person, after being seized with a violent fever, was covered all over with vesicles and small pustules (vesicis ac minutis pustulis). The vesicles were white, hard, unyielding, and very painful. If the patient survived to their maturation, they broke and began to discharge, when the pain was greatly increased by the adhesion of the clothes to the body. . . . Among others, the Lady of Count Eborin while labouring under this pest was so covered with the vesicles that neither her hands, nor feet, nor any part of the body remained exempt, for even her eyes were wholly closed up by them. When nearly at the point of death she received some of the water in which the tomb of the blessed Saint had been washed at the Lord's Passover. This having been taken as a drink, and applied to her sores, the fever abated, the discharge from the vesicles was made without pain, and she was soon after healed.' Gregory further says that 'the most dangerous period of the pusulæ [= pustulæ] is the time of scabbing, or incrustation of the virus.' It is a point of interest to find that Marius, Bishop of Avenche, a contemporary of Gregory, when writing of what Willan believes to have been the same epidemic,¹ uses the word 'variola' in describing it. He says:—'This year (i.e. A.D. 569–570) a violent fever, with flux of the bowels and variola, afflicted both Italy and France.' It is on account of this statement that Marius is claimed as the first who used the word variola in its modern sense.²

In Europe in the Middle Ages.—Freind, in his 'History of Physic,' and Moore,³ and nearly all later writers, agree in attributing the spread of small-pox in Europe mainly or entirely to the Arabians. Moore traces its history from the time when small-pox broke up the Abyssinian army at the siege of Mecca, in 569 or 570 A.D. In the course of their conquests the Arabians carried it to the countries along the southern shores of the Mediterranean in the latter half of the seventh century, whence, he says, it was introduced by the Moors, through Spain and France, into the continent of Europe, so that by the ninth or tenth century it had reached Saxony,

¹ Marius refers to France and Italy in the year 570, and Gregory to the State of Tours soon after 573 (Willan, p. 96).

² 'C'est Marius d'Avanches qui le premier paratt avoir employé le mot *variola* pour désigner la maladie qui nous occupe' (*Nouveau Dictionnaire de Médecine et de Chirurgie* (Jaccoud), tome xxxviii. pp. 306–7, 'Variole.')

³ Moore is one of the few really brilliant English writers on medical subjects. He was a son of Dr. Moore, of Glasgow, and a brother of Sir John Moore. His most notable works are: *A Reply to the Anti-Vaccinists* (1806), and the *History of Small-pox* (1815). He was Director of the National Vaccine Establishment.

Switzerland, and England. But in Europe in the Middle Ages only glimpses of small-pox can be obtained through the darkness which everywhere prevailed. In the practical absence of medical literature, they are to be looked for mainly in monastic writings, and are more incidental than direct. Referring to the plagues of the period, Creighton¹ says 'the common name for all such epidemics is pestis, or pestilentia, or magna mortalitas.' Willan, Woodville, Moore, and others, give various references to small-pox, many of them having to do with miraculous cures, both of the disease itself and of the resulting loss of sight. It is recorded that Baldwin, son of Arnulph, Earl of Flanders, died of small-pox in 961 A.D.,² and the disease is here called 'variolas, sive poccas.' The annals of the convent of St. Gall, in Switzerland (founded by an Irish monk who had been educated in his own country, and had then gone to Switzerland), contain the record of the attack by small-pox (variola) of the bishop of the diocese, and of his cure without pitting by one of the monks of the convent. The writer of this part of the annals died in the tenth century, which so far fixes the date of the occurrence (Moore, p. 90 *et seq.*).

In Iceland the records of the Danish Government show that small-pox was epidemic in 1241-42; caused several thousand deaths in 1257-58; so also in 1291; killed 1,600 in 1810-11; was very severe in 1847-48; prevailed again in 1879-80; caused a loss said to amount to 8,000 lives in 1480-82; prevailed again in 1462-68, 1472, 1511 (very severely), 1555-56 (killing 2,650); in 1574 (likewise severely), and so on with varying intensity in 1590-91, 1616, 1685-86, 1655, 1658, 1670, and 1672, and worst of all in 1707, when, among a population somewhat over 50,000, it carried off 18,000.³

In Ireland, if anywhere, with the lamp of learning burning all through the Dark Ages, we should expect to find evidence of the existence of a disease like small-pox. Nor is the expectation disappointed. The Irish dictionaries (O'Brien's, O'Reilly's, &c.) contain two names for small-pox—*bolgach* or *bolgagh*, and *galar* (plural *galra*) *breac*, the latter meaning the speckled distemper. In the beginning of this century Dr. Barry, of Cork, wrote (in the 'Medical and Physical Journal') of *galar breac* as the Irish name in his neighbourhood, and on inquiry through Dr. (now Sir Francis) Maccabe, medical officer to the Irish Local Government Board, we learn that in Mayo, where the native language is still spoken, small-pox is so designated in the present day. As was mentioned by Dr. Maccabe in his evidence before the Vaccination Commission, the Report on the Census of Ireland of 1851 (Part V. vol. i.) contains a long chronological record of Irish events—famines, storms, epidemics, &c. The Report states (p. 88) that both terms for small-pox (*bolgagh* and *galar breac*) were still in use. The list of events is made up, partly from the 'Annals of the Four Masters,' and partly from the monastic records themselves. *Bolgagh* reigned in 675 A.D., and in the years 679, 680, 742, 772, and 778. Small-pox is referred to also under the year 1061, though the native name is not mentioned. In 1827 *galar breac* prevailed; in 1868 the deaths of two persons of note are recorded as due to it. Under the year 1402 there is a similar entry, and so also in 1482, 1488, 1488, and 1498. These dates bring us fairly to the end of the Middle Ages.

¹ *History of Epidemics in Britain*, 1891.

² 'Circa natale Domini Baldwinus filius Arnulphi Flandriae Comitum morbo, quem medici variolas sive poccas nominant, corripitur, et in die circumeisionis Domini immediate sequente cursum praesentis finivit vitae. . . . Et in hoc monasterio sepultus fuit anno Dom. CMLXI, Regis Lotharii anno VIII.' The *Chronicon Bertinianum*, from which the above extract is taken, was written by John Iperius, or de Ipra, who was abbot of the monastery in or near St. Omer, and died in 1383.

³ Simon's papers on the *History and Practice of Vaccination*.

It will be seen that the earlier of them indicate the disease to have existed in Europe previous to its admission through Spain in the eighth century.

In England.—As to England, it is not conceivable that in the presence of Danish and other invasions the country could remain free from small-pox while it prevailed in Iceland, in Flanders, and in Ireland. Needless to say, the records are of the scantiest, but they are quite sufficient to show that the malady was well known. The 'leechdoms,' or prescriptions of an old Anglo-Saxon leech-book, have been published in one of the volumes of the Rolls Series. In the contents, No. XI. is 'Leechdoms and Drinks and Selves for Pock Disease. In all six.' In the work itself the prescriptions are given in full detail. And if anyone, regardless of the date (the tenth century), were to suggest that the disease might be great-pox, and not small-pox, it would be sufficient to point out that part of the treatment of the pocks is to 'delve away each one of them with a thorn.' There is also a prescription for a 'pock in the eye,' which may refer to injury of sight by small-pox. The leech-book belonged to Bald, a physician who had Gild for his amanuensis. It is judged to have been written either in the earlier or the later part of the tenth century.¹

The Harleian collection of MSS. in the British Museum² contains a Latin prayer or exorcism of the tenth or eleventh century, which the Anglo-Saxons used for deliverance 'potestate variolæ,' and which concludes in Saxon with an invocation for defence against 'the lathan poccas' (the loathed or loathsome pocks), indicating, as Woodville notes, 'that the inhabitants in these early times lived in continual dread of the small-pox.' The Cottonian collection has another Latin prayer, 'to St. Nicaise, which seems to have been intended for the consecration of amulets made by nuns, and, inscribed with his name, to be worn as a protection against the small-pox.' The MS. belongs to the end of the tenth century or beginning of the eleventh century. It begins by making the probably imaginative statement that 'Sanctus Nicasius habuit minutam variolam.' Of course, the point for consideration in the present connection is not the monkish capacity for inventing stories about the saints, but the fact that in the tenth or eleventh century small-pox in England was a disease to be guarded against by amulets and prayers.

Leaving the monks (who also practised medicine), we turn to the two earliest English physicians of note whose writings are now extant, Gilbert Anglious and John of Gaddesden. Freind gives evidence to show that Gilbert flourished early in the reign of Edward I., though Bale (about 1540) had placed him rather earlier. John of Gaddesden, the author of the '*Rosa Anglica*,' comes later, about 1320. Like all their contemporaries, both were Arabists, their works being largely founded on those of the Arabian writers, with alterations and additions of their own. Measured by modern standards, their writings are of little value, though Chaucer, as Creighton notes, gives Gaddesden an honoured place; while Leland (who travelled through England in the reign of Henry VIII., and made a large collection of ancient MSS. and antiquities of all kinds), speaks very highly of both. Even so recent a writer as Freind (who thinks that Gilbert was acquainted with the medicinal waters of Bath) says as to Gaddesden, 'there can be no dispute but that he was as great a practitioner as any of his time.' Both wrote of small-pox. Gilbert recommended the avoidance of cold in all forms, as of cooling medicines and cooling diet; while Gaddesden, who was a kind of

¹ *Leechdoms, Wortcunning, and Starcraft of Early England*, collected and edited by the Rev. Oswald Cockayne, M.A. Cantab. Longmans, 1865.

² Harleian MSS. No. 585, p. 202. These MSS. have been quoted by Moore, Willan, and others. We give their dates after consultation with the authorities of the British Museum.

Court physician, relates that he treated the king's son by taking care 'that everything round the bed should be of a red colour,' and he adds that the result was excellent, no pitting having been left. Of course, the red treatment was not original, nor was the opening of the pustules, which both recommended, and which had been practised in the tenth century by Rhazes in Arabia and by Bald in England.

In 1866 small-pox prevailed in England. In the British Museum there is preserved¹ a chronicle which goes down to 1419, and which was repeatedly printed in later years. Under the year 1866 it says:—'Also that tyme fell a sekness that men call ye pokkes, slogh both men and women thorgh ther enfectyne.' It was probably an extension of this epidemic that appears in the Irish Chronicles as *galar breac* in 1868. Two hundred years afterwards, when it had become desirable to distinguish variola from another disease—syphilis—that had by that time also obtained the name of *pock* in England, Holinshed in his Chronicles very sensibly—though nowadays we would not take even so small a liberty without expressly noting the fact—altered pokkes into 'small pocks.' To the same century (the fourteenth) belong the lines:—

Kynde came after with many keen sores,
As pokkes and pestilences, and much people shent,

contained in the 'Vision of Piers the Ploughman.'²

Between the years 800 and 1400 the terms *variola*, *vayrola*, *veyrola*, *vayrora*, *variolas*, and *Morbus varicus*, are mentioned in the 'Acta Sanctorum.'³ One of the references given by Dr. Fresne is to a writer (Agnellus) who died in the tenth century, and who uses the expression *variolosus in facie*. In an old English-Latin vocabulary written in 1440 '*Pokke, sekenesse*,' is given as meaning *porrigo*, and also as *Variolus vel morbulus, secundum medicos*. The work is the '*Promptorium Parvulorum sive clericorum*,' and is of unquestioned authenticity. The author wrote in the dialect of Norfolk, and gives a list of the authorities on which his vocabulary is founded. The most convenient edition is the Camden Society's, published in 1848.

The Revival of Learning.—Having indicated the prevalence of small-pox in the Dark Ages, we now come to the time of the revival of learning in Europe. Medicine, however, revived more slowly than literature, at least in England. In regard to the sweating sickness of 1529, the influenza of 1557–58, the plagues of 1603 and 1625, Dr. Creighton calls attention to the silence of native writers, and to his indebtedness to foreign sources of information. So it is to some extent with small-pox, though references occur to the disease in England in the years 1518, 1587, and, in Thomas Elyot's '*Castell of Health*,' in 1541.⁴

In the British Museum there is an interesting old work on '*Sickness and Health*,' by a writer named Bullein, published in 1562. It includes a '*Book of Simples*,' which has the following with regard to small-pox:—'*The distilled water of aples, camphere, vinegare, and milke, is a good medicen to anoint the faces of children that have the small Pockes, when the said Pockes be ripe, to kepe them from pittes or erres, provided, that they have given them in their milke saffron or mithridatum, to expelle the venome, and kepe them from the aire, during the said sicknesse. . . .*' (fol. xiii.). He speaks of syphilis as the *Frenche Pockes*. In 1598 there was published (along with '*A Defensative against the Plague*') '*A Short Treatise of the Small*

¹ Dr. Creighton, *op. cit.*, pp. 453–4.

² Quoted by Dr. Creighton, *op. cit.*, p. 452. To those who have read Dr. Creighton's work it is not necessary to explain that he entirely disagrees with the interpretation which the author puts upon these passages.

³ Du Fresne's *Glossarium ad Scriptores media et infima Latinitatis*. See also Willan.

⁴ Dr. Creighton, *op. cit.*

Pockes, showing the means how for to gouverne and cure those which are infected therewith.' In the next century, Sydenham's work stands pre-eminent.

Putting aside passages which might possibly apply to great-pox, small-pox was evidently well known to Shakespeare and his contemporaries. In 'Love's Labour's Lost' Rosaline exclaims: 'O, that your face were not so full of O's!' to which the Princess replies, 'A pox of that jest!' Donne, in his 'Anatomie of the World,' first published in 1611, asks, 'Are these but warts and pock-holes in the face of the earth?' Fletcher's 'Fair Maid of the Inn' (produced after the author's death in 1625) refers both to small-pox and the resulting pit-holes. Ben Jonson's 'Epigram to the Small-pox,' beginning as it does—

Envious and foul disease, could there not be
One beauty in an age, and free from thee?

indicates how general the disease must have been, not only in the poorer, but in the wealthier classes. This brings us to the time of the London Bills of Mortality, which began in 1629, and which fall to be referred to later on. Reviewing the evidence as a whole, there seems no reason to doubt that small-pox was well known in England all through the Middle Ages.

Nomenclature: Variola.—The word variola is not classical, but was probably coined when necessity arose for a Latin term to distinguish small-pox from other forms of pestilence. It is understood to be formed from *varius*, spotted, or *varus*, a pimple, a term which was itself applied to acne of the face by Celsus and Pliny.¹ Thompson² traces the Latin word to a Hebrew root meaning a spot or speck. The French form of the word is *vérole*, the Spanish, *viruelas*,³ and the Italian, *vajuolo*. As we have seen, it is claimed for Bishop Marius that he was the first (A.D. 570) to apply the name variola to small-pox.⁴

Pock.—The Anglo-Saxon word for small-pox is derived from *pocca*, a bag or pouch. In Dutch the form is *pok*, in German *pocke*, in Danish *kobbe*, in Swedish *pockor*. Its use has been already mentioned as a synonym for variola in the Anglo-Saxon MSS. of the British Museum, and in the tenth-century leech-book of the physician Bald, and in the Bertinian Chronicle, written in the fourteenth century regarding the death of Baldwin in the tenth century from 'variolas, sive poccas,' and in the vocabulary of 1440.

Great-pox and Small-pox.—But in the beginning of the sixteenth century some confusion arose in the application of the Anglo-Saxon term. In 1494 the great pandemic of syphilis began in Southern Europe, whence it spread rapidly through the Continental nations and Britain. At first it was called the Neapolitan disease, or the Morbus Gallicus, but soon the name pox was also given to it. Whether this was due to the 'pocky' appearance of the local sore, or of a general syphilide, it would be useless to speculate. The adjectives 'French,' or 'great,' came to be frequently prefixed to the word pox as used for the newer disease, while the prefix 'small' was retained for, or applied to, the older, which, as we have seen, had long before been described as variola minuta—the pustules being small as compared either with the size of the everyday objects with which human beings have to deal, or with ordinary boils, and with the buboes which accompanied the true plague. Salmasius ('De Annis Climacteribus,' Elzevir, 1648) refers to the variolæ, 'Quas ad distinctionem parvarum, magnas indigetamus,' and then he explains

¹ Moore, *op. cit.*, and Jacquod's *Dictionary*.

² *Inquiry into the Origin, Nature, and Cure of Small-pox*. London, 1752.

³ Dr. Mason Good makes the Spanish form the origin of the mediæval Latin variola.

⁴ The same claim is sometimes made for Constantinus Africanus in the eleventh century.

that the latter 'Lues est Venerea sive Morbus Gallicus;' and Cotgrave in his French and English Dictionary (1611) gives 'vérole' as meaning 'the small pocks,' and 'La grosse vérole' as the 'French pockes.' But the explanatory adjectives have been by no means invariably prefixed, so that when the word pox itself is used, unless the context makes the meaning clear, there is abundant room for disputation—as regards, for example, Fabian's statement that Edward IV., in 1468, was 'vysyted with the Sykenesse of pockys;' John Bale's statement, in his 'Image of Both Churches,' written about 1545, that 'ymages were soughte of euery where, Sayncte Job for the poxe, sayncte Roke for the pestilence;' and Marlowe's and Shakespeare's use of the expression 'a pox on it.' But in 'Pericles' (Act iv. scene. 6) the reference is clearly to the venereal disease.

Prevalence.—So far our attention has been confined mainly to the existence of small-pox in past centuries, though one or two incidental references have been made to its prevalence. Of course, in times before statistics began to be systematically collected we have to depend almost wholly on general statements, which are sometimes, perhaps, even more impressive than figures. Gregory of Tours writes of the epidemic of the sixth century, already referred to, that 'the State of Tours was desolated' by it. Isaac the Jew, who wrote before Rhazes, in the ninth century, says that small-pox 'happens to almost all persons.' Rhazes himself states that 'hardly anyone escapes it,' and his Greek translator in the tenth century writes, 'every man is born liable to it.' Emerging from the Dark Ages, Vidus Vidius¹ (1550) incidentally refers to it as attacking all persons in the course of their lives; and Mercurialis (born 1580) holds that almost every person must have it once (Moore). Still later, Helvetius,² physician to the King of France, speaks of 'the almost unavoidable necessity of undergoing it at one time or another.' Hillary³ (1785) says 'there is no distemper more difficult to guard against than they are, and always have been, ever since they appeared in the world.' In 1767, Schultz⁴ wrote that 'in our countries seldom any escape the small-pox.' Lettsom (1801) said that 'in reflecting upon its ravages the mind revolts in horror.' Goldson, a critic of vaccination, thought the small-pox 'probably killed more than war. Sir Gilbert Blane opined that it had destroyed a hundred for every one that had perished by the plague. George Bell, of Edinburgh, in 1802, wrote that 'the small-pox, one of the most severe and dangerous diseases to which mankind is subject, ever since its introduction into Europe, more than a thousand years ago, has descended with undiminished violence from generation to generation, and every effort hitherto made to extirpate it has failed.' Passages like these might be multiplied indefinitely. They serve to show the general impression made on the minds of physicians of all epochs by their knowledge of the disease. Wherever it existed it came to be looked on as an all-prevailing scourge.

In the New World.—The full power of the disease can be seen only on its first appearance among unprotected populations. Our knowledge of it in the New World begins in the year 1507, in the West Indies, where 'it was so disastrous that whole tribes were exterminated.' Simon gives a number of examples of its ravages in the Western Hemisphere in the sixteenth century. 'In Mexico it even surpassed the cruelties of conquest, suddenly smiting down 8,600,000 of population, and leaving none to bury them. In

¹ Cross, *History of the Norwich Epidemic*, 1820.

² *Observations upon the Small-pox* (English translation), published 1728.

³ *A Rational and Mechanical Essay on the Small-pox*, by William Hillary, M.D.

⁴ *An Account of Inoculation for the Small-pox*, by David Schultz, M.D. London, 1767.

Brazil, in the year 1568, it extirpated whole races of men. About the same period, in the single province of Quito (according to De la Condamine), it destroyed upwards of 100,000 Indians.'

Share of Total Mortality.—Various calculations referring to the last century have been made of the share of total mortality due to small-pox. Hahn returned it in ordinary years at 80 deaths in every 1,000. De la Condamine held it responsible for about 10 per cent. of the deaths in France, and Rosenstein, as a result of a Commission of Inquiry in Sweden, wrote that in that country it killed every tenth boy and every ninth girl.¹ For Russia the calculation (by Dr. Alexander Creighton, physician to the Emperor, 1812; quoted by Moore) is 'that before the introduction of (vaccine) inoculation every seventh child died annually of the small-pox.' Colon set down the deaths in France as 60,000 to 72,000 annually.²

Among statistics on a large scale are those collected by the Epidemiological Society, and published in their Report presented to Parliament previous to the Vaccination Act of 1858. The following figures are taken

Terms of years respecting which particulars are given	Territory	Approximate average annual death-rate by small-pox per million of living population	
		Before introduction of vaccination	After introduction of vaccination
1777-1806 and 1807-50	Austria, Lower	2,484	340
" " "	Austria, Upper, and Salzburg	1,421	501
" " "	Styria, Upper	1,052	446
" " "	Illyria	518	244
" " 1838-50	Trieste	14,046	182
1777-1808 and 1807-50	Tyrol and Vorarlberg	911	170
1777-1806 " "	Bohemia	2,174	215
" " "	Moravia	5,402	255
" " "	Silesia (Austrian)	5,812	198
" " "	Galicia	1,194	676
1787-1806 " "	Bukowina	3,527	516
1776-1780 and 1810-50	Prussia (Eastern Provinces)	3,321	556
" " "	Brandenburg	2,181	181
" " 1816-50	Westphalia	2,643	114
" " "	Rhenish Provinces	908	90
1781-1805 and 1810-50	Berlin	3,422	176
1776-1780 " 1816-50	Saxony, Prussian	719	170
1774-1801 " 1810-50	Sweden	2,050	158
1751-1800 " 1801-50	Copenhagen	3,128	286

from a table prepared by Sir John Simon from that report.³ The last column does not concern us in the present connection, but the column of death-rates per million living in the later part of the last century shows the mortality of the disease for the populations mentioned. Further information may be obtained from the sources indicated.

In England, the London Bills of Mortality go back to 1629, and the contribution made by small-pox to deaths from all causes is shown diagrammatically opposite p. 488. It will be observed that it was greater in the eighteenth than in the seventeenth century, and that the highest point was

¹ Haygarth's *Sketch of a Plan to Exterminate the Small-pox*, 1798.

² *A Treatise on the Cow-pox, &c.* By John Ring, Mem. Roy. Col. Surg. London, 1801, p. 928.

³ From the table as given by Sir John Simon we have excluded eight populations as to which the statistics for last century are either entirely wanting or are given only for a single year.

reached in 1796, when the proportion was 184 deaths from small-pox in every 1,000 deaths from all causes.¹ But the London Bills appear to omit all deaths from small-pox under two years of age, so that the deaths recorded from small-pox are perhaps little more than half the actual deaths.

The following are the death-rates in England and Wales since the Registration Act came into force:—

England and Wales.—Small-pox Death-rate per Million living, 1888-1890.²

Year	Rate	Year	Rate	Year	Rate	Year	Rate
1838	1,064	1852	401	1865	808	1878	79
1839	589	1853	171	1866	141	1879	25
1840	661	1854	151	1867	116	1880	29
1841	400	1855	184	1868	98	1881	124
1842	168	1856	119	1869	70	1882	54
1843	—	1857	204	1870	116	1883	89
1844	—	1858	332	1871	1,015	1884	87
1845	—	1859	195	1872	824	1885	107
1846	—	1860	188	1873	101	1886	18
1847	246	1861	66	1874	91	1887	21
1848	397	1862	80	1875	40	1888	86
1849	264	1863	280	1876	108	1889	1
1850	262	1864	367	1877	178	1890	1
1851	389						

The London death-rate per million living from all causes and from small-pox for various periods from 1660 till 1890 are given in the table at p. 485. In Edinburgh, in 1744-68, in a total of 11,618 deaths, 1,185 were due to small-pox.³

The death-rates in England and Wales from 1888, and in successive quinquennials from 1850 onwards, are also given at p. 485.

GEOGRAPHICAL DISTRIBUTION

Hirsch's great work, which is the most convenient source of information on this part of the subject, states that 'at the present time the dominion of small-pox extends over almost the whole inhabited globe.' Following Hirsch, it is hardly possible to avoid reference to vaccination, though that subject falls to be treated of subsequently. In India, where the beginnings of the history of small-pox are the remotest, it still prevails to an enormous extent. 'In China, also, where vaccination was introduced in 1805, a considerable decrease of the disease has shown itself at certain places; whereas many other regions . . . where vaccination is very imperfectly practised and inoculation still in full repute constitute permanent centres of the disease, and have often been ravaged by disastrous epidemics of it.' In Japan it is diffused everywhere. In Siberia it first showed itself in 1630, with the terrific result usual in new countries.

Since Australia was colonised the disease has been very successfully kept at bay, in spite of two accidental importations. Tasmania and New Zealand have also escaped, while many of the smaller islands of Australasia have suffered largely, though others have enjoyed 'an absolute immunity.'

¹ The greatest number of small-pox deaths occurred in 1772, when 3,992 died, but, relatively to the total deaths, the year 1796 was the worst, though the small-pox deaths were only 3,548.

² From 1889-1887 inclusive the figures are taken from Dr. Ogle's table, p. 114 (*First Report, Roy. Commission on Vaccination*), 1889, and for 1888-90 from the Registrar-General's Reports.

³ *An Account of the Inoculation of the Small-pox in Scotland*, by Alexander Monro primus. Edin. 1765.

In Asia Minor, Syria, Persia, and in Arabia, where Rhazes obtained his knowledge of the disease, it is as prominent as of old. In Egypt, the country of Ahron, and in the rest of the Nile basin, it is specially prevalent, 'so that,' as Courbon states, 'one seldom finds an Abyssinian without the marks of small-pox.' In the East Coast of Africa and in Madagascar it is 'permanently active,' and there is 'frequent prevalence' among the natives of Cape Colony. Lichenstein, who travelled through Kaffirland in 1804, found many of the natives pitted with small-pox, and the general opinion of the inhabitants was that the disease had always prevailed among them. On the Guinea Coast, as also on that of Senegambia, though not always present, it 'occurs from time to time as an epidemic, sometimes so disastrously that whole villages are ravaged by it.'

In Europe, 'the small-pox up to the beginning of this century, or to the introduction of vaccination, had been one of the most widely distributed, most frequent, and most destructive of pestilences,' and 'even at the present day small-pox takes no inconsiderable place in the aggregate sickness of European countries,' though 'since the introduction of vaccination, and especially since its legal enactment and official supervision, the occurrence of the disease has been confined within tolerably narrow limits.'

In the New World, Hirsch states that in the West Indies 'the visitations down to recent times have usually been severe; that in St. Thomas, in 1848, at least one-sixth of the population was attacked; and that only those islands where vaccination has prevailed have enjoyed comparative immunity. The importation of negroes has been a very active cause of its spread in America. In the United States, after the colonisation of the country, the disease appeared early in the seventeenth century; but in parts which have been occupied by the whites only since vaccination came in vogue it never spread largely, though in the native populations it has made frightful havoc.' In South America it has greatly prevailed, chiefly owing to its introduction by slave-ships. In Brazil and Chili it is the chief scourge, in the former country vaccination having been carried out in a very careless and imperfect way.

Climate and Soil.—What we have seen regarding the geographical distribution of the disease leads to agreement with Hirsch when he says that 'not many of the acute infective diseases show in their incidence and diffusion so complete an independence of the conditions of climate and soil.' It certainly prevails more in warm countries than in cold—in India and Africa than in Siberia and Greenland. But the very sparse population of the latter countries, compared, say, with the teeming millions of India, is doubtless largely concerned in the question.

Race.—The frequency of small-pox in warm climates may have to do as much with race as with locality. So far, at least, as concerns the negroes, it has been established that, whether in their own country or elsewhere, as in America, they are specially susceptible both to attack and death by small-pox. They have repeatedly been the means of importing it into the West Indies, and have themselves suffered very severely when the disease has appeared in American towns where they lived in slavery. Possibly the depressing and dispiriting conditions of slave-life may have had something to do with the excessive susceptibility in such circumstances, but we are not aware that their reaction towards other acute infectious diseases has been similarly influenced.

Season.—Season has a considerable effect on the spread of small-pox. In Oriental countries it appears to be favoured by cold and retarded by heat. Hirsch considers that this rule holds good in India, China, Cairo, Constanti-

nople, Tunis, Brazil, &c. In Calcutta the greatest number of deaths take place in March, and in Bombay in April. In ninety-nine epidemics which occurred in Europe and North America, sixty-seven were at their height in the cold season, and thirty-two in the warm season. It is evident from these figures that the rule has many exceptions; and this is further shown by a table which Hirsch gives of the number of deaths that occurred in the several parts of the year in England, the Netherlands, several Continental cities, and Philadelphia. In winter the total deaths were 58,729, in spring 55,078, in summer 40,914, and in autumn 54,552. In tropical countries the rainy season puts a check on the disease.

Sex.—At most ages the mortality is greatest among males, but in the third quinquennium, and in children aged between one and three years, the reverse is, to a slight extent, the case (Whitelegge).

Periodicity.—To avoid fallacies due to the practice of vaccination, it is necessary to go to pre-Jennerian times for illustrations of the periodicity of small-pox. The diagram opposite p. 488 indicates the facts for London, but the disease was so constantly present there that there may be difference of opinion as to what standard of epidemic prevalence should be set up. In the forty-seven years there given of the seventeenth century, it may be said that there were fifteen outstanding years, and in the whole of the eighteenth century thirty-four such years. This would give a periodicity of about one year in three. In Copenhagen,¹ in the latter half of the eighteenth century, there were thirteen years which showed beyond their fellows with regard to small-pox—1750, 1755, 1759, 1764, 1769, 1775, 1779, 1782, 1785, 1789, 1791, 1794, 1797; but from about 1775 onwards the distinction between epidemic and non-epidemic years is not so marked as in the previous twenty-five years. The periodicity here may be said to be four years. In Berlin the intervals were irregular, there having been epidemics in 1759, 1766, 1770, 1786, 1789, and 1801.² In Edinburgh, in the twenty years beginning 1744, there were seven epidemics, though the disease was never entirely absent.³ In Glasgow, in the twenty years beginning 1788, small-pox was constantly prevalent, but about six of the years showed such excessive amount as may be taken to indicate its periodicity. In Kilmarnock (which has always had a good deal of communication with the neighbouring city of Glasgow), in 1728–64 there were nine epidemics, or one about every four and a quarter years. In Geneva (population 26,000), it appears from Dr. Haygarth (who had an official letter from the town authorities in 1791) that epidemic were sharply and clearly distinguished from non-epidemic periods, and that they occurred regularly every five years. In Bengal, according to Holwell,⁴ ‘every seventh year, with scarcely any exception, the small-pox rages epidemically . . . during the months of March, April, and May, and sometimes until the annual returning rains, about the middle of June, put a stop to its fury.’ Hirsch gives the following years for Boston, U.S.A.:—1649, 1666, 1678, 1690, 1702, 1721, 1780, 1752, 1764, 1776, 1788, 1792; about one year in twelve being epidemic;⁵ and for Vienna, 1742, 1745, 1749, 1757, 1759, about every fourth year being epidemic.

These statistics relate, with the exception of Bengal, to centres of population varying in size from about 4,500 (Kilmarnock) to about half a million (London). For purely rural districts exact data are almost wanting. Hillary

¹ Simon, *op. cit.*

² Dr. S. W. Abbott, Mass., U.S.A., in the *Reference Handbook of the Medical Sciences*.

³ Monro *primus*, *op. cit.*

⁴ *Op. cit.*, p. 4.

⁵ The population of Boston was 4,000 in 1678 and 20,000 in 1792 (Abbott, *loc. cit.*).

(1785) says the great part of his 'Rational and Mechanical Essay on the Small-pox' was written 'when the small-pox was epidemical in Ripon and the circumjacent parts of the country several years since;' and he continues that 'this disease is observed to return and visit the same parts of the country once in five or six years (or oftener if populous).' Black¹ states, however, that 'numbers, without doubt, in the country live and die at an advanced age without undergoing this distemper;' and other authors indicate similar views. The more isolated the locality, the seldomer was it invaded by small-pox. In a 'Description of the Western Islands of Scotland,' by W. Martin, in 1716, it is stated that 'the small-pox commonly comes once in seventeen years' time.' In Shetland it was epidemic about every twenty years.² This being so in insular regions, probably we may take it that in the mainland rural districts outbreaks would not be less frequent than once in a dozen years or so. In Iceland we have seen (p. 887) that the intervals were often even longer, but varied very greatly from four years up to twenty and thirty and forty years, and, indeed, no epidemic is recorded between 1480 and 1511, a period of eighty-one years.

Hirsch thinks that only two factors determine the recurrence of an epidemic of small-pox—'one, the necessary number of persons susceptible of the morbid poison, and the other, the introduction of the virus itself.' No doubt these are important agencies. But the remarkable regularity with which the disease reappeared in places like Geneva and Kilmarnock, and the periodicity of its excessive prevalence in London, where it was never absent, indicate the existence of other causes. In Kilmarnock, each year's births numbered about 184, but it was not until the births had accumulated for four years, or thereby, that the disease broke out. And the fact that the recurrence was never delayed beyond five years shows that the other essential conditions, whatever they might be, were never very long absent. In this respect small-pox bears some resemblance to other acute specific diseases of childhood—measles, whooping-cough, and scarlet fever, which recur with a rhythmic regularity that cannot be accounted for merely by the introduction of the poison to a susceptible population, and which prevail more frequently in populous towns than in rural districts.

Age.—In places where small-pox regularly prevailed previous to Jenner's time, it was a disease of childhood to an extent that is hardly realised in the present day. The table at p. 486 shows that in Geneva (1580 and 1760), out of every 1,000 small-pox deaths, 961 were of children under ten years. In Kilmarnock, in the years 1728 to 1764, only 1·2 per cent. of small-pox deaths occurred over ten years old; and in London, previous to the vaccination law of 1858, 815 per 1,000 were under that age. In an epidemic in Chester in 1774, in a total of 202 small-pox deaths, 51 were under one year, 38 between one and two years, 42 between two and three years, 49 between three and five years, and 22 between five and ten years. There were none over ten years.³ Arguing from such data, a writer at the beginning of this century might well have held that the period of childhood specially predisposes to small-pox, while advancing years have a strong protecting influence. But just as measles, when admitted for the first time into a community embracing all periods of life, attacks old and young without distinction, so small-pox, when it gets a similar opportunity—as in the West Indies after the discovery of America, or in Iceland in 1707, after having been absent for a generation—shows that it is no respecter of adults. In Iceland

¹ Black's *Observations, Medical and Political, on the Small-pox*. London, 1781.

² Sir John Sinclair's *Statistical Account of Scotland*.

³ C. E. Paget, *Trans. Epidem. Soc.* 1883-84.

about 86 per cent. of the total inhabitants perished, so that the disease was not confined to children; and the table at p. 447 shows that in the present day, in the absence of special protection, no time of life is free from it. The reason that in places like Geneva and Kilmarnock in former centuries *only* children died of small-pox, is to be found in the fact that, practically, *all* children were attacked by it, and so obtained protection against attack, and especially against fatal attack, in later life. Indeed, the periodicity of small-pox is the governing factor in its age-distribution. In Kilmarnock and Geneva, epidemics came every four or five years, and the disease was, to nine-tenths of its extent, a disease of children under five years old, the average age at death in Kilmarnock being not much over the average age of the children born since the previous epidemic. But in Boston, U.S.A., where epidemics came every twelve years, the average age at death would be greater; and so also in country towns and villages, and in the Hebrides, Iceland, &c. In London, small-pox attacks among adults appear to have been not at all uncommon, and the explanation is doubtless to be found in the largeness of the numbers of immigrants from country districts, where opportunities of attack were less frequent. How this whole subject of age-incidence is nowadays linked with vaccination is shown in detail at p. 485.

FATALITY

Of late years, and especially since Dr. Barry, in his Sheffield Report, used the word 'fatality' as a substitute for 'case mortality,' it has conveniently supplanted the latter term.

In *new countries* the fatality of natural small-pox has varied considerably in different epidemics. Marson¹ refers to an outbreak among a tribe of American Indians in which 'every individual of the tribe was swept away.' In Greenland,² in 1784, nearly two-thirds of the whole population (numbering from 6,000 to 7,000) were destroyed. In Mexico, in 1520, it is said that 'half of those infected died of the distemper.'³ Oatlin mentions that, of twelve million American Indians, six millions fell victims to small-pox. In the Icelandic epidemic of 1707, where 86 per cent. of the population of the island perished, the fatality must have been enormous. It would thus appear that the fatality, like the mortality, is greatest in new countries and among unseasoned peoples. But there is another and a most important factor that must be borne in mind in this connection, and that has a bearing on these high death-rates.

Influence of Age.—The fatality differs at different ages. It is very high in infancy, decreases steadily and rapidly to its minimum in the third quinquenniad, and then rises regularly all through the rest of life. Now, we have already seen that the periodicity of the disease determines its age-distribution. Making allowance for the natural infrequency of the disease in the first few months of life, and for people who happened to escape during one or two outbreaks in the locality in which they lived—we are thinking of the last century—the average age of those attacked would be somewhat greater than the average age of the people born since the preceding epidemic. Thus, when we come to countries where the disease seldom found entrance—as in those above mentioned—we get a very high fatality, because the average age of the victims is far beyond the 10–15 years at which the disease is least mortal. Similarly, at the other extreme of life, in places like Geneva, with epidemics every four or five years, there would, *ceteris paribus*, also be a high fatality. So far as age has to do with death-rates, those places, therefore, would be best off that had epidemics occurring regularly at, perhaps,

¹ Reynolds' *Medicine*, vol. i.

² Simon, *op. cit.*

³ Black, *op. cit.*

twelve or fifteen years' interval, with a great many of the attacks at ages of from eight to fifteen years.

Jurin's Statistics.—The fatality of English small-pox in the last century is often stated at about 16 to 18 per cent. or thereby. This statement is chiefly based on the data collected by Dr. Jurin, secretary to the Royal Society, and later by Scheuchzer, and published in yearly pamphlets from 1728¹ to 1729. These pamphlets give detailed statements of the places, cases, and deaths on which the percentage-fatality is based; and, as they are largely relied on by opponents of vaccination, they merit more than a passing notice. In the first place, it is to be observed that, of about 18,000 cases, nearly 6,000 (5,742) belonged to Boston in New England. Among these, 841 deaths occurred, giving the low rate of 14·6 per cent., and so lowering the average of the whole list. But we have learned from Hirsch (p. 895) that small-pox had been epidemic in Boston in 1690, in 1702, and next in 1721, and it was the figures of the last of these epidemics that Jurin had got. The average age of those attacked in that outbreak would be (calculated as above described) about twelve years. In other words, the low fatality was due to the fact that the age-incidence of the disease was about as favourable as it could well be. Not only so, but when we compare Jurin's rates with present-day rates, we must recollect that an unknown proportion of people may have had some measure of protection from the various forms of modified small-pox that occasionally prevailed, under such names as swine-pox, horn-pox, &c., and that some of the recorded percentages may have belonged to outbreaks which, though modified, were still recognised as variolous.

Looking next to the other places on Jurin's list, we find that the fatality varied very greatly. In a part of the parish of Halifax it was only 10·4 per cent., in Hatherfield 11·1 per cent., and in Ware 11·8 per cent.; while in Bradford it was 27·9 per cent., in Stockport 25·4, and in Leeds 28·9 (Jurin's first Account, 1724). The joint population of Ware, and of the adjoining part of the parish of Amwell, to which the statistics refer, was 2,515 (Royal Society's MS. Papers on Inoculation, p. 21). Unfortunately, we do not know how often epidemics occurred in these places, nor, excepting as to Ware, do we even know their population; but the suggestion is that the places with a high fatality were the towns, where epidemics might be frequent and attacks confined mainly to early childhood; while the places with a low fatality were rural districts or villages, with fewer epidemics, and a consequently more favourable range of ages.

Jurin's published reports do not in any case give the ages at death. But it happens that the volume of manuscript papers just mentioned, preserved

Small-pox.

Ages	Cases	Deaths	Deaths per cent. of cases	Ages	Cases	Deaths	Deaths per cent. of cases
0-1	—	—	—	25-30	9	8	88
1-2	—	—	—	30-40	12	8	25
2-3	3	2	67	40-50	10	4	40
3-4	4	1	25	50-60	4	1	25
4-5	6	—	—	60-70	4	2	50
5-10	15	1	7	70	2	1	50
10-15	33	3	9				
15-20	14	1	7				
20-25	16	3	18	Totals	132	25	18·9

¹ *A Letter to the Learned Caleb Cotesworth, M.D., containing a Comparison between the Natural Small-pox and that given by Inoculation, London, 1728. An Account of the Success of Inoculating the Small-pox in Great Britain, 1724; &c.*

in the archives of the Royal Society, gives the details for Aynho, in Northamptonshire, where the percentage-fatality was 18·9, the cases having been 182 (wrongly stated in Jurin's Account for 1726 as 183) and the deaths twenty-five. The facts, though scanty, are so interesting that we tabulate them in full. In this table the average fatality is just about that resulting from the whole of Jurin's statistics. And it is obvious that the cause is to be found in the large number of cases at the more resistant periods of life—from five to twenty-five years, when the fatality is low. This is exactly what we have been arguing for in the case of Boston, in New England, and it goes a long way in interpreting Jurin's statistics.

Another point, equally important, has to be noted, namely, that no deaths from small-pox are here recorded under two years of age. This is entirely contrary to modern experience, though in a small population it might conceivably be a singular accident. But in Jurin's Letter to Dr. Cotesworth (1728) we find the remarkable opinion that the risk ('hazard') of death from small-pox 'increases after the birth, as the child advances in age;' and in the same letter, in the course of his argument, he assumes 'that out of every 1,000 children that are born, 886 die without having the small-pox,' these 886 being 'not above one or two years of age,' and constituting, under such headings as 'Overlaid, Chrysoms, and Infants, Convulsions,' &c., all the deaths under that age. It would thus seem that in making his calculations Jurin did not knowingly set down any deaths under two years old as due to small-pox, putting them all aside as chrysoms (i.e. dying unbaptized) and infants, &c., and that some of his correspondents, in furnishing him with statistics, followed the same rule. Knowing, as we now do, that small-pox is exceedingly fatal under two years of age, it is obvious that Jurin's percentages are irretrievably damaged by this extraordinary omission, and that they are of very little value unless where, as in the case of Boston, we know the date of the previous epidemic, or where, as in Aynho, we have a statement of ages as well as deaths. Summing up the small-pox statistics published regarding Geneva, the Hague, Kilmarnock, Edinburgh, Manchester, Warrington, and Chester, for former centuries we find that of 86,755 small-pox deaths no less than 17,252 were under two years of age.

Hospital Fatality.—A prominent anti-vaccinationist has made the extraordinary blunder—in which he has been unquestioningly followed by other opponents of vaccination—of assuming that Jurin's statistics referred to hospital cases, though they belonged to a period nearly a quarter of a century before the London Small-pox Hospital was opened, and when, so far as we can find, there was nothing to call a small-pox hospital anywhere in England. The hospital at King's Cross, now at Highgate, was opened in 1746, and in the years 1746–68 the cases were 6,456 and the deaths 1,684, the fatality-rate being 25·8 per cent. In the last twenty-five years of the century it rose to 82·5 per cent.¹ In 1836–51 the rate fell to 21·9 per cent., and in 1852–67 to 18·5 per cent. The hospitals of the Metropolitan Asylums Board were opened in 1871. In 1870–72 the total rate of the London small-pox hospitals (in 14,808 cases) was 18·7 per cent.; in 1876–80 (15,171 cases) it was 17·6 per cent.; and in 1881 the rate in the Deptford Hospital (8,185 cases) was 17·8 per cent., and in the Fulham Hospital (1,752 cases) 14·2 per cent. But, again, it must be pointed out that to judge of the actual value of these rates it would be necessary to tabulate the mortality at each separate period of life.

Though age must have been by far the most important factor in small-pox fatality in the last century, it is not to be supposed that it was the only factor. We have seen that the disease appears to have taken on a special

¹ Seaton, *Handbook of Vaccination*, p. 190.

intensity of type in new countries, as in Greenland, Iceland, Mexico, &c. And, on the other hand, in places where it was almost endemic, as in London, and in Glasgow and Kilmarnock, there may have been a corresponding mildness of type, due partly to the weeding out in course of time of families specially susceptible to fatal small-pox. Probably, also, local conditions had their influence, and such differences would exist between one epidemic and another as we know to belong to measles, scarlatina, whooping-cough, and other similar maladies.

CAUSE AND MODE OF DISSEMINATION

'As to what may be the *essence* of small-pox, I am, for my own part, free to confess that I am wholly ignorant; this intellectual deficiency being the misfortune of human nature, and common to myself and the world at large.' So wrote Sydenham 250 years ago, and so, it must be confessed, he might again write if he were alive to-day. Like other members of the group of exanthemata to which it belongs, small-pox is an acute infectious fever with specific characters—and especially an eruption—distinguishing it very clearly from all other maladies, these characters reproducing themselves unmistakably from one case to another; so that the descriptions by Rhazes and Sydenham are as true now as when they were first published. Its power of reproduction shows the organic nature of the contagium, but we have as yet practically no knowledge of its dependence on any specific organism. All that Flügge says is that 'Cohn, Weigert, Koch, and others, found micrococci in the pustules and in various internal organs in persons who died of small-pox,' but that 'cultivations have not yet been made.'

Infectivity.—The first known approach to the discovery of the infectious nature of small-pox was made by Ali Abbas (980 A.D.). A few years later (992 A.D.) Avicenna was quite clear on the subject, both as to small-pox and measles. 'Et variolæ quidem et morbillus sunt de summa ægritudinum contagiosarum' (Moore). In England in 1866 (p. 889, *ante*) the infectivity of the malady was evidently recognised, but the fact appears sometimes to have been forgotten, and even Sydenham is silent regarding it. Ultimately Boerhaave, in the beginning of the eighteenth century, distinctly asserted it, and it is not likely again to be lost sight of.

The disease is disseminated from the sick to the healthy mainly by means of the air, and is doubtless received into the body through the respiratory tract. It can be carried by fomites, as by epithelial debris, cloth fibres, &c., but as the contagium itself has never been separated, it is useless to speculate as to the particular form or condition in which it can pass into the atmosphere. The bodies of persons who have died of the disease, the beds on which they have lain, the furniture of the room, and all such ordinary means of infection, have their share in spreading small-pox. Dr. Pringle mentions that in Hindostan he traced several cases to the fact that small-pox crusts had in some way got mixed with a supply of sugar for domestic use.

Aërial Convection.—One of the characteristics of small-pox is its remarkable carrying-distance. Haygarth, in the last century, based his 'Plan to Exterminate the Small-pox' on the view that infection through the atmosphere failed at a very short distance. In 1784 he instanced the case of three persons who did not catch the disease though only half a yard distant from a child with pustules and scabs on its skin, and in 1798 he quotes experiments in the same direction by a Dr. O'Ryan. O'Ryan put a dossil of cotton soaked in small-pox matter on an oval table whose smallest diameter was three feet, and set round it, day after day, six children. He renewed the cotton regularly, but the children were not infected. But O'Ryan and

Haygarth seem to have neglected the obvious fact that the conditions necessary to produce infection in the individual are not always present. In an experiment like O'Ryan's it would appear to be the condition (fluidity) of the small-pox matter that is at fault; while in an epidemic, persons who are exposed to infection and escape are likely to do so through being themselves for the time insusceptible, the insusceptibility in the present day being usually due to vaccination.

The well-known investigations of Mr. Power regarding the influence of the Metropolitan Asylums Board's Hospitals on the spread of small-pox have led him to conclude that the virus can sometimes retain its activity while passing through a quarter of a mile or more of London air. The main investigation took place with regard to Fulham Hospital, and the facts are set forth in detail by Mr. Power in an appendix to the Hospitals Commission's Report of 1882. They are supported by subsequent reports by the same observer in the Local Government Board's Annual Supplement. Taking Fulham Hospital as a centre, spot-maps were prepared with concentric circles distant one-quarter, one-half, three-quarters, and one mile respectively. Dealing with fortnightly periods in the years 1877-80, the amount of small-pox within the hospital was shown, and also the extent to which, during successive fortnights, houses within the several quarter-mile belts were newly invaded by the disease. No sooner did the fortnightly admissions of acute cases amount to thirty-five or forty, than the influence on the surrounding neighbourhood made itself felt. The following table shows the main facts during five epidemic periods.¹ In other tables it is shown that the rates per 100

Admissions of Acute Small-pox to Fulham Hospital, and Incidence of Small-pox upon Houses in Several Divisions of Special Area, during Five Epidemic Periods.

Cases of acute small-pox admitted	In epidemic periods since opening of hospital	Incidence on every hundred houses within the special area and its divisions				
		On total special area	On small circle 0- $\frac{1}{4}$ mile	On first ring, $\frac{1}{4}$ - $\frac{1}{2}$ mile	On second ring, $\frac{1}{2}$ - $\frac{3}{4}$ mile	On third ring, $\frac{3}{4}$ -1 mile
327	March 1877 to end of 1877 .	1·10	3·47	1·37	1·27	·86
714	Jan. 1878 to Sept. 1878 .	1·80	4·62	2·55	1·84	·67
679	Sept. 1878 to Oct. 1879 .	1·68	4·40	2·63	1·49	·64
292	Oct. 1879 to Dec. 1880 .	·58	1·85	1·06	·80	·28
515	Dec. 1880 to April 2, 1881 .	1·21	3·00	1·64	1·25	·61
2,527	Five periods	6·37	17·35	9·25	6·16	2·57

houses outside the special area were much less than within it. The general conclusions were:—(1) That the hospital contributed largely to the prevalence of small-pox in the neighbourhood; (2) that the incidence bore a very exact relation to propinquity to the hospital; (3) that houses in the chief thoroughfares were not exceptionally affected; (4) that variations in the use of the hospital were followed by corresponding variations outside, but that convalescent cases were not factors in the influence; (5) that the influence was greatest when admissions to the hospital were beginning to increase rapidly; (6) that the comparison held good with regard to successive epidemics; (7) that the hospital administration was not responsible; (8) that the conditions contributory to the small-pox around the hospital pertained to the hospital; and (9) that 'during the present epidemic period, and most probably during former similar periods, there has arisen in the atmo-

¹ Reports of the Medical Officer for 1882, 1884, 1885, and 1886.

spheric circumstances of the time peculiar facility for the dissemination in an undamaged state of any matter that may have been given off from the hospital.' The influence of a rising epidemic is probably important, and its presence or absence may help to explain differences of experience by other observers. Later inquiries, also conducted by Mr. Power, led him to the same conclusion. In these inquiries a detailed comparison was made of the prevalence of small-pox in various metropolitan districts—(1) previous to the use of the Metropolitan Asylums Board's hospital, and (2) subsequently. The effect of the inquiry went to show that those districts in which hospitals were erected were thereby brought to occupy a worse relative position in regard to small-pox prevalence than they had occupied previous to 1871, when the hospitals first came into use. In particular, a district (Hackney) which had one of the lowest mortalities in London in the former period came to have the highest in the latter period, and in the other hospital districts the facts tended in the same direction.¹

Insusceptibility.—As in the case of other similar diseases, a few persons appear entirely insusceptible to the contagion of small-pox. Kilpatrick² (1761) thought it possible that two persons in a hundred might be naturally exempted, but he mentions that Reidlin thought this percentage too large. Cantwel said that in France every fiftieth or hundredth person entirely escaped.³ Haygarth and Sauvage thought the proportion of exempted persons amounted to about 5 per cent. (Cross), and Cross, in the Norwich epidemic, 1820, found, in 608 persons, that ten to fifteen were insusceptible. Woodville gives one in twenty as representing the temporary insusceptibility of adults, while among children he states it as one in sixty.

Incubation.—Defining incubation as that period after reception of the poison during which there are no symptoms, either febrile or eruptive, then the length of incubation of small-pox is twelve days (twelve times twenty-four hours). But between the reception of the poison and the appearance of the eruption the interval is from about thirteen and a half to fourteen days, the initiatory fever occupying the last part of this period. The eruption therefore appears on the fourteenth or fifteenth day after infection. In three or four cases Marson found the time rather less, but these were vaccinated persons, in whom other stages of the disease are often shortened.

Period of Infectiveness.—This, which is considered to date from the commencement of the initial symptoms of illness, varies according to the length and severity of the attack and the time of separation of the crusts. After crusts and scales have entirely disappeared, hot baths should be given every day or two for ten or twelve days, after which the patient may be looked on as free from infection.

From the returns of the Asylums Hospital Board, London, Wynter Blyth⁴ has found that the mean time in vaccinated cases, from the beginning of the eruption till the discharge from hospital, is twenty-five days, the maximum being fifty-five and the minimum eighteen.

PROTECTION

The following general considerations anticipate to some extent what follows in more detailed order under separate headings.

1. Protection of the individual from *attack* by small-pox is obtainable in three separate ways: (a) by previous small-pox attack acquired in the

¹ Dr. Buchanan's Supplement to the *Sixteenth Annual Report of the Local Government Board*.

² *An Analysis of Inoculation*, by J. Kilpatrick, M.D., 2nd edit., 1761.

³ Schultz, *op. cit.*, p. 127.

⁴ *Manual of Public Health*, 1890.

‘natural’ way; (b) by successful inoculation of small-pox (variolation); (c) by successful vaccination.

2. Formerly—at the beginning of this century—‘protection,’ in whichever way obtained, was considered complete and practically permanent. Nowadays we have seen reason for modifying this view.

3. But now, as formerly, we hold that ‘protection’ in course of being acquired is very differently exhibited, according as it is resulting from (a), from (b), or from (c). For instance, in the (a) case a person suffers a malady from which he perhaps barely escapes with his life, whereas in the (c) case he would not be considered ill at all but for the manifestation of a locally induced affection well known to be commonly correlated with constitutional indisposition.

4. And just as protection is to be obtained, as a result of (a), of (b), and of (c), so also it is obtainable from differing degrees of (a), differing degrees of (b), and differing degrees of (c). Thus as regards (a), the protecting *small-pox attack* may be of the confluent sort, or it may be discrete and mild. Commonly it is one or the other. But it may be as trivial as that which has hitherto passed under the names horn-pock, swine-pock, nirl-pock, chicken-pock, water-pock, &c.; or it may be even destitute of eruption in the form of so-called ‘*variulous fever*’; (b) the protecting variolation may have associated with it eruption of confluent or discrete small-pox, most commonly the latter. But exceptionally the operation may be wholly dissociated from general variolous eruption, and result merely in a local variola, perhaps unassociated with constitutional indisposition; (c) the protecting vaccination may have associated with it (though rarely) a general vesicular eruption with considerable fever. But most commonly the *vaccinia* induced is limited to the point or points at which lymph was inserted, and is supplemented by more or less of constitutional indisposition. *Vaccinia* in the above sense and local variola without constitutional indisposition are, be it observed, at opposite poles in their respective classes. The former represents complete expression of the malady sought to be induced, whereas the latter (the local variola without constitutional indisposition) indicates the minimum expression of the variolous process, and the comparative protection given by the one process and by the other is not to be measured by the same standard of presence, or absence, or plentifulness, or scantiness of a general exanthem, *vaccinal* in one case and *variulous* in the other.

5. But though ‘protection’ results from (a), from (b), and from (c), and from differing degrees of each of them, it is not to be regarded as equally lasting in all. Thus we may think of (1), an attack of confluent natural small-pox, as a more lasting protection than an average variolation or an average vaccination. In like manner it is open to us to regard (2), a severe attack of natural small-pox, as likely to have commonly been a more lasting protection than a mild or doubtful attack; (3) a local variolation followed by general variolous eruption as likely to have commonly been a more lasting protection than an exceptional local variola without such eruption. In just the same way as (4) we now regard a complete (multiple) and efficient vaccination associated with constitutional indisposition as a more lasting protection than an incomplete (single) and inefficient vaccination doubtfully followed by constitutional indisposition.

Indications of differing values of ‘protection’ within the limits of each of the several classes (a), (b), (c) tempt us to surmise what may be, and especially what in times past *may have been*, the relative value of one and another ‘protection’ under equal conditions of subsequent exposure to the contagion. For instance:—In past times was or was not a mild, discrete

attack of natural small-pox any better (in the sense of lasting) protection than an average variolation, and an attack of 'variola fever' (i.e. small-pox without eruption) any better protection than a Suttonian inoculation? Similarly, and in the beginning of the present century, what were the relative values of a Suttonian variolation and a vaccination (efficient as was casual vaccinia), *both of them having been undergone in the first year of life, with thereafter (as above) equally frequent and sustained exposure to the contagion of small-pox?*

The possible importance of equally frequent (and when incurred equally sustained) exposure to small-pox will be referred to presently; but, meanwhile, it is necessary to say a word as to the probable importance of *equal ages at reception of protection* when one sort of 'protection' is to be compared with another. Much may depend on this. It is coming to be seen nowadays that revaccinated adults resist small-pox attacks to a far greater extent than adolescents, who depend solely for their 'protection' on a primary vaccination done in infancy. Lest, however, it be assumed that the protection bestowed by a revaccination is something superadded to that remaining from primary vaccination, i.e. that the two together add up, so to speak, to a total stock of protection larger in amount than that possessed by the individual at the date when in infancy his primary vaccination had just run its course, it is necessary to point out that a revaccinated adult has neither more nor less protection than was obtained by his ancestor who was primarily (and efficiently) vaccinated in adult life. A person revaccinated in adult life, his ancestor primarily vaccinated at twenty-five years of age, and the vaccinated baby of to-day would, immediately after their vaccination had run its course, react, or would have reacted, in exactly similar fashion to variolation or to exposure to the infection of small-pox. Less sustained resistance, then, of infantile vaccination as compared with adult vaccination or adult revaccination is not referable to less amount of (less complete) vaccination in the infant at the time it was obtained; rather it is due to a more rapid loss of protection by the infant than by the adult, such loss being due to the circumstance that after it has been obtained the infant and child grows and develops, whereas the adult merely maintains the *status quo ante*.¹ In the one case, that is, there is simply slow repair of the tissues as they waste; in the other there is rapid multiplication of—addition to, dilution of—the tissues which were modified in the 'process of protection.'

With reference to the different estimate by ourselves and by our grand-fathers of the permanence of 'protection,' we have found reason for including within the term 'small-pox' minor eruptive malady, or maladies, the variolous nature of which they denied; and further, we have had, which they had not, opportunity of observing the behaviour towards small-pox of large groups of 'protected' persons of various ages exposed to infection of that disease at long intervals after their 'protection' was acquired. For us, history and experience are to the following effect:

At the beginning of this century the majority of the adult population of this country had already passed through small-pox. Some adults had had natural small-pox, some of them small-pox by inoculation; but, whatever their protection, i.e. whether by natural small-pox or by variolation, *they*

¹ The Germans have required, since 1874, for females *two* vaccinations, but for able-bodied males *three*—viz. both sexes in infancy; both sexes at eleven or twelve years; males only on entering the army. So that, if revaccination at eleven or twelve years does not protect through the remainder of life, we shall know of it in the future by fatal small-pox in adult Germans, *confined mainly at first to the female sex.*

had continued thereafter (at any rate in towns) to be (like their ancestors) frequently exposed to the infection of current small-pox. So, too, with adolescents. At that date a considerable proportion of them had also had small-pox or had been variolated; and they, like their seniors, had been afterwards exposed, in their degree, to frequently recurring epidemics of small-pox. Meanwhile children, and especially babies, instead of being left as during last century to take their chance of current small-pox, or to await a favourable opportunity, delayed, perhaps to five years of age or later, for inoculation, were getting vaccinated, along with certain of their seniors who had escaped small-pox, and had not been purposely variolated, in rapidly increasing numbers.

Small-pox, therefore, in the early years of this century, when it tended to become epidemic here and there in this country, found far fewer persons than ever before susceptible to its infection. And, as a consequence, small-pox epidemics were, seventy to ninety years ago, less easily set going; they were less widespread, and the intervals between them became longer than had been the case last century. As a further result of diminished small-pox, and of its less frequent epidemicity, persons who had obtained 'protection,' in whatever way, lived less constantly than their forefathers in the presence of small-pox; their 'protection,' therefore, was less frequently tested, and tested only at longer and longer intervals. It was under these circumstances that there occurred in the first quarter of this century, and seemingly only, or most conspicuously, in the presence of small-pox epidemics arising after several years' freedom from the disease, multiple cases of transient indisposition associated for the most part, though not invariably, with general eruption on face or body, or both, of papules, pimples, vesicles, which ran a short course and were not attended by a so-called 'fever of maturation.' Mainly, such attacks were taken note of among children that had been vaccinated in infancy (but they were not confined to such persons, or to those that had not had small-pox) living or associating with unvaccinated relatives and others that were undergoing unmistakable small-pox. As to a variolous nature of these attacks there was much and long controversy, in the course of which the advocates of permanent protection by vaccination did not fail to point out that such eruptions had been not uncommon, and under similar conditions of epidemic small-pox, long before vaccination was heard of, and that persons who were marked by previous attacks of small-pox had in those other days been subject to them, or at least to one variety that had obtained the name of 'horn-pock.'

The time at length came—vaccination having greatly increased and become more and more an affair for babies, and small-pox having meanwhile continued to diminish, and the intervals between epidemics to become longer—when the adults of this country had been in the main vaccinated in infancy, and had been, unlike their forbears, little exposed since birth to the infection of small-pox. So that in the end, small-pox epidemics, when they have recurred, while affording among young vaccinated persons plenty of the anomalous cases, which early in the century had given rise to controversy, have afforded also in association with such cases, and mainly among older vaccinated persons, plenty of cases of undeniable small-pox, some of it having a fatal termination.

In the light of the above considerations, presumption may be thought to arise that disbelief by our forefathers in 'second' small-pox is likely to have had a very similar basis to disbelief by our more immediate ancestors in 'post-vaccinal' small-pox. Thus, our grandfathers, in the first quarter of the century, starting with the assumption that a person once protected was always protected, declined to accept eruptive malady occurring (in the face

of epidemic small-pox) in vaccinated persons as other than *spurious* small-pox ; and commonly their attitude in this respect was fortified by the circumstance that, few persons in those days being able to go many years after vaccination without getting exposed to small-pox, they rarely saw in vaccinated persons any but much modified small-pox. When, however, they did exceptionally see (say in countrymen, and by reason of long interval between vaccination and exposure to an attack by small-pox) unmodified small-pox in a vaccinated person, they straightway inferred that such person's vaccination had been *spurious*.

In just the same way, their own ancestors, starting on a like assumption (at a time, too, when nearly everybody was frequently exposed to small-pox), when they happened, as was perhaps not rarely the case, to see modified small-pox in the adult follow small-pox that in the person's infancy had left unmistakable pitting, are likely to have declined to regard this modified small-pox as other than *spurious*, and to have preferred to call it horn-pock, swine-pock, and the like. Again they, when they more rarely saw, say in countrymen at long intervals after reputed primary attack, unmodified small-pox in an adult said to have had small-pox (mild) in infancy or childhood, are likely forthwith to have dubbed such primary attack as *spurious* (chicken-pock, water-pock, and the like) ; and no doubt they were helped to so regard it if their patient bore no marks whatever of his primary small-pox attack.

Should it be contended that, if our great-grandfathers really did see (although they did not recognise them) plenty of 'second' attacks of small-pox, we ourselves of to-day ought to be seeing, where small-pox is epidemic, a good many second attacks, the answer is, that a century ago primary small-pox attack, whether severe or mild, was undergone habitually in early life, whereas nowadays primary attack is in the main in adult life. Not only, therefore, was there opportunity for an interval between first and second attacks longer on the average in our forefathers' days than now, but 'protection' by small-pox attack in their day, being usually obtained in early life, was, owing to further growth and development thereafter of the individual, less stable than with us, now that small-pox attack occurs for most people at an age when growth and development are complete.

PROTECTION BY NATURAL SMALL-POX

The protective power of natural small-pox—small-pox received by infection in the ordinary way—is distinctly greater than that conferred either by artificial variolation or by vaccination.

Second Small-pox.—But it is by no means absolute nor invariable, and, indeed, the evidences of second small-pox are as ancient as Rhazes, who states that it is possible for the disease to come not only twice, but thrice. Avicenna sets down second small-pox as a frequent occurrence. Gaddesden also mentions it. Fortestus in the sixteenth century records that his own son was thrice attacked, and that he had seen the same thing in many other patients. Mercurialis and others testify similarly, and Diemerbroek in the seventeenth century saw many instances of it (Moore). Maitland, the first medical man who practised inoculation in England, also knew of it. But in the eighteenth century it was denied by Mead, Dimsdale, and others, and Van Swieten opined that the *real* small-pox could only take place once. This qualification, as we shall see, is important. Coming to our own century, the possibility of repeated small-pox is again asserted and insisted on, and various collections have been made of references to second cases, notably by Professor Thomson¹

¹ *Historical Sketch respecting the Varieties of Small-pox.* 1822.

and Dr. Hennen, of Edinburgh, by Plocquet,¹ who gives a list of seventy-four reporters of such cases, by the Provincial Medical Association (239 cases), and by the Epidemiological Society.

Modified Forms of Small-pox.—But, after all, the difference of opinion between eighteenth-century writers on the one hand, and their predecessors and successors on the other, is mainly a difference of names. While there is ample evidence that second small-pox may be severe, confluent, and even fatal, yet the disease is, as a rule, so much modified that it has given room for dispute as to what is and what is not *real* small-pox. John of Gaddesden declared 'Item notandum quod variolæ sunt duplices, propriæ et impropriæ.' Sydenham wrote of genuine and bastard small-pox, and his contemporary, Morton, spoke of genuine and 'spurious.' To the latter sort many names were given, depending on the particular appearance of the pocks—as stone-pox, water-pox, wind-pox, sheep-pox, swine-pox, horn-pox, nirl-pox, &c.; and when we come to the writers who denied the existence of second small-pox, we still find Van Swieten talking of the first three of these forms—but not as *real* small-pox. In Scotland, the favourite terms were horn-pox, and nirl-pox.

It may safely be asserted that these names nearly always mean modified small-pox, and that in the great majority of cases the modifying agency was a previous attack of the disease, either by infection or inoculation. Some of the terms, as stone-pox, horn-pox, &c., would denote an eruption which never got beyond the papular stage, while water-pox and chicken-pox—which term was also used in this connection—would refer to a form in which the vesicular stage predominated. Of course, what we now know as chicken-pox may also often have been called water-pox, and sometimes swine-pox, though the other names could hardly have been applied to that disease. A curious feature in connection with modified forms of small-pox is that they sometimes became so established in a locality, and so fixed in type, that they prevailed as epidemics. Jenner discusses such an epidemic, and Sydenham describes an outbreak of variolous fever without eruption. Indeed, it is evident that when such fixity of character was temporarily obtained, the disease in its epidemic form attacked not only persons who had already had small-pox, but sometimes also entirely unprotected people, who in this way would get a considerable amount of protection. And where the modification of type was not very considerable, the variolous nature of the disease was sometimes clearly enough recognised, as by Sydenham, whose epidemic of variolous fever ran *pari passu* with another epidemic of unmodified and unmistakable variola. It was a common saying, as recorded by Wagstaffe, that there were some kinds of small-pox that a doctor could not cure, and others that a nurse could not kill. In the present century, with small-pox itself much less prevalent, and occurring at a higher range of ages, the evidences of second attacks are scantier, but every epidemic adds to them. Jenner, Munro *tertius*, Thomson, Cross, Seaton, Marson, &c., record many cases.

Opinions as to the frequency of second small-pox vary greatly. Curschmann² says the statements run from 1 in 250 (Eichhorn), to 1 in 10,000 (by Condamine), and Marson gives the fatality at 19 per cent., while the Epidemiological Society puts it as low as 8·3 per cent. These differences doubtless depend, as before, on the acceptance or rejection of more or less modified cases. But there can be no question that small-pox after natural small-pox is comparatively rare, and that a great majority of the cases are much modified. The chief factors in the production of a second attack, and

¹ Seaton's *Handbook of Vaccination*, p. 302.

² Ziemssen's *Cyclopædia of Medicine*.

in its severity or otherwise, may be stated as (1) mildness of the first attack; (2) its occurrence in infancy; and (3) a lengthened interval since the first attack. But there appears to be a small number of individuals who through constitutional idiosyncrasy are peculiarly susceptible not only to repeated but severe attack.

PROTECTION BY SMALL-POX INOCULATION

It was doubtless at an early period in the history of mankind that the idea was first conceived of trying to forestall and baffle a severe attack of a disease by the induction of a milder attack. Previous to reaching this point, other elementary conclusions on the subject of disease incidence must have been arrived at. The chief of these would be (1) that there were certain infectious maladies from whose attack comparatively few persons might expect to escape; (2) that some of these maladies were very unlikely to occur twice in the same person, or, in other words, that one attack had the power of preventing a second; (3) that where this was the case, a mild attack might be relied on to possess the prophylactic quality; and (4) that this mildness of type in one case was likely to induce similar mildness in cases infected from it.

In places where atmospheric infection was understood, the most rudimentary procedure in the direction indicated would probably consist in the intentional exposure of persons who had not already passed through the disease to infection from a favourable case of a prevailing distemper. In the present day such a practice is by no means unknown. In outbreaks of measles and scarlatina of the requisite gentleness of behaviour, we find many parents who believe that it is better for their children to commute the risk on the easy existing terms. In remote parts of the Highlands of Scotland, in the last century, children were sometimes put to bed with patients suffering from 'a good mild small-pox' as Monro terms it, with this end in view. A nearer approach to modern methods was found, also in the Highlands, in the practice of saturating woollen threads in the contents of small-pox pustules, and tying them round the wrists of the subjects whose infection was desired.

It is an interesting fact that perhaps all the really serious attempts which, up to quite a recent date, have been made to obtain subsequent immunity from a disease by inoculation of the virus, either of the same disease (as in small-pox inoculation) or of another believed to possess the necessary power (as in cow-pox inoculation) have had reference to the one malady, small-pox. Very likely the practice, as applied to this disease, had its origin in the occasional occurrence of accidental contact of small-pox matter with an open sore on the body of some nurse or attendant, and the observation of the trifling nature of the ensuing illness. If so, it is obvious that in ordinary fevers, in measles, in scarlatina, &c., there could be no such accidents, as these zymotics are not characterised by an eruption of vesicles or pustules containing an inoculable fluid. But in imitation of variolation, various attempts have been made by Home,¹ Wachsel, Speranza, Katona, Thomson and others to induce a mild attack of measles by a similar method.² Attempts of a like nature have also been made with regard to plague.³

Methods.—As has already been mentioned, the oldest accounts of small-pox inoculation come from Hindostan, where the practice was in the hands of the Brahmins (Holwell). Their method of operating was as follows.

¹ *Medical Facts and Experiments*, 1759.

² For an interesting account of these attempts, see a paper by Dr. Hugh Thomson in the *Glasgow Medical Journal* for June 1890.

³ Schultz, *op. cit.* Rees' *Cyclopædia*, article 'Inoculation,' edition 1819.

They used stored matter from inoculated cases of the previous year, and they practised only in the cold season—early in February as a rule. By friction with a dry cloth they caused a flow of blood to the capillaries of the skin on the outside of the arm. Then with a sharp instrument they made slight scratches, scarcely drawing blood, over an area of a groat or a sixpenny-piece. On this they bandaged a moistened pledget of variolated cotton. At the end of six hours the bandage was removed, and the pledget left to fall off of its own accord. They used the cold douche over the head and shoulders up till the time of the eruptive fever, and resumed it on the appearance of the pustules. A vegetable diet was prescribed, and the patients were freely exposed to the open air, this being accomplished where there was much fever by causing the patient to lie on a mat at the door. The eruption usually consisted of from 50 to 200 pustules, though the patient was looked on as pretty secure even if only a few pimples appeared round the inoculated part. All the pustules were opened and their contents wiped away by means of a linen rag dipped in warm water. In most respects the Brahmin method of what we would now call attenuation of virus, and of general treatment, was as nearly perfect as later experience ever made it, even in the last century in this country, and it was only after many years' elaborate blundering that the Eastern simplicity was finally returned to. In China, where next we hear of variolation, a much less satisfactory procedure was followed. It was called 'Sowing the Small-pox,' and consisted in fixing into the nostrils small-pox crusts, perfumed with a little musk, and wrapped in wool. According to Schultz, the African method was to draw a needle and thread through a mature pustule, and afterwards through the loose tissue between the thumb and the first finger.

In Persia, Armenia, Greece, &c., the practice is also of great antiquity, but may have originated in Arabia. When the subject began to be of interest in this country, the Circassian operation was in the hands of old women, who employed three needles tied together, and pricked the body in five places, in all of which matter was inserted. The practice was called 'Buying the Small-pox' owing, according to Moore, to some trifling present being given to the child from whom matter was taken. Woodville refers to a similar expression prevailing at Pavia, in Italy, and it is an extraordinary fact that after variolation came into vogue in England, it turned out that among the peasantry in South Wales¹ 'Buying the Small-pox' was not unknown either in name or in fact. The Welsh method consisted in either rubbing the matter directly into the skin or inserting it by means of pricks with pins. It would almost appear as if in some obscure way inoculation had travelled, like small-pox itself, along the northern shores of Africa into Southern Europe,² and thence extended northwards, where in out-of-the-way places it had lingered possibly for centuries, at the same time that it had entirely passed out of the knowledge of the larger centres of population. *Monro primus*³ records that even in the remote island of St. Kilda, 'the small-pox, according to Dr. Macauley, is communicated by rubbing the matter of small-pox upon the skin of the elbow-joint.'

Phenomena of Inoculation.—The phenomena of inoculation, as described by Trousseau,⁴ one of the latest writers who can speak from personal observa-

¹ Dr. Perrot Willan, in Jurin's *Comparison, &c.*, 1723.

² In Italy, according to De la Condamine, 'it was the custom from time immemorial amongst nurses to communicate small-pox to children by rubbing the palms of their hands with recent matter of small-pox.' (*Observations on the Different Kinds of Small-pox*, by *Monro tertius*.)

³ *Op. cit.*

⁴ *Clinical Medicine*, Sydenham Society's translation, vol. ii.

tion, are briefly as follows. At the seat of insertion of virus, a small red pimple appears on the second day. By the fifth day it has become an acuminate vesicle, but sometimes depressed in the centre. On the seventh day the vesicle has become a pustule, with an areola, which increases in extent up till the tenth day, the pustule also enlarging, becoming more depressed in the centre, and bluish in colour. Its edges are uneven (unlike *vaccinia*), and within the area of the areola from ten to twenty secondary pustules appear. The distinctive irregularity in shape of the vesicle, with its surrounding satellites, is shown in Ballhorn's and Stromeyer's plate reproduced by Crookshank,¹ but much more clearly in Pearson's plate, prefixed to his work published in 1802. The axillary glands are affected, especially on the ninth day. The pustule as a rule dries up in a fortnight, and successive crusts form and separate, but sometimes a deep slough forms, leaving in twenty or thirty days a misshapen cicatrix. Constitutional symptoms—headache, vomiting, &c.—begin on the ninth or tenth day, and from the eleventh to the thirteenth day the specific eruption shows itself, following the course of normal or modified small-pox.

In England.—In England the practice began to be talked of about the year 1714, through the publication in the 'Philosophical Transactions' of a letter to Dr. Woodward, of Gresham College, from Dr. Timoni, who, after studying in England, practised in Constantinople. Other accounts came from Pylarini, the Venetian Consul at Smyrna, and from Mr. P. Kennedy, an English surgeon who had been in Turkey. It was in April 1721 that variolous inoculation was first (within modern knowledge) performed in England by Mr. Maitland, Surgeon to the Ottoman Embassy. Lady Mary Wortley Montagu, the wife of the Ambassador, had had one of her own children operated on by Mr. Maitland in 1717, at Constantinople, and on returning to England she got a second child inoculated, and did much in advocacy of the procedure. In August of the same year Mr. Maitland was permitted to inoculate six condemned criminals who purchased their lives by submitting to the experiment. The operation as at first done by Maitland himself imitated in most respects that of the old Greek women, and was consequently accompanied by comparatively little danger to the individual. But theory soon stepped in, and the teachings of Eastern experience were subordinated to the speculations of the prevalent pathology. It was urged that the small-pox eruption meant the exodus from the blood of foul humours, and that if variolation were to give protection while at the same time producing only a slight eruption, this could not be accomplished except by making a deep and severe local wound, and establishing a free discharge of the peccant variolous matter from the system.

In New England inoculation had come into use also in the year 1721.² Two incisions were made, larger than were used for issues, and into these were inserted bits of lint that had been dipped in variolous matter taken indifferently from discrete, confluent, or inoculated cases. The sores were poulticed with warm cabbage leaves, fever was treated by vomits, bleeding, &c., and to encourage the eruption blisters were applied. Little wonder that when the eruption came the pustules were often several hundreds in number, 'frequently more than what the accounts from the Levant say is usual there.' From the sores so treated there was 'a plentiful discharge,' and as to their drying up, 'the later the better,' was the prevalent opinion.

Dr. Nettleton, of Halifax, was one of the first to follow up inoculation in.

¹ *History and Pathology of Vaccination.* Lewis, London, 1889.

² Henry Newman, *Phil. Trans.* XXXII., year 1724.

England.¹ For preparation he advised purging for children, and vomiting and bleeding for adults, and as to the incisions he taught that 'when they were made pretty large, the quantity of matter discharged afterwards at those places was greater: and the more plentiful that discharge the more easy the rest of the symptoms generally are.' During the fever, the wounds 'continued to be very sore and swell very much, so as to appear very large and deep, and to discharge a great deal of matter,' and 'the longer they did so the better.' Jurin expressed similar opinions.²

Partly in consequence of such methods, mishaps of various sorts, severe eruptions and fatalities, began to be heard of. It was also discovered that the inoculated disease was capable of spreading small-pox by infection. Such facts, along with a good deal of clerical and medical lampooning and religious prejudice, soon brought the practice into desuetude, so that at the end of eight years only 897 inoculations were recorded, with a total of seventeen deaths.³

About the year 1740 inoculation began to revive, and in the manner of operating a gradual improvement set in, so that the mortality was reduced to one death in from 800 to 600 cases. Even so late, however, as 1761, we find Kirkpatrick describing a method of operation differing nothing in severity from that pursued by Nettleton.

The Suttonian Method.—But under Daniel Sutton the acme of safety was attained. He practised at Ingatestone, in Essex, and is said to have operated, in 1764–6, on nearly 14,000 persons, in addition to 6,000 inoculated by his assistants. By the year 1796 he alleged that his cases had reached 100,000. His proceedings were enveloped in much mystery, and accompanied by a good deal of quackery. Secret powders and potions were given, and great virtues were attributed to these, and to his own special skill and discrimination. But, apart from irrelevancies, there seems no doubt that he carried out most of those principles of procedure which in our own day have rendered vaccination practically harmless. He used clear fresh virus, applied it directly from case to case by means of a lancet, avoided threads, cotton pledgets and plasters, and left the little wounds to heal in peace. In addition he carried out the 'cool regimen,' which the Brahmins had used in their inoculation practice, and Sydenham had recommended in the treatment of natural small-pox. At the same time, Sutton wisely avoided the bleedings enjoined by that author. As to his results, Houlton, a clerical mercenary, whom he employed to advertise him, stated that twenty or thirty pustules formed in his practice 'a full burthen,' but Chandler, another Suttonian, said that he had seen hundreds of pustules. The mode of practice was reported on by Sir George Baker, and in a general way adopted and advocated by many operators, chief of whom was Baron Dimsdale. Much discussion took place among Sutton's contemporaries as to the cause of his success. Schultz thought the preparation the main point. Baker attributed absence of severity to the free exposure to cold air. Glass⁴ thought that by the 'critical sweat' induced by Sutton's medicines, 'a considerable part of the variolous matter or poison, when the eruption proves slight, is discharged and carried off.' Chandler⁵ thought the material point was 'the taking the infecting humour in a crude state, before it has been, if I may be allowed the expression, ultimately variolated by the succeeding fever;' and with this aim in view, in his own practice, he took matter from the seat of inoculation—

¹ *Phil. Trans.* XXXII.

² Jurin's *Account, &c.*, 1724, p. 15.

³ Scheuchzer's *Account of the Success of Inoculating the Small-pox*, 1729.

⁴ *A Second Letter from Dr. Glass to Dr. Baker*, 1767.

⁵ *An Essay towards an Investigation of the present Successful and most General Method of Inoculation*, by B. Chandler, Surgeon at Canterbury. London, 1767.

the 'mother pustule' as it is now conveniently called—before the general eruption appeared. Dimsdale,¹ thought that the success consisted chiefly in using 'recent fluid matter,' though he 'gives a preference to the matter taken during the eruptive fever.' He always liked to see a good areola, 'a circular or oval efflorescence . . . surrounding the incision, and extending sometimes nearly half round the arm, but more frequently to about the size of a shilling,' which appearance 'is a very pleasing one.' In his later years, Sutton himself confessed that he had attached more importance to choice of matter than to any other part of his system. Timoni had reported that the Greek women at Constantinople were indifferent whether the matter for inoculation was taken from the mother pustule or from the secondary eruption. This opinion was shared in by Blake² and by Schultz,³ and was evidently also accepted by Woodville, the historian of inoculation,⁴ who declares that Dr. Frewen, of Rye, was the first 'to discover' that the choice of variolous matter 'was of little importance.'

There was just one danger attached to efforts after safety—namely, that the operation might be whittled down to absolute nothingness, as we sometimes see indeed with regard to vaccination in the present day. Gatti,⁵ a famous Italian inoculator who practised in Paris, probably sometimes reached this point by simply pricking the loose cuticle between the thumb and the forefinger with a fine needle moistened with a very minute quantity of lymph. But when a patient of his, the Duchess de Boufflers, afterwards took small-pox, he explained that he had looked on a merely local result of variolation, not as conferring protection, but as indicating insusceptibility. But the procedure which took firmest hold of the profession in England was that described and followed by Dimsdale, whose work on the subject already quoted went through at least seven editions, and whose practice, as Woodville tells us in 1796,⁶ held the field up to that year—the year which witnessed the performance of Jenner's first cow-pox inoculation. It is evident, however, that all through the century there never was complete unanimity as to the details of inoculation, and that much variety prevailed, alike in the manner of operating, in the mildness or severity of the induced disease, and in the protection afforded against subsequent attack by small-pox.

As to the immediate effect of the operation on the individual, mildness of type shown by absence of eruption continued to be looked on as exceptional, though academical discussions took place as to whether non-eruptive cases could be considered as protected against small-pox; Black, for example, arguing on the one side and Kirkpatrick on the other. Maitland, in the second edition of his 'Account of Inoculating the Small-pox,'⁷ mentions a case in which he got over 1,000 pustules. In 1758, Dr. Daniel Cox records as 'peculiar' a case in which there was no eruption, and Baker writes as if more or less eruption were a matter of course in Sutton's hands. Woodville, also, in the course of his discussion of supposed vaccinal eruptions, makes a similar assumption regarding inoculation; and, in 1802, George Bell, of Edinburgh, said that 'by inoculation pits are often produced over the whole surface, so as to disfigure the most beautiful features.' Goldson, a critic of vaccination, looked on a hundred pustules as a mild result of variolation, and John Ring, an

¹ *The Present Method of Inoculating for the Small-pox*, by Thomas Dimsdale, M.D. Third edition, London, 1767.

² *A Letter to a Surgeon on Inoculation*, by John Blake, Surgeon at Bristol. London, 1771.

³ *Op. cit.*

⁴ *History of Inoculation*, 1796, by William Woodville, M.D.

⁵ *New Observations on Inoculation*, by Dr. Gatti, Professor of Medicine in the University of Pisa. Translated by M. Maty, M.D. London, 1768.

⁶ *Op. cit.*

⁷ London, 1722.

advocate on the other side, says that as to small-pox, 'a case without eruptions is so rare an occurrence that a physician from the country lately mentioned it at the Lyceum Medicum Londinense as an extraordinary phenomenon, having once met with it in his practice.'¹ Brown, of Musselburgh, who himself urged variolation in addition to or instead of vaccination, also looked on an eruption as its normal result.² In the Report of the Newcastle Dispensary for the year 1800 a table is appended of variolations performed from 1786 to 1800, when the mild methods were in vogue. In 8,181 cases only 217, or 6·8 per cent., were without eruption. Inoculation was, indeed, an operation whose results never were under the control of the operator, so that, in 1802, Bell wrote that 'it is known that many die annually from it.'

Artificial Attenuation of Variolous Virus.—Attempts have repeatedly been made to reduce variola to vaccinia without the intervention of the cow. Trousseau made, along with Dr. Delpech, a careful series of experiments with human small-pox, the object being to reduce the eruption till it consisted merely of the mother pustule. In some children he got the result that 'the pustule of inoculation was alone developed, and that around it there were little pustules, its satellites.' 'But in others, in which the very same virus had been employed, there were general eruptions, and, worse still, communication of small-pox to non-inoculated persons.' 'In one case . . . the small-pox resumed all its original violence, after having passed through a succession of individuals in a series of inoculations.' Owing to such occurrences he was compelled to discontinue his experiments and to 'renounce inoculation.' Long before Trousseau's day, Dr. Adams,³ Woodville's successor at the Small-pox Hospital, had experimented with a mild sort of small-pox, which he calls pearl-pox. It appears from his records as if from the very beginning, and without any series of cultivations, this 'pearl-pox' gave locally the 'vaccine appearance,' and in the course of several removes he had some cases without any general eruption. Still earlier, in 1789, Jenner, quietly and thoughtfully working around his favourite subject, inoculated his first-born child with matter from the so-called 'swine-pox,' evidently recognising the variolous nature of the disease; and subsequently he repeatedly applied to the case the variolous test, which later on played so important a part in the early history of vaccination. Guillon⁴ experimented in another direction: he inoculated small-pox in children or youths (*écoliers*) who had undergone vaccination, and declares that the results were indistinguishable from those of vaccination. In a subsequent attempt, however, some cases had eruptions.

Insusceptibility to Inoculation.—As in natural small-pox and in vaccination, a few people appeared to be insusceptible to variolation. In the first 897 cases reported by Jurin and Scheuchzer, thirty-nine could not be infected, and the addition to these of the cases that had been done in New England gave forty-five failures in 1,179 operations (Kirkpatrick). On the other hand, some people were found to be excessively susceptible, and Moore⁵ records the case of a lady who was successfully inoculated seven times.

Alleged Evil Results of Inoculation.—Evil results were attributed to inoculation, as to vaccination in later times. Schultz writes that 'Dr. Cantwell believes that inoculation is the occasion of the spotted fever, rash, consumption, and nervous complaints, which are so rife in England.' Dimsdale

¹ Ring, *op. cit.*, pp. 104-5.

² *Inquiry into the Anti-variolous Power of Vaccination*, 1809, pp. 29-30.

³ Trousseau's *Clinical Medicine*, vol. ii., pp. 91-2.

⁴ *Journal Général de Médecine*, 1827.

⁵ *Reply to Anti-vaccinists*.

says that it was occasionally followed by secondary eruptions, ophthalmia, erysipelas, axillary abscesses, ulcerations, sloughs, &c.; and Blake (1771) advocated the mild method of operating, to avoid 'deep phagedenic ulcers at the places of insertion, which sometimes prove very tedious and difficult of cure.' From such materials, and in spite of the verbal resemblance, no anti-inoculator seems to have thought of suggesting that small-pox was an analogue of great-pox, though, as a matter of fact, it was debated whether variolation was not capable of conveying the venereal disease (Blake).

Protective Power.—The amount of protection which variolation gave against natural small-pox was, on the whole, very considerable. But various cases of natural small-pox so early as two or three years after inoculation are related by Kirkpatrick, Ring, Lettsom, and others. In 1746, a Dr. Pierce Dod objected to inoculation on account of such failures,¹ and in particular it is recorded by Moore that Woodville 'often said that patients were frequently brought into that (the Small-pox) hospital, covered with small-pox, who declared that they had been inoculated in their infancy, and had learnt from their mothers they had had the disease.' It is important to recollect that variolation, unlike vaccination, was not at all confined to the first few months of life. The operation was done at any age among those who had not had small-pox. Hence there was no opportunity for the question of re-inoculation to attain such prominence as re-vaccination has done in our own day. But the passage just quoted suggests that, under similar circumstances, a similar necessity for the repetition of the operation would very likely have arisen. Along with unquestionable cases of typical small-pox, in spite of previous variolation, we have throughout the inoculation period, as has already been indicated, all those eruptions which were variously described as stone-pox, water-pox, wind-pox, swine-pox, &c. &c.; and it need not be doubted that in very many such cases the system was still partly protected against the full effect of the small-pox virus, and that the protecting agency was artificial variolation.

Local Variolation.—It may be noted that the efficacy of the operation at the time of its performance could be ascertained in a way which, when vaccination was introduced, formed the basis of Bryce's well-known test. Blake states that where the signs of infection were dubious several days after inoculation, he repeated the operation, and that (unless done so late as within thirty or forty hours before the eruptive symptoms due to the first inoculation) 'the last punctures rise as well as the first, and in a few days are full as forward as, and proceed through the succeeding stages of maturation and exarescence with the first.' This shortened cycle of inoculation was looked on as evidence that the system was protected against the disease. Even in persons permanently protected by either inoculated or natural small-pox, there hardly ever seems to have been any difficulty in producing a purely local pustule of variola. Maitland, Kirkpatrick, Schultz, Maty, and others mention such occurrences. Pustules (due to contact) on the arms and faces of nurses in charge of children suffering from small-pox were common. Willan,² in speaking of such occurrences, says: 'The matter contained in the pustule thus excited, either after the small-pox, or after vaccine inoculation, is found capable of communicating the small-pox to those who have not before been affected with that disease.' Mr. Samuel Hill, Surgeon, Portsea, in 'Experiments proving Vaccination or Cow-pox Inoculation to be a permanent security against Small-pox,' says, regarding a local result obtained in certain cases of variolous inoculation after the system had been

¹ *Med. Phys. Jour.*, July 1800.

² *On Vaccine Inoculation*, p. 15. London, 1806.

protected by vaccination, 'I have no doubt but that I could with lymph from the punctures have given the small-pox to any one susceptible of that disease.' Mr. Richard Dunning, of Dock, Plymouth,¹ speaks of 'irritability in the skin, which we so often see excited by the local application of variolous matter in persons who have passed the small-pox.' When inoculation was first introduced, similar facts were mentioned. Maitland caused one of the Newgate prisoners, whom he had been allowed to inoculate, to nurse and sleep with a small-pox patient, and he observes: 'I once saw some heats and little pimples upon her, as nurses commonly have under such confinements.' Similarly, 'Mrs. Heath also, by being night and day constantly with her children, and always embracing and caressing of 'em, tho' she had had the small-pox many years before, struck out in several pimples or pustules on her face and hands, but without anything of sickness or disorder, which, we know, has often happened to nurses, by attending people in this distemper, and even by washing their linen.' In 1777, John Mudge, surgeon, Plymouth,² wrote thus: 'Whenever I have inoculated a patient at a distance, whom for that reason I could not conveniently see for some days after, in order to know whether the matter applied, more especially if not quite fresh, was sufficiently active, I have usually put it to a similar scratch on my own hand, which if it was efficacious, but not otherwise, never failed to inflame, and of course informed me whether or no, on my next visit, it would be necessary to provide myself with fresh matter for my patient.' Perhaps the most remarkable example of local susceptibility combined with constitutional protection is the case, evidently well known in Edinburgh, of a surgeon who regularly kept up matter for small-pox inoculation by means of a local pustule renewed over and over again on his own arm.³ In the present day a somewhat parallel condition of things prevails as to vaccination. People who are quite insusceptible to small-pox by atmospheric infection yet, in very many cases, develop a pustule by vaccination, though the pustule is often irregular in appearance and course. So that susceptibility either to variolation or vaccination is by no means equivalent to liability to small-pox by infection. Intentionally to perform a second inoculation years after the first seems hardly ever to have been thought of, but a case is recorded by Dunning, of Plymouth, in which a Dr. Stewart, whose daughter had been inoculated 'when young,' with, as a result, an eruption of two hundred pustules, re-inoculated her six years later, and, to his surprise, she suffered more from the second than from the first operation, had a more severe illness, and a considerable eruption all over her body (Moore's 'Reply,' &c.).⁴

Profit and Loss by Inoculation.—We come now to the question of profit and loss by inoculation. It is asserted, on the one hand, that the inoculated went about spreading the disease broadcast; and, on the other, that the number of persons saved from natural small-pox by inoculation was so great as to more than counterbalance the cases in which the disease was propagated by this means. It is quite clear that the amount of infectious material given to the atmosphere from the average eruption resulting from one of Dimsdale's operations was very small indeed compared with the quantity given off by a person suffering from a confluent attack of the natural disease. It may also be held that as the type of the inoculated disease was usually very mild, the type which it set up, even by atmospheric infection, would also

¹ *Minutes of Experiments, &c.*, p. 1

² *A Dissertation on the Inoculated Small-pox.* London, 1777.

³ *Report of the Surgeons of the Edinburgh Vaccine Institution*, 1809; also Ring's *Reply to Goldson*, p. 18.

⁴ See also Woodville's *History*, p. 184, and Ring's *Treatise*, p. 121.

tend to be mild, and that the deaths resulting would be comparatively few ; and that this fact would tend to lower the fatality of the disease in the past century, as compared with its fatality among the unvaccinated in the present century. Indeed, this thesis might be carried to the point of asserting that, as small-pox, independently of inoculation, was a disease comparable in prevalence to measles and scarlatina in the present day, inoculation, if it originated outbreaks of a favourable type, was really saving life by supplanting more formidable epidemics. But it is very difficult to tell on which side the balance should be struck. As the writer has shown elsewhere,¹ the evidence is conflicting and the decision doubtful. In places where very few were inoculated, more harm than good may have been done, but there can hardly be a doubt that where inoculation was very generally performed, it was of immense benefit : so that, as compared with its very general performance towards the end of last century, the diminution in the practice of inoculation which took place early in this century may have had a tendency rather to spread than to lessen the prevalence of small-pox. In Scotland there is abundant evidence that inoculation lessened small-pox in many places, and indeed increase of population is attributed to the practice.²

An objection which militated strongly against the universal adoption of inoculation was the danger to life which attended it. The fatality rate varied in the hands of different practitioners, and at different epochs, but even under the Suttonian system the deaths, though not approaching to 1 in 100, as in the earlier years, would be sufficient to create constant and well-founded anxiety ; and in addition to the mortal cases, there would be numerous severe attacks, giving rise to much anxiety and leaving permanent disfigurement or other serious sequelæ. With the introduction of vaccination, variolous inoculation became less and less prevalent, and in 1840 it was made illegal, owing to the possibility of its spreading infection among the unprotected.

PROTECTION BY VACCINATION

Jenner.—Somewhere about the year 1768, a young woman called for advice at the surgery of a country doctor in a Gloucestershire village. Referring in the course of conversation to small-pox, she remarked, 'I cannot take that disease, for I have had cow-pox.' The remark was heard by the doctor's apprentice, Edward Jenner, and sank deep into his mind. When he himself had become a doctor, it formed the subject of much private cogitation, of occasional conversation with a friend, of discussion at 'Medico-convivial' meetings, of numerous inquiries among dairy folk, and ultimately of experimental investigation. The experiments consisted mainly : (1) in applying the test of small-pox inoculation to persons who had, at some time in their lives, suffered from cow-pox ; (2) in vaccinating (though that word had not then been coined) directly from the cow's teats ; and (3) in arm-to-arm vaccinations. The results were published in 1798 in 'An Inquiry into the Causes and Effects of the Variolæ Vaccinæ.' In April 1799 he issued 'Further Observations on the Variolæ Vaccinæ or Cow-pox,' and in 1800, 'A Continuation of Facts and Observations relative to the Variolæ Vaccinæ or Cow-pox.' He subsequently wrote short papers published in the 'Medical and Physical Journal,' besides carrying on an enormous correspondence with medical men and others all over the world.

His views were very briefly as follows. Many human diseases may have their origin in those of domestic animals. The teats and udders of cows

¹ *Vaccination Vindicated*, pp. 156-80.

² Sir John Sinclair, *op. cit.*, vol. ii. p. 569-70, and vol. v. p. 199.

are subject to a variety of eruptive maladies, indiscriminately called cow-pox by the dairy people. One of these has the power, when inoculated accidentally (casually) or intentionally on the human subject, of permanently preventing an attack of small-pox. This pustular disease is true cow-pox, the others are spurious. A spurious disease may also be produced by inoculating with decomposed cow-pox matter, or matter taken from an ulcer following cow-pox, or a peculiar morbid matter generated by a horse. True cow-pox is derived from an eruptive disease of the horse, called horse grease, and possibly human small-pox was originally derived from the same source. Cow-pox (which, with the insight of genius, he named *variola vaccinae*), either on the bovine or the human animal, is not spread by atmospheric infection. It is not followed by eruptions. On the hands of dairymaids, accidentally inoculated, it appears as circular vesications, bluish in colour, with depressed centre, and surrounded by an inflamed areola, the appearance being that of a pearl on a rose leaf. The axillary glands become affected, and there are febrile symptoms. The sores are apt to ulcerate unless attended to. The most material indisposition does not arise primarily from the first insertion of the virus, the worst symptoms being due to inflammation and irritation of ulcers, where these occur, as in the casual disease. It is, therefore, only an accidental circumstance which can render cow-pox a violent disease. The matter of horse grease is most active before it becomes purulent. Similarly in small-pox inoculation, done for its own sake, pure pus will probably entirely fail to convey the disease; and if old or putrefying matter be used, which has been stored, either in phials or on threads, &c., and carried about in the warm pocket, though local inflammation and even eruption may be set up, the constitution is not always protected from future small-pox. Similarly again, in small-pox inoculation done as a test for the efficacy of cow-pox, fresh limpid virus should be used, and should be applied directly so as to avoid confusion as to the meaning of the result obtained. Once more similarly, in vaccination only fresh clear lymph should be used, taken before the formation of the areola. Children suffering from herpes, &c., should not be vaccinated.

Following almost immediately on Jenner's 'Inquiry,' Dr. George Pearson published 'An Inquiry concerning the History of the Cow-pox.' He had been stimulated to the investigation by Dr. Jenner's work, and its result was to support Jenner's practice, Pearson having found quite a number of doctors in the dairy districts who, in their inoculation practice, had met with case after case in which cow-poxed milkers were found insusceptible of small-pox. From Ireland, Dr. Barry, of Cork, sent him similar testimony. But Pearson dissented from Jenner's opinion that cow-pox had always its origin in horse 'grease' or horse-pox, and in this dissent he has in later days been followed by the whole medical profession. In November 1798, however, Tanner, a veterinary student whom Jenner knew, inoculated a cow with horse-pox and raised lymph 'which proved efficacious in infecting both human subjects and cattle;' ¹ and the well-known experiments by Loy, of Whitby, published in 1801, gave evidence that the horse is subject to a disease capable of giving rise to cow-pox in the bovine animal. It is passing strange that at this day, when Jenner is being attacked in his vaccination practice where he has up till now been generally deemed invulnerable, those parts of his system which have been looked on as the weakest should be in a fair way to be proven strong. For recent writings give a general support to the views of Loy, who held that there were different diseases classed under the term horse-grease, and that one of these was true horse-pox, which, when

¹ Baron's *Life of Jenner*, vol. i. p. 249.

inoculated on the cow, showed itself as true cow-pox. And, similarly, modern inquirers are tending more and more to the view that from cats, poultry, cows, and other such animals we may receive diphtheria, tubercular diseases, and scarlatina.

Dr. Woodville.—Dr. Pearson and Dr. William Woodville, Physician to the Small-pox Hospital, were the first to follow up the Jennerian practice on a large scale. They obtained their first lymph from London cows in January 1799, Woodville's supply being from an outbreak of cow-pox in a dairy in Gray's Inn Lane, and Pearson's partly also from cows belonging to a Mr. Willan in Marylebone Fields.¹ Jenner got some of Woodville's lymph, and in the spring of the same year a supply was obtained for his use at North Nibley, in Gloucestershire, where cow-pox had appeared; and afterwards, in April, he got lymph from a dairy in Kentish Town. The separate history of these stocks is not recorded. A fact that has been entirely overlooked in recent discussions is that within the first year or so of his beginning to vaccinate, Woodville often went back to the cow for fresh lymph, and he and Jenner and Pearson must have distributed various vaccine stocks throughout the world without any record being kept of their exact genealogy. Woodville states, for example, that he got matter from Jenner from the Kentish Town stock, and used it in the hospital. And in his 'Observations on the Cow-pox,' published in 1800, he writes: 'At various times I procured the vaccine virus, as produced in different cows, and with it inoculated patients in the hospital; but the effects of all the matter I tried were perfectly similar, and pustules proved to be not less frequently the consequence of these trials than of those made with the matter formerly employed.' This brings us to a curious phase in the history of vaccination. Woodville did a great many of his inoculations at the Small-pox Hospital, and this fact gave rise to much confusion. In a number of his cases there appeared a general pustular eruption, which he (in his 'Report of a Series of Inoculations for the Variolæ Vaccinæ' published in 1799) set down as belonging to the phenomena of cow-pox, and it is evident that matter was occasionally taken from these eruptions and sent out as cow-pox. Jenner, however, tersely observed of such eruptions, 'Where variolous pustules have occurred I believe variolous matter to have occasioned them.'² Woodville himself ultimately came to adopt the view that vaccination never did produce such eruptions, and he stated this to a Select Committee of the House of Commons in 1802. But in the interval there had been much heartburning over the question, and in some cases matter which had been sent out from the hospital as vaccinal was followed by eruptions apparently variolous. The main cause within the hospital must have been that the patients had been infected by the variolous atmosphere of the place. In the case of Ann Bumpus, for example, from whose arm Jenner himself received lymph, variola and vaccinia were present simultaneously, and there were 800 pustules. But a careful examination of the facts shows that Jenner got his lymph from the arm two days before the general eruption appeared, and in Gloucestershire this lymph gave results indistinguishable from those of other vaccine lymph used contemporaneously with it. And freedom from eruptions was the general experiences of those who tried Woodville's lymph, both at home and abroad. Woodville himself said, 'Respecting those to whom I have communicated the infection out of the hospital, or among my private patients, I have not yet met with one instance in which variolous-like pustules took place;'³ and this was exactly the experience of Colon, of Paris, in using Woodville's lymph there.⁴

¹ *Report of Vaccine Pock Institution*, 1803.

² Lettsom's *Observations on the Cow-pock*.

³ *Med. Phys. Journal*, Feb. 1800.

⁴ Ring, *op. cit.*, pp. 62-3.

So, too, Pearson distributed lymph to about 200 practitioners, and he records that with two or three exceptions no general eruption took place;¹ nor did De Carro, of Vienna, the chief vaccinator there, ever see eruptions from the lymph with which he had been supplied by the English operators.

In one other respect did Woodville's first results differ from Jenner's. The local symptoms were milder. There was no tendency on the part of the vesicle to end in an ulcer, the inflammation was less extensive, and there was less implication of the axillary glands. In other words, Woodville's lymph was very free from the effects on it of the rough manipulations of the milkers, and the admixture with the filth of the cow-houses. The pus and inflammatory products which are apt to be inserted along with direct cow-pox virus are extraneous substances, with which, unlike Jenner, Woodville does not appear to have been troubled; and very likely this was due to a circumstance to which Dr. Creighton (though from a very different standpoint) has called attention: that he got his first stock of lymph at an early stage, both of the disease in the cow-house and the vesicle on the milkmaid's hand.

The Variolous Test.—In the early years of vaccination, the variolous test was very extensively employed, and the literature of the time (especially the 'Medical and Physical Journal') abounds with evidence of the efficacy of vaccination in preventing subsequent variolation. Willan, writing in 1806,² mentions that from personal inquiries among eighty-six medical men whom he had met in the course of the previous three months, he had been made aware of over 18,000 vaccinations done by them, all the cases having remained free from small-pox, 'though most of them were afterwards either inoculated with variolous matter, or exposed at different periods to contagion.' These did not include Woodville's cases. At the same time, it was no uncommon thing for parents, hesitating between the old practice and the new, to have some of their children vaccinated and some variolated; and Willan³ states that Mr. Wachsel, of the Small-pox Hospital, had noted this 'in more than an hundred families, without observing one instance of variolous fever and eruption among the vaccinated children.' It is to be kept in mind that the practitioners of the period were intimately acquainted with all the phenomena of small-pox inoculation, and that their usual preference for the Suttonian practice, with its mild results, would lead them to observe the slightest evidence of successful variolation following vaccination; and yet their testimony, not only in England, but wherever all over the world vaccination found its way, was practically unanimous that the vaccinated exhibited no susceptibility to the small-pox poison. This was the verdict of persons acquainted with the results of testing variolation by a secondary and supplementary insertion of variolous matter, who, when they came to test their vaccinations in similar fashion, obtained a precisely similar local result. In many cases indeed there was no result. In others, there was a very early inflammation, following almost directly on the insertion of the virus, and lasting only two or three days, which, when variolous inoculation was done for its own sake and not as a test of vaccinal success, had always been considered an evidence of failure, the true symptoms of infection being of later occurrence.⁴ Pearson in this country, and Ballhorn and Stromeyer on the Continent, published plates showing side by side the appearances at successive stages of inoculated variola and inoculated vaccinia, and demonstrating that though there was a notable generic resemblance, yet the specific characters were so distinct that, independently of the variolous eruptions, there was really no possibility of confusion between

¹ *Med. Phys. Jour.*, Oct. 1799.

² *On Vaccine Inoculation*, p. 14.

³ *On Vaccine Inoculation*, p. 18.

⁴ Samuel Hill, *op. cit.*

them. Needless to say, if all Woodville's lymph had been variolous, and if it had been the only stock in use, no such plates could have come into existence. And Jenner, Aiken, Bell, and others published plates showing vaccinia exactly as we know it in the present day.

In spite of much and angry opposition, vaccination made its way rapidly in professional favour, and, in view of the facts, it could not well be otherwise. Every medical man who practised it and tested it for himself, as did the eighty-six whom Willan writes of, was bound to become its advocate. As a disease it was mildness itself, and the proof abounded that it gave protection against what was in their generation the greatest scourge of the human race, and that it did so, moreover, without the risk to others that had been inseparable from small-pox inoculation. At the same time, the actual demonstration of its powers, afforded by every test, would fall in with what they had read in 'Pearson's Inquiry,' regarding the strong faith in its virtues existing among both medical men and laymen in the dairy districts.

'Spurious' Small-pox and Cow-pox.—Much fault has been found with Jenner for speaking of 'spurious small-pox' and 'spurious cow-pox.' But, as a matter of fact, spurious small-pox had been discussed long before vaccination was heard of—by Bond, of Philadelphia, in 1784; by Nieldt, of Halle, in a monograph on the subject, in 1792; by Hufeland, in 1798; and by others (Thomson). Bond had given the name 'spurious' to the non-protective outcome of the inoculation of putrid small-pox matter, and Jenner gives exactly parallel causes of failure in the case of cow-pox. Ceely, forty years after Jenner's writing, mentioned the yellow-pock, the bluish- or black-, and the white-pock, as part of a list of 'eruptive diseases and spurious pocks,' to which milch cows in the Vale of Aylesbury were subject. And quite lately we have had some of these 'spurious' eruptions on the cow's teats investigated by Klein, Power, McFadyen, Crookshank, and others. It is more than likely that Jenner sometimes attributed to 'spurious' cow-pox, failures to protect against small-pox, which we now have learned to be incidental to vaccination. This would especially be the case in his later years (he died in 1828), when the protection afforded by vaccination, originally efficient, had in some instances worn out, and before the need for re-vaccination had been recognised. But if we look to Jenner's early appreciation of the importance of using lymph before the areola appeared (his golden rule), to his refusal to vaccinate children suffering from skin eruptions, and to his interpretation of the eruptions that occurred in Woodville's practice, we must conclude that though he did not know all that is now known regarding vaccination, he was far quicker and displayed far more wisdom in arriving at correct conclusions on the essential parts of the practice than his London co temporaries with their immensely greater fields of observation.

Small-pox after Vaccination.—It is to be remembered that for a decade or two after the introduction of the practice all vaccinations would be comparatively recent: time had not modified to any appreciable extent the protection afforded to those who had undergone the operation; and hence the publication by Goldson and others of a few instances in which vaccinated persons had taken small-pox (the vaccination then consisting only of a single vesicle less than one third of an inch in diameter) was so contrary to general experience that little heed was paid to them, though they contained the first hint of what we now know to be a truth, that the protection afforded by vaccination tends to wear out with lapse of time. Such occurrences were set down to imperfect or spurious vaccination, or the variolous nature of the disease was questioned, or when the fact of small-pox was admitted, it was pointed out that parallel facts could be cited, of variolous inoculation and even of natural

small-pox itself failing to protect against a second attack. All these replies were quite to the point, but we would now say that they did not cover the whole ground, as it is proved that the protection by vaccination is less complete and less enduring than was believed in the early days. It is probable that this loss of protection through advancing years is in part, at least, the explanation of such small-pox statistics as those of Copenhagen. In the years 1787-1807 the annual deaths averaged 284 from the disease. In 1801 vaccination was introduced, and a Royal Commission on Vaccination appointed and maintained till 1825, when the Board of Health took its place. The deaths from 1802 to 1810 fell to 18 per annum on the average. Then followed thirteen years (1811-28 inclusive) without a single death. But in 1824—and this is the point of interest in the present connection—the fatalities recommenced, and continued, irregularly, till 1850, with which year the table concludes. For the years of this period 1828-50, the deaths were as follows : 41, 12, 29, 4, 1, 29, 8, 0, 8, 19, 26, 484, 81, 1, 2, 0, 2, 0, 85, 111, 88, 7, 0, 0, 2, 7, 0.

The mitigation of small-pox by vaccination was observed almost as soon as the first cases of post-vaccinal small-pox occurred. In the year 1807 the Royal College of Physicians gave formal expression to the doctrine. The College reported that 'where small-pox has succeeded vaccination, it has been neither the same in violence nor in the duration of its symptoms, but has, with very few exceptions, been remarkably mild, as if the small-pox had been deprived by the previous vaccine disease of all its usual malignity.' As we will see further on (p. 445 *et seq.*), the truth of this statement has been amply confirmed by subsequent observations.

Ceely, Estlin, Badcock, and Bousquet.—About fifteen years after Jenner's death, a new spirit of inquiry seems to have sprung up regarding cow-pox as a source of vaccine lymph. It was believed by some observers that the vesicles produced at this period were not at all comparable with those of earlier times, and that the protection afforded was not so great. How far there was a real degeneration due to neglect of the rules of vaccination as laid down by Jenner, and how far there was merely an opinion founded on the occurrence of post-vaccinal small-pox resulting from the want of revaccination, it is impossible now to say. Mr. Estlin, of Bristol, in 1837-38, Mr. Ceely, of Aylesbury, in 1838-40, and Mr. Badcock, a chemist in Brighton, in 1840-41, and on the Continent, M. Bousquet in 1836, were the principal investigators. Estlin experimented with lymph obtained from an outbreak of cow-pox in Gloucestershire, and Bousquet originated what has since been known as the Passy stock.

Their early results were what might have been expected. Estlin's stock began with matter taken on the eleventh day from the arm of a child that had been domestically inoculated from a milker's hand; while Bousquet,¹ who held the opinion that 'lymph which begins to get turbid,' taken from pustules surrounded by a 'large and vivid' areola, with 'the subjacent tissue much infiltrated,' is yet 'none the less suitable for inoculation,' began by using 'thick white purulent matter . . . as if it were an abscess discharging;' and, lax as were his views regarding selection of lymph, even he was impressed with the fact that 'in a few hours all chance would have been lost' of getting any inoculable fluid at all. With such materials and with such views, the wonder is, not that, in the early removes there were occasionally troublesome inflammations and other such sequelæ, but that complications were so few. The worst results that Estlin could discover from his lymph occurred in a series of cases in Somersetshire,

¹ Crookshank, *History and Pathology of Vaccination*, vol. ii.

where among sixty children (of whom one died, though the connection with the operation was doubtful), four had 'erythematous eruptions of a violent character,' two had 'highly inflamed ulcerated arms,' one had 'severe erysipelas,' one failed, and fifty-two showed normal results. Bousquet found that where he vaccinated in three places there was very considerable inflammation, sometimes along the whole arm, and one particularly bad case led him for the first time to understand 'les frayeurs de Jenner.' Jenner's fears, of course, existed only when his early trials were being made, before he had discovered his golden rule, and when he used lymph contaminated by the filth of uncleanly cow-houses and of milkers' dirty hands; and it was in circumstances essentially parallel that Bousquet was enabled to realise these anxieties. Though in the case of some contagia the inoculation of a strong virus may cause a local slough which is not produced by the same virus when attenuated, there is no evidence that the local mildness of vaccination, as compared with casual cow-pox, is due to such a cause. Facts go to show that the mildness here has its origin not in attenuation, but in removal of contamination. In the present day, if one chose to take, on the eleventh or the twelfth day, pus of inflamed sores with hardened base and erysipelatous areola, from the arms of dirty children, living in close, ill-ventilated, and overcrowded houses, the natural breeding-ground of septic disease; if all this were done, and the lymph carefully cultivated from bad to worse, it would, in the course of a few backward removes, be quite easy to set up a stock which would realise Jenner's early difficulties. But in doing all this we should not be going back to pure cow-pox. On the contrary, we should be abandoning it, and going back to a mixed infection, its material composed of any or every variety of extraneous contamination, along with some uncertain proportion of true vaccine lymph.

Ceely, whose writings are acknowledged alike by friends and foes of vaccination to be by far the most valuable of those published by the group of observers under discussion, was quite clear on this subject of the essentials and the accidentals of pure lymph. Of course both Estlin and Bousquet may have been more or less aware of how the matter stood, and they do not indeed complain of their results—it is their latter-day readers who do so—but they do not insist on the distinction as Ceely does. He speaks both of 'the general mildness of the disease,' and of the fact 'that its topical severity depends almost wholly on the rude traction of the milkers;' while on the cows themselves he points out that 'there is rarely any manifestation of fever or constitutional disturbance.' On the milkers' hands the same class of influence is at work. 'Although there can be no doubt of the greater severity of the local and constitutional symptoms attending the casual cow-pox on the hands, &c., it is equally clear that these symptoms are greatly aggravated by rupture of the vesicles on parts so vascular, tense, and sensitive, and subject to motion.' In addition, probably by Ceely's time, a great number of the casual cow-poxes of milkers were of the nature of re-vaccinations, which are now objected to as a source of vaccine lymph, owing to greater liability to severity of symptoms.

As to the constitutional symptoms which often accompanied the early vaccinations, Jenner himself italicises his opinion: 'That the most material indisposition, or at least that which is felt most sensibly, *does not arise primarily from the first action of the virus on the constitution*, but that it often comes on *if the pustules be left to chance as a secondary disease*;' and in another place he says: 'I am more and more convinced of the extreme mildness of the symptoms arising *merely from the primary action* of the virus on the constitution.' Natural cow-pox, therefore, though characterised by greater

activity and severity on a cow's paps than on a child's arm, is by no means the wild and virulent malady, analogous to syphilis in man, which has recently been depicted to the profession and the public.

The Relationship of Small-pox to Cow-pox.—Variolation of Cows.—But Ceely did more than examine and describe natural cow-pox. The question of the relationship of cow-pox to small-pox occupied his attention, and to answer it he made a series of experimental inoculations on the cow with the virus of human small-pox. Many of his attempts failed, but some succeeded, and from these he raised a stock of lymph which was used for vaccination. In the same way Mr. Badcock, of Brighton, at various times inoculated with small-pox ninety cows, and from the successful operations took lymph which he used himself and distributed very widely, many thousands being vaccinated with it, with excellent results. These facts were accepted by the profession as setting at rest a question which had been a puzzle since vaccination was introduced—the question, namely, of the *rationale* of the prophylactic power of cow-pox. Jenner's contemporary, Pearson, though an eager advocate of vaccination, had held the two poisons of cow-pox and small-pox 'as of distinctly different species.'¹ Huggan, another believer in vaccination, said that the assertion, 'that the cow-pox is only the small-pox having undergone a certain modification by passing through the quadruped,' was 'without the least shadow of proof.'² Jenner's friend, John Ring, insisted that 'it is certain that the cow can neither receive nor communicate the small-pox;' and Pearson¹ says that Odier, of Geneva, invented the name '*la vaccine* or *vaccina*, rejecting as absurd the name of the English *variola vaccina*;' and not many years before the investigations of Ceely and Badcock, the 'Medical Repository' contains a reference to 'what physicians have long laboured to discover—a satisfactory and simple explanation of the protective power of cow-pox against small-pox.' Naturally the proof, as it was believed, that cow-pox was after all cow small-pox was held to be complete, and so it continued to be held until quite recently. But the whole subject has again been through the melting-pot.

Crookshank (vol. i. p. 808 *et seq.*) mentions several successful attempts at variolation of cows, but with the untoward result of setting up variola, not vaccinia, in individuals subsequently inoculated from the cows. He states that this occurred in Massachusetts in 1886, at Munich in 1889, at Berlin in 1847; and he also refers to Chauveau's well-known experiments in 1868-5 and again in 1871. It seems clear enough indeed that in these cases the virus of small-pox underwent no essential change after its insertion on the cow. There are, on the other hand, various cases on record in which the disease induced on the human subject by transference of matter from the insertions on the cows took on the true character of vaccinia, as observed by Ceely and Badcock.

The most recent of these is related by Dr. Cory in the Second Report of the Royal Commission on Vaccination. Dr. Simpson, Medical Officer of Health for Aberdeen (now of Calcutta), inoculated a cow's teat with small-pox lymph taken from an unvaccinated patient on the fifth day of the eruption. A vesicle resulted, and was opened 150 hours after the inoculation. 'It was very like a cow-pox vesicle.' The lymph was taken on points, and on November 21, 1885, Dr. Cory and Mr. Shirley Murphy inoculated a calf in five places with this lymph. In five days three of the places had some appearance of vesiculation. From these one child and one calf were vaccinated. In five days more, six of the seventeen insertions made in this calf developed vesicles. From these, three children and one

¹ *Med. Phys. Jour.*, vol. iii. p. 100.

² *Ibid.*, p. 244.

calf were vaccinated. In the child vaccinated from the first calf there resulted 'five perfectly normal and well-formed pearly vesicles,' and in each of the other three children there were 'five vesicles, normal in appearance and average amount of areola.' In all, 89 calves and 1,247 children were inoculated by Dr. Cory and Mr. Murphy with Dr. Simpson's lymph, and 98.8 per cent. of the cases were registered as successful. All the vaccinations were from calf to arm. Dr. Cory states that the lymph 'never produced anything but vaccine,' and there was 'no instance of contagion having taken place from one child to another at that time, after the manner in which small-pox is propagated;' indeed, he 'never saw any suspicion of other pustules,' though 'the first cases especially were very carefully observed.' It is not easy to discover any fallacy in this narrative, nor any evidence of the possibility of the resulting disease having been small-pox, and the cases of Ceely and Badcock seem equally conclusive.

Dr. Hime ('Brit. Med. Jour.' vol. ii. 1892) gives an account of a varicella of the cow, the result obtained being vaccinia; and Surgeon-General Cornish follows this with a reference to a successful experiment of the same nature carried out in India by Surgeon-Major W. G. King in April 1891.

But in connection with the writings of Ceely and Badcock, a reply has been made which demands attention. It is, that the practice with these stocks of lymph has its parallel, not in natural small-pox, nor in small-pox inoculation as carried on by Maitland and the other early operators, but in the Suttonian or mild method of inoculation, in which the results were very mild and (it is alleged) probably also non-infectious. In this view, small-pox virus may, by cultivation from arm to arm, be reduced to a disease consisting of a single pustule, without contagious properties. But Crookshank, who tells us this, also insists that its protective properties must at the same time disappear, so that the fluid used by Badcock and his clients was powerless alike for good and evil. In this connection we have already seen that in protected subjects a strictly local cultivation of small-pox by inoculation, without any accompanying general infection of the system, is quite possible; and that therefore the view may be legitimately held that, though a cow be incapable of constitutional infection, yet when inoculated with small-pox a local pustule may follow, the matter from which will have the power of giving small-pox and not cow-pox.

Conceivably, therefore, an experimenter who inoculates a cow with small-pox (the cow being protected by previous cow-pox, or being otherwise constitutionally insusceptible), and gets any local result at all, *may* get therefrom small-pox and not cow-pox. But the question is, Did Badcock and Ceely and Simpson get through the cow variola instead of vaccinia? The evidence is that they did not. In the first place, the local result of vaccination, as performed on the human subject, though it has a generic resemblance to that of small-pox inoculation especially about the eighth day, is easily distinguished from it, as has already been shown. Now Badcock and Ceely lived at a time when medical men had sufficient knowledge of small-pox inoculation and its local phenomena. So, too, they were well acquainted with the eruption both of natural and inoculated small-pox. But neither Badcock nor Ceely, nor any one of the observers of their results, nor of the many practitioners who used Badcock's lymph, makes mention of any doubt whatever as to the nature of the disease. It was vaccinia, not variola. It has been recently alleged that Ceely's assistant, who was accidentally inoculated during their experiments, took small-pox, and, in so far as the symptoms are recorded, they might stand either for much modified small-pox or for very active cow-pox. Ceely, however, describes the eruption as 'modified vaccine

in a sanguine habit, with roseolar or vesicular or vaccine lichen.' He says further that 'in no adult, except in the case of my assistant, Mr. Taylor, was there any attendant eruption; nor in any child the slightest approach to anything of a varioloid character. Roseola, strophulus, lichen, were the principal eruptions.' Besides, so distinguished an authority as Trousseau, with his attention solely devoted to the object of reducing inoculated small-pox to the mother pustule, entirely failed to get that uniformity of result obtained by Badcock, who had no such end in view. We have already given Dr. Cory's evidence that Simpson's lymph produced no eruptions of any sort whatever.

Nor do Badcock's numerous correspondents note any such occurrences, though in one case, on the tenth day, where there was a very extensive areola on the arm, 'red blotches appeared on the back and lower limbs of the child.' On the arm of Badcock's child (the first to receive the virus) on the seventh day 'the vesicle presented the usual progressive appearance,' and on subsequent days the progress was equally satisfactory. Richardson, of Brighton, said that 'to describe the appearances produced would be merely to describe the true Jennerian vesicle with which we are all familiar.' Dr. Coleman, of Horsham, and Dr. Plummer, wrote similarly, and in short, of two dozen correspondents, including Ceely, Estlin, and Cordy Burrows, not one makes mention of any pustular eruptions following the vaccination. And it is simply inconceivable that, if Badcock's lymph had been variolous, of the many thousands inoculated with it, no single case should have presented such symptoms as would have clearly indicated its true nature. As to any power it had of spreading small-pox by atmospheric infection, it may be noted that Mr. W. S. Sankey, of Dover, who had in 1848-45 vaccinated with Badcock's lymph 181 persons, of whom 118 had four vesicles, complains that 'not a single case of small-pox has occurred to test the protective value of the lymph.' One correspondent had an admirable opportunity for making a useful comparison. He had received lymph from Estlin (derived from natural cow-pox) on the day before he got Badcock's, and he writes: 'I have continued to use both kinds, and have been very careful to keep them separate. I cannot see that there is any difference in the appearance of vesicles produced by either kind.' In the Suttonian practice, the whole object was to produce a mild disease, and every part of the procedure had this as its aim. But eruptions were nearly always present. In the use of Badcock's and Simpson's lymph, several punctures were made and plenty of virus used. Yet the cases were indistinguishable from those of perfect vaccination, and in none, except in two or three vaccinated in the presence of small-pox, was there any secondary eruption.

While such facts seem quite sufficient to prove that small-pox inoculated on the cow has in some cases produced a result which on the human subject showed itself as cow-pox, yet those related by Chauveau and others seem also quite sufficient to prove that small-pox inoculated on the cow has in other cases produced a result which on the human subject showed itself as small-pox. If Ceely, and Badcock, and Simpson, and Cory are trustworthy, so also are Chauveau and the other members of the Lyons Commission.

To what conclusion, then, are we driven? On the whole subject it is necessary to speak with the greatest reserve. It appears to be sometimes a most difficult thing to get any result at all from inoculating cows with small-pox. So skilled an experimentalist as Klein entirely failed, even when he had the advice and assistance of Ceely. Dr. Cory also failed. Yet obviously the operation is at other times successful. Success seems possible even where it is not desired. In a letter to Badcock (Crookshank, vol. ii. p. 529),

W. R. Mott, of Brighton, writing in 1841, says: 'It is now more than forty years since, I believe, I inoculated the first child in the county of Kent, with the vaccine lymph, and about that time I well remember old Dr. Dobell saying that he believed it to be identical with small-pox. He was a celebrated man for inoculation, and received patients into his house. *He found his cows frequently attacked from the female patients occasionally milking the cows.*'

It will be necessary, therefore, to see if the writings on the subject throw any light on the differences of result. In Ceely's second case (that of an animal ten months old), the first attempt at variolation failed. A second attempt was made, and on the fifth day the punctures were enlarged and elevated. On the sixth day 'all present the appearance of the vaccine vesicle.' Of eight punctures, four 'seem only tubercular,' but the other four 'have a deep damask hue, and appear like oval or circular solid elevated rings, with central depressions; from one of these took clear lymph with much difficulty, and scantily charged thirty-nine points.' On the ninth day 'the four vesicles enlarging; again opened the inner margin under the daily increasing central crust of the vesicle first opened, and charged twenty points: tubercles diminishing.' On the tenth day the vesicles and one of the tubercles were larger. 'Charged twenty-seven points. Vesicles have a bluish-reddish, glistening appearance; two of them rather red at the base; one or two rather raw on each side.' Eleventh day: 'Brown crusts cover the centre of the vesicles, which appear declining.' Twelfth day: 'Declining, with increasing crusts of a blackish-brown colour, within a slightly elevated margin.' Such was a successful case. Here is an example of what Ceely calls 'a failure.' 'Third day: two or three punctures a little tumid. Fourth day: some rather hard and elevated. Seventh day: one puncture rather more tumid, about the size of a vetch. Eighth day: three or four tubercles rather larger. Ninth day: one (the largest tubercle) seems pustular, the others appear subsiding. I considered this a failure, and of course did not use the pustular product of the tubercle.'

Turning now to Chauveau's alleged successful cases, we find, according to Seaton,¹ a local result consisting of very small papules, 'scarcely projecting above the surface, slightly conical, and with the puncture of inoculation visible at the centre; they are at their fullest development by the fifth day, and are quite gone by the twelfth, leaving an extremely small blackish crust or scale at the point of puncture: at no period of their course do they exhibit any tendency whatever to secretion.' Have we not here simply what Ceely looked on as 'a failure,' characterised, too, by the short cycle which alike in variolation and vaccination has always been held distinctive of failure? It is not surprising to learn that the Lyons Commission itself had at first set aside such results, and that the majority of competent witnesses of the cases regarded the papules 'as the simple result of inflammatory action round an inoculated point, and as indicative of no specific infection.' Matter for inoculation was got by removing the papules 'and scraping well their inner surface.' (Seaton.) Indeed, the variolous virus originally inserted appears to have remained practically unchanged in the tissues, and to have been simply removed again and reinserted on human beings, who naturally developed variola and not vaccinia.

Now that fresh interest has been aroused in this question of the variolation of the cow, it is not likely to remain long without settlement. But in the meantime the whole tendency of the evidence is to show that vaccinia really is, as Jenner had supposed it, variola of the cow, and that the virus of human

¹ *Op. cit.*

small-pox is so attenuated in the bovine animal as to be deprived both of its quality of atmospheric convection, and of its tendency to cause a generalised eruption. Whether in all cases it is proper to trust, for the metamorphosis, to a single bovine transmission, is a point which is still open to discussion. Conceivably it might happen that if Trousseau's attempts at attenuation by successive cultivations of the mother pustule on the human subject were now repeated and persevered in long enough, some series of such experiments might ultimately result in a permanent modification similar to vaccinia in its freedom both from eruption and from atmospheric convection; but if, as Crookshank avers, this could be attained only at the cost of total loss of protective power, then it would differ from vaccinia in its all-important characteristic. Possibly by-and-by bacteriological science may be able to tell us why the one mode of attenuation or transformation should differ in its results from the other, but in the meantime we must be satisfied with the fact that the change in the calf from variola to vaccinia is short, sharp, and decisive, removing the objectionable and retaining only the valuable part of the original disease.

THE VACCINAL OPERATION

The operation of vaccination is usually performed on the outer aspect of the arm near the insertion of the deltoid muscle. The object being the introduction of the vaccine lymph in such a manner as will secure its absorption, various procedures may be adopted. The lymph may be taken on the point of a lancet and inserted by punctures or by scratches, or by rubbing it on an abraded surface. Or it may be laid on the skin, and scratches, &c., made through it, but probably this is not quite so good a method. The lancet is usually held at an angle so as to make a valvular scratch or incision, which should enter, but not go beyond, the true skin. Some vaccinators find that a half-blunt lancet is better than a very sharp one. Others use a variety of instruments, fitted with needle points, &c., but the great advantage of the lancet is that it is very easily kept clean.

Instructions to Public Vaccinators.—The following 'Instructions for Vaccinators under Contract' issued by the Medical Department of the Local Government Board in 1887, form an excellent guide as well for the private as for the public vaccinator. Private practitioners would even find it of great service to keep a register of vaccinations similar to that used at the public stations. It is occasionally useful to be able to trace back to its source, step by step, any lymph that is being employed.

Instructions for Vaccinators under Contract

1. Except so far as any immediate danger of small-pox may require, vaccinate only subjects who are in good health. As regards infants, ascertain that there is not any febrile state, nor any irritation of the bowels, nor any unhealthy state of skin; especially no chafing or eczema behind the ears, or in the groin, or elsewhere in folds of skin. Do not, except of necessity, vaccinate in cases where there has been recent exposure to the infection of measles or scarlatina, nor where erysipelas is prevailing in or about the place of residence.

2. In all ordinary cases of primary vaccination make such insertions of lymph as will produce at least four separate good-sized vesicles or groups of vesicles, not less than half an inch from one another. The total area of vesiculation on the same day in the week following the vaccination should be not less than half a square inch.

3. Direct that care be taken for keeping the vesicles uninjured during their progress, and for avoiding afterwards the premature removal of the crusts. Do not use any needless means of 'protection' or of 'dressing' to a vaccinated arm; but if in a particular case you find reason for means of 'protection' or of 'dressing,' define the material and the manner of use of the appliance best adapted to the case, avoiding all such as cannot readily be destroyed and replaced whenever they become soiled.

4. Enter all cases in your register on the day when you vaccinate them, and with all particulars required in the register up to and including the column headed 'Initials of Person performing the Operation.' Enter the results on the day of inspection. Each of those entries must be attested by the initials of the person who inspects the case. In cases of primary vaccination, register as 'successful' only those cases in which the normal vaccine vesicle has been produced; in cases of re-vaccination, register as 'successful' only those cases in which either vesicles, normal or modified, or papules surrounded by areolæ, have resulted. When any operation (whether vaccination or re-vaccination) has to be repeated owing to want of success in the first instance, it should be entered as a fresh case in the register.

5. Endeavour to maintain in your district such a succession of cases as will enable you to vaccinate with liquid lymph directly from arm to arm at each of your contract attendances; and do not, under ordinary circumstances, adopt any other method of vaccinating. To provide against emergencies always have in reserve some stored lymph; either *dry* on ivory points, thickly charged and constantly well protected from damp; or *liquid*, in fine, short, uniformly capillary (not bulbed) tubes, hermetically sealed at both extremities. Lymph, successfully preserved by either of these methods, may be used without definite restrictions as to time. With all stored lymph caution is necessary, lest in time it have become inert, or otherwise unfit for use.

6. Consider yourself strictly responsible for the quality of whatever lymph you use or furnish for vaccination. Never either use or furnish lymph which has in it any, even the slightest, admixture of blood. In storing lymph, be careful to keep separate the charges obtained from different subjects, and to affix to each set of charges the name, or the number in your register, of the subject from whom the lymph was derived. Keep such note of all supplies of lymph which you use or furnish as will always enable you to identify the origin of the lymph. Do not employ lymph supplied by any person who does not keep exact record of its source.

7. Never take lymph from cases of re-vaccination. Take lymph only from subjects who are in good health, and, as far as you can ascertain, of healthy parentage; preferring children whose families are known to you, and who have elder brothers or sisters of undoubted healthiness. Always carefully examine the subject as to any existing skin disease, and especially as to any signs of hereditary syphilis. Do not take lymph from children who have any sort of sore at or about the anus. Take lymph only from well-characterised, uninjured vesicles. Take it at the stage when the vesicles are fully formed and plump. Do not take it from a vesicle around which there is any conspicuous commencement of areola. Open the vesicles with scrupulous care to avoid drawing blood. Take no lymph which, as it issues from the vesicle, is not perfectly clear and transparent, or which is thin and watery. From a well-formed vesicle of ordinary size, do not, except under circumstances of necessity, take more lymph than will suffice for the immediate vaccination of five subjects, or for the charging of seven ivory points, or for the filling of three capillary tubes; and from larger or smaller vesicles,

take only in like proportion to their size. Never squeeze or scrape or drain any vesicle, and do not use lymph that has run down the skin. Be careful never to transfer blood from the subject you vaccinate to the subject from whom you take lymph.

8. Scrupulously observe in your inspections every sign which tests the efficiency and purity of your lymph. Note any case wherein the vaccine vesicle is unduly hastened or otherwise irregular in its development, or wherein any undue local irritation arises; and if similar results ensue in other cases vaccinated with the same lymph, desist at once from employing it. Consider that your lymph ought to be changed, if your cases, at the usual time of inspection on the day week after vaccination, show any conspicuous areolæ round their vesicles.

9. Keep in good condition the lancets or other instruments which you use for vaccinating, and do not use them for any other purpose whatever. When you vaccinate, have water and a napkin at your side, with which invariably to cleanse your instrument after one operation before proceeding to another. Never use an ivory point or capillary tube a second time either for the conveyance or for the storage of lymph, but when points or tubes have once been charged with lymph and put to their proper use, do not fail to break or otherwise destroy them.

In the writer's practice two additional points have been attended to. One is to use boiling water instead of cold water for cleaning the lancet after each operation. This involves a momentary delay for cooling the blade of the instrument. But where time is of consequence, two lancets could be used alternately. The other is, that where the exigencies of private practice result in the use of lymph stored in capillary tubes, these tubes should be sterilised, as in the practice of Dr. Neil Carmichael at the Vaccination Station in Ingram Street, Glasgow. That sterilised tubes are less liable to contain foreign matter is shown by the rapidity with which water or lymph is drawn into them as compared with ordinary tubes.

Calf Lymph.—Since 1881 Government has had in operation at Lamb's Conduit Street, London, an animal vaccine lymph station. With the exception of a series of cases in 1885-86, vaccinated with Simpson's lymph, already referred to, the only stock of lymph used since March 1882 had its origin in a case of natural cow-pox which occurred at Lafôret, near Bordeaux, the lymph being supplied by Dr. Dubreuilh. It has never been humanised, direct vaccination being in all cases done from the calf. The calves are vaccinated on the shaven abdomen, and the lymph is taken on the fifth day. It is from this source that the Local Government Board supplies calf lymph to all practitioners who require it.

At first it was feared that the 'insertion success' would be less than with the humanised lymph, but the remarkable uniformity of result obtained by Dr. Cory has shown this fear to be illusory. Five insertions are made in each case, and in the year 1888-89 Dr. Cory was successful in 96·8 per cent. of the insertions, while it was only in 0·8 of the cases that all the insertions failed.

The resulting vesicles are in no way distinguishable from those produced by humanised lymph; but when calf lymph is sent out to vaccinators in the provinces it should be used at once on being received. In 32,000 cases operated on between April 1882 and September 1889, one died from erysipelas and one from cellulitis. In addition, other six deaths were noted as having occurred within from a week to two months of the operation, but on investigation no connection therewith could be traced. Three were certified as due

to convulsions, one to small-pox (which appeared on the fifth day after vaccination), one to enteritis, and in one case, where death occurred two months after the vaccination, the cause could not be ascertained. In Prussia, calf lymph is exclusively used, on the ground that in this way the invaccination of syphilis is rendered impossible. But in this country, such accidents have been so much rarer than on the Continent that medical men as a rule have no hesitation in using humanised lymph in their own families, so that the use of calf lymph appears rather to be found in the allaying of groundless alarm—certainly a most important point in itself—than in any intrinsic superiority. In the face of a threatened epidemic, however, the ease with which a stock of calf lymph can be multiplied from calf to calf makes it incomparably more useful than the scanty supplies of humanised lymph that are obtainable even from the largest stations. There are no facts to show whether, as a prophylactic, it is more or less powerful than the other. When used by Mr. Goude for revaccinating the nurses in the Highgate Small-pox Hospital, it conferred the usual complete immunity.

The advantage of direct vaccination from arm to arm, or from calf to arm, as compared with the use of stored lymph, is mainly that the insertion success is much greater, the failures either total or partial being much fewer. Indeed, thoroughly experienced vaccinators hardly believe in any genuine insusceptibility to vaccination among infants. It is true that in England every year many children are certified as 'insusceptible.' In 1886 the number was 1,278. But Dr. Cory states (in Sir George Buchanan's 1887 Report) that among 88,000 primary operations with unstored human or calf lymph, in only one case did he fail twice in an attempt at vaccination. As to the failures of a first attempt, he had fourteen among 16,000 cases with humanised lymph, or one failure in 1,140 children; and 70 among 21,781 cases with calf lymph, or one failure in 311 children. Obviously, therefore, practically the whole of the 1,278 children who in 1886 were registered as insusceptible are to be regarded simply as unvaccinated children, open to attack by small-pox.

Phenomena of Primary Vaccination.—The normal result of infantile vaccination cannot perhaps be better described than in a circular issued by the War Office in 1865. 'When vaccination has been successfully performed on a healthy infant, the puncture may be felt elevated on the second or third day; and soon afterwards, if examined with a magnifying glass, appears surrounded by a slight redness. On the fifth or sixth day a distinct vesicle is formed, having an elevated edge and depressed centre. On the eighth day it appears distended with a clear lymph. The vesicle, on this, its day of greatest perfection, is circular and pearl-coloured, its margin is turgid, firm, shining, and wheel-shaped. Late on the seventh or early on the eighth day an inflamed ring or areola begins to form around the base of the vesicle, and with it continues to increase during the two following days. This areola is of a circular form, and its diameter extends from one to three inches. When at its height, on the ninth or tenth day, there is often considerable hardness and swelling of the subjacent cellular membrane. On the tenth or eleventh day the areola begins to subside, leaving as it fades two or three concentric circles of redness. The vesicle now begins to dry in the centre, and acquires there a brownish colour. The lymph which remains becomes opaque, and gradually concretes; so that about the fourteenth or fifteenth day the vesicle is converted into a hard round scale of a reddish-brown colour. This scale contracts, dries, blackens, and about the twenty-first day falls off. It leaves a cicatrix, which commonly is permanent in after life, circular, somewhat depressed, dotted or indented with minute pits, and in some instances radiated. The

above-described local changes, while in active progress, are attended by feverishness ; first, from the fifth to the seventh day, so slight that often the fact passes unobserved, and again more considerable during those days when the areola is about its height ; the infant now being restless and hot, with more or less disturbance of stomach and bowels. About the same time, especially if the weather be hot, children of full habits not unfrequently show on the extremities, and less copiously on the trunk, a lichenous vascular or vesicular eruption which commonly continues for about a week.'

Phenomena of Re-vaccination.—In re-vaccination the results are sometimes as above described, but frequently the place of vesicles is taken by papules, surrounded by an areola, the maximum of effect occurring on or before the fifth day. 'Or if vesicles form, their shape is apt to vary from that of the regular vesicle, and their course to be more rapid, so that their maturity is reached on or before the sixth day, their areolæ declines on or before the eighth day, and their scabbing begins correspondingly early. In either case the areolæ tend to diffuse themselves more widely and less regularly, and with more affection of the cellular membrane, than in primary vaccination ; and the local changes are accompanied by much itching, often by some irritation of the axillary glands, and in some cases on the fourth or fifth day by considerable febrile disturbances.' The operation, indeed, is far from being so easily performed with success as that of primary vaccination. In the army the results are classified as—(1) perfect vaccine pustule, (2) modified vaccine pustule, and (3) failure. The 'modified' pustule consists often of a papule, not strictly a pustule at all. In the ten years 1879–88, the average per 1,000 of results was—(1) perfect 427, (2) modified. 314, and (3) failure, 258. In the third class, the regulation is that when re-vaccination has failed, the operation will be repeated from another source when practicable ; and in the event of the operation still proving unsuccessful, the names of the individuals will be recorded, with a view to their being re-vaccinated at some subsequent date. But unfortunately no separate statistics of these re-vaccinations after failures are furnished. It is not to be understood that the difficulty in vaccinating these adults is entirely owing to their having undergone primary vaccination. Among primary vaccinations of soldiers and recruits in the same ten years, only 582 per 1,000 gave a perfect result, while 254 gave a modified pustule and 212 failed. These figures, however, perhaps exaggerate the difficulty. For it is stated that the term 'primary vaccination' as here used embraces all the men who showed no marks. Very likely many of these had some fraction of resistance remaining from an infantile vaccination, of which the evidences had disappeared.

Prevalence of Vaccination in England.—In presence of the propaganda against vaccination there has been in recent years a slight decrease in the practice in England, this being due rather to special default in certain localities, notably in Leicester, Keighley, and Dewsbury, than to any widespread neglect. The following is the percentage of cases 'not accounted for' (these including postponements) in the metropolis and in the rest of England since the coming into force of the Act of 1871. This Act has not perhaps greatly increased the numbers operated on. Its main results have rather been, first, to reduce the average age at which primary vaccination is performed, and second, to improve the average quality and quantity of vaccination (and especially of public vaccination done under the superintendence of the Local Government Board) in each individual case.

At the same time, and so far as mere number is concerned, it is to be

borne in mind that the percentage which gets vaccinated of the children born in any one year is no doubt much greater than the percentage which had been vaccinated of the persons who die in the same year; so that in this way, though the primary vaccinations were fractionally less in 1886 than in 1885, yet at the end of the year 1886 the vaccinated percentage of the total population would doubtless be greater than at the end of the year 1885.

Percentage of Births 'Unaccounted For.'

Year	Metropolis	Rest of England	Year	Metropolis	Rest of England
1872	8.8	4.5	1881	5.7	4.3
1873	8.7	4.2	1882	6.6	4.5
1874	8.8	4.1	1883	6.5	4.9
1875	9.3	3.8	1884	6.8	5.3
1876	6.5	4.0	1885	7.0	5.5
1877	7.1	4.1	1886	7.8	6.1
1878	7.1	4.3	1887	9.0	6.7
1879	7.8	4.5	1888	10.3	8.2
1880	7.0	4.5	1889	11.6	9.6

In Scotland, the annual percentages of cases unaccounted for have been less than in England. For the years 1875-80 they have been as follows:—2.08, 2.02, 1.84, 1.94, 1.98, 1.89, 2.05, 2.09, 1.97, 2.19, 2.29, 2.39, 2.45, 2.51, 2.63, 2.48.

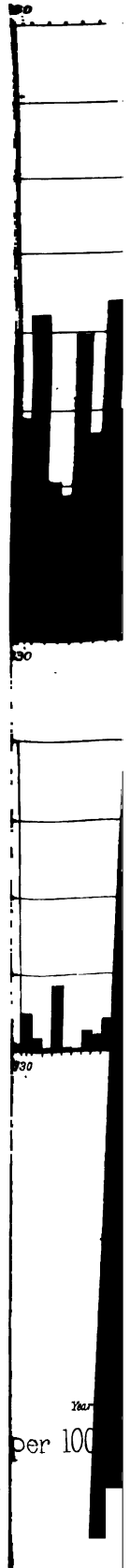
THE VALUE OF VACCINATION

Some important evidences of the value of vaccination have been already referred to—especially the experiences of the dairy people and of the country practitioners in the dairy districts in Jenner's own day, and the numberless variolous inoculation tests applied by the early vaccinators. In their scientific aspects these latter were, as Dr. Wilks observes,¹ equal in value to the experiments carried on in the biological laboratories of the present day, while in number they were infinitely greater. All over the civilised world, thousands of persons who had not undergone a certain preliminary operation were inoculated with the virus of small-pox, and, with a very small percentage of failures, all received the disease; and simultaneously all over the civilised world thousands of persons who had undergone the preliminary operation in question were also inoculated with the virus of small-pox, and, with a very small percentage of exceptions, all resisted the disease. The conditions of the two masses of subjects experimented on were similar in every other possible respect. They were taken practically at random from people of all ages and conditions of life, with the proviso only that they should resemble each other in the one fact, that they had not suffered from small-pox. And the particular preliminary operation which divided them into two classes was that which in the dairy counties had long been held to possess the power of preventing small-pox, and which had been introduced to public notice by Jenner specifically on account of his belief in its anti-variolous power. The law now prevents this huge experiment of variolous inoculation from being repeated in any single instance; and in respect of the value of the evidence which it would afford, the prohibition is unfortunate.

Another class of evidence, the striking value of which we nowadays have

¹ *Lancet*, February 1890.





tunity of realising, was that of the accidental or intentional unvaccinated and recently vaccinated persons to the contagion x , which was then a matter of everyday occurrence. Jenner's series had continually demonstrated to them the worth of vaccine the immunity with which the vaccinated members of a family themselves to the infection of a disease which successfully attacked sons and sisters who had not taken advantage of the new prophylactic outbreaks like those of Montreal and Sheffield in recent years, in daily life of our small-pox hospitals, we have occasional opportunities of obtaining proofs of a similar character, and these will be referred to.

Statistical Evidence.—The statistical evidence is so voluminous that it is difficult. Examples of it may be given under several headings.

The Decline in the Total Small-pox Mortality since the Introduction of Vaccination.—The table at p. 892 shows the remarkable decrease that took place in the small-pox mortality in various European countries, following on the introduction of vaccination. It has been urged that this was due, not to the introduction of vaccination, but to the decrease of variolous inoculation. But we have already seen that a lessened amount of inoculation did not necessarily result in a lessened spread of natural small-pox by inoculation, and that the mortality resulting from inoculation as practised in the latter part of the last century, at least in this country, was comparatively a slight one, with very little infectious material than natural small-pox. And as regards the continent of Europe, it happened that in Sweden and Copenhagen inoculation came largely into vogue, yet, as is seen in the table, the rate in Sweden fell from 2,050 per million living in the last century to 168 in the present century, while the corresponding figures for Copenhagen were 8,128 and

London Bills of Mortality.—The attached diagram gives the contributions to the total mortality in London, by small-pox, measles, and diphtheria-cough from 1629 to 1890. Up to 1838 the figures are calculated from the London Bills of Mortality¹ and subsequently from the Registrar-General's reports. Dr. Ogle shows² that the Bills of Mortality understate considerably the number of deaths, and that this is especially the case in the closing years of the period for which they are available. Fortunately this does not necessarily affect the proportion of small-pox mortality to the total mortality; but even here there are doubtless inaccuracies, especially in the years more immediately preceding 1838, when the registration system came into force. 'In the case only of very obvious diseases,' says Dr. Ogle, 'can much reliance be placed' on the statements of the old women who acted as searchers. But though difficulties of diagnosis arise even yet in the cases of mild small-pox, no disease could well be more obvious than small-pox which has ended fatally. And the domestic diagnosis of measles and whooping-cough must have been sufficient to furnish the searchers with fairly accurate information as to these diseases.

In the diagram, a striking fall is observable in the small-pox curve, while the curves of the other two maladies (infantile diseases, as was small-pox before vaccination times) show a rise rather than a fall. Had the small-pox fall been due to the improved sanitation, then the other two should have shown the effects of the same agency.

Bearing on this question of the influence of sanitation as a factor in the

¹ Guy, *Trans. Stat. Soc.*
VOL. II.

² *Jour. Stat. Soc.* September, 1892.

small-pox decline, the figures of the following table should be noted (see also 'Vaccination Vindicated,' p. 19).

Death-rate per Million living in England and Wales per Annum.

Period	Measles	Scarlatina and Diphtheria	Whooping-cough	Fever	Diarrhoea
1888-90 (3 years)	580	770	500	1,110	220
1841-42 and 1847-50 (6 years)	430	870	490	1,160	710
1851-60 (10 years)	410	980*	500	910	920
1861-70 (10 ") : : :	440	970 190	530	890	970
1871-80 (10 ") : : :	380	720 120	510	490	920

* Includes some diphtheria.

The lesson taught by these statistics is that, in reference to sanitation, each disease has to be considered by itself. Fevers have decreased enormously. But for typhoid fever sanitation has meant the removal, to a great extent, of the faecal impurities which we know to be its principal cause. Their removal, however, has not lowered the death-rate from measles. And for typhus fever, sanitation has meant the diminution of personal filth and overcrowding. But that diminution has not brought down the diarrhoea death-rate. Similarly for small-pox, sanitation has meant the greater abundance and efficiency of vaccination. But vaccination has not lessened the mortality from whooping-cough. Doubtless to some degree personal and municipal cleanliness, good food and pure water, have a beneficent effect on all diseases, whether zymotic or not; and in the benefit small-pox has had its share. But the lessening of small-pox cannot be set down to improved drainage, any more than can the lessening of enteric fever be set down to vaccination.¹

London Small-pox.—In London, owing partly to the existence of the great small-pox hospitals, which are held by Power and others to act as centres for the propagation of small-pox, the decline in small-pox has not until in recent quinquennia been so observable as in the provinces, as the following figures show: ²—

Deaths from Small-pox per Million living.

—	1838-42	1847-49	1850-54	1855-59	1860-64	1865-69	1870-74	1875-79	1880-84
London	755	460	300	237	281	276	654	292	244
Provinces	547	274	271	192	175	122	889	48	84

But even London gives indubitable evidence of the decrease of small-pox, as is seen in the following table.³

If it be pointed out that here the total mortality has also diminished, the answer is to be found in observing that in the successive periods since vaccination was introduced (beginning 1801-10), the fraction contributed by small-

¹ In the decade 1881-90, scarlatina, whooping-cough, fevers, and diarrhoea have shown a further fall, and measles and diphtheria a rise; but these changes do not, of course, affect the teaching to be derived from the figures in the table. In 1881-90 the rates have been: Measles, 440; scarlatina, 840; diphtheria, 160; whooping-cough, 450; fevers, 240; diarrhoea, 660.

² Supplement to the *Fifteenth Annual Report of the Local Government Board*, p. xi.

³ For the first five periods the figures are mere estimates (by Dr. Farr), and are necessarily more or less inaccurate.

Years	Average annual deaths per million from all causes	Average annual deaths per million from small-pox
1660-79	80,000	4,170
1728-57	52,000	4,260
1771-80	50,000	5,020
1801-10	29,200	2,040
1831-35	32,000	880
1838-53	24,900	518
1854-71	24,200	388
1872-90	21,400	178

pox to the total mortality has been reduced somewhat as follows :— $\frac{1}{15}$ th, $\frac{1}{10}$ th, $\frac{1}{50}$ th, $\frac{1}{60}$ th, and $\frac{1}{120}$ th.

The small-pox deaths per million living in England and Wales have been as follows in successive quinquennials.

Years	1838-40	1841-2 and 1847-49	1850-4	1855-9	1860-4	1865-9	1870-4	1875-9	1880-4	1885-90 (6 years)
Death-rate	771	295	279	199	191	148	433	83	62	38

In Scotland under optional vaccination, 1855-64, the annual death-rate from small-pox was 84 per 100,000 inhabitants; under compulsory vaccination, 1865-89, the 84 was reduced to 8, and during the last ten years of this period, it was only 0.48.

Swedish small-pox statistics go back to 1774. From that date to 1800, the average annual death-rate was 2,008 per million inhabitants. From 1801 to 1815, vaccination was optional, and the rate fell to 681. Then the operation became compulsory, and the average rate from 1816 to 1885 inclusive has been 178 per million. For the eight years 1877 to 1885 it was 41 per million. (Dr. Edwardes.)

The Age-incidence of Small-pox.—This subject has already been referred to at p. 896. The evidence which it yields of the value of vaccination now falls to be pointed out. The Registrar-General gives the following figures :¹

Mean Annual Deaths from Small-pox, at successive Life Periods, per Million, living at each such Life Period, 1847-53, 1854-71, and 1872-87.

Period	All ages	0-5	5-10	10-15	15-25	25-45	45 and upwards
1. Vaccination optional, 1847-53*	305	1,617	837	94	109	66	22
2. Vaccination obligatory, but not efficiently enforced, 1854-71	223	817	243	88	163	181	52
3. Vaccination obligatory, but more efficiently enforced by vaccination officers, 1872-87		114	242	120	69	122	107

* In this table the period of optional vaccination begins with 1847, not with 1838, because the deaths were not abstracted in combination with ages until 1847.

The table shows that under five years of age, when the influence of primary vaccination is most active, the decrease in small-pox is at its maximum—from one hundred in the first period to fifteen in the third period; that in the second quinquennial of life the decrease is from one hundred to thirty-six, and that in the third quinquennial it is only from one hundred

¹ *First Report of Vaccination Commission*, p. 114.

to seventy-three, while in the remaining periods of life there is an actual increase in the mortality. All this corresponds exactly with the doctrine of vaccination, and the increased mortality of older persons points to the need of re-vaccination. In the last century, when almost everyone took small-pox—in the great majority of cases at some time between birth and fifteen years of age—the survivors were in large measure permanently protected from attack and especially from death by small-pox, and hence at advanced ages the mortality was low. But with the gradual substitution of the less permanent protection of infant vaccination, there has been a corresponding increase in the deaths at ages when that protection has diminished.

The same lesson is taught even more strikingly by noting how the age-incidence of fatal small-pox has altered since the introduction of vaccination. The following table is from the Report of the Medical Department of the Local Government Board for 1884 :—

Contributions of Various Ages to 1,000 Small-pox Deaths at all Ages.

Ages at death	Geneva, 1690-1760	Kilmarnock, 1728-1764	London, 1848-1851	London, 1884		
				Unvaccinated community	Vaccinated community	Total inhabitants
0-10	961	988	815	612	86	343
10-20	26½	5	59	146	173	170
20-30	10	7	83	108	819	213
30-40	2½	—	82	72	221	142
40 and upwards			11	62	201	132
Total	1,000	1,000	1,000	1,000	1,000	1,000

These figures show (1) that when vaccination was unknown, from 96 to 99 per cent. of all small-pox deaths in Geneva and Kilmarnock occurred under ten years of age—that indeed small-pox was in such places a disease of childhood; (2) that in London in 1848-51, when vaccination was partially in vogue without being enforced by law, 80 per cent. of the victims were under this age; (3) that in London in 1884, under general vaccination, only 84 per cent. of the total small-pox deaths were under ten years; and (4) that among the vaccinated community of London less than 9 per cent., while among the unvaccinated community 61 per cent., were under ten years. Thus we find that, with the spread of vaccination, children as a whole, and especially vaccinated children, bear less and less of the total small-pox mortality, while among the unvaccinated the distribution approaches more nearly to that of pre-vaccination times.

(2) *Age-incidence in the Unvaccinated.*—But it will be noted that even among the unvaccinated the 96 to 99 per cent. of last century is represented by only 61 per cent. in London in 1884. Whence the change? It is because vaccination has in this connection two results. It protects the individual, and, by reducing the available material for the spread of small-pox, it renders epidemics less frequent. In this latter benefit the unvaccinated share. In Kilmarnock, in the last century, epidemics came on an average every four-and-a-quarter years, and less than 12 per cent. of the persons who died in one epidemic had been alive during the previous epidemic. The disease had to secure its victims almost wholly from the population that had come into existence since its last visitation. One outbreak left almost no victims for

its successors—in the same way hardly any had been left to it by its predecessors. Obviously, therefore, if by vaccination the frequency of epidemics is lessened, the deaths even of the unvaccinated will occur at a higher range of ages, and hence it is that the 96 to 99 per cent. furnished by children in places like Kilmarnock in the last century, when all were unvaccinated, has been reduced to 61 per cent. among the unvaccinated of the present day; and hence it is also that before the Royal Commission on vaccination Dr. Collins (Q. 891) was able to show that in the years 1881–7, of 8,099 unvaccinated deaths only 89 per cent. were under five years of age, as compared with about 80 per cent. in the last century. Thus, while of 100 unvaccinated deaths 89 are under five years old, of 100 vaccinated deaths only 9 are under that age, the former figure being obtained under the influence of the infrequency of epidemics, and the latter under that influence plus the direct individual protection by vaccination.

The same difference between vaccinated and unvaccinated is illustrated by the Registrar-General¹ in the following table based on the records for England and Wales for the years 1872–4 and 1877–80:—

Proportion of Deaths under and over 15 Years of Age per 1,000 Deaths from Small-pox in Vaccinated and Unvaccinated Persons respectively.

Age	Unvaccinated	Vaccinated
Under 15 years	672	384
Over 15 „	328	666
Total	1,000	1,000

For Japan and the Netherlands the following are the corresponding figures under and over 10 years of age:²—

—	Japan, 1879–80		Netherlands, 1870–78	
	Vaccinated	Unvaccinated	Vaccinated	Unvaccinated
Under 10 years	331	785	180	682
Over 10 „	669	265	820	318
Total	1,000	1,000	1,000	1,000

Taking five years of age as the dividing period, Brussels and Buda-Pesth give these results (Abbott):—

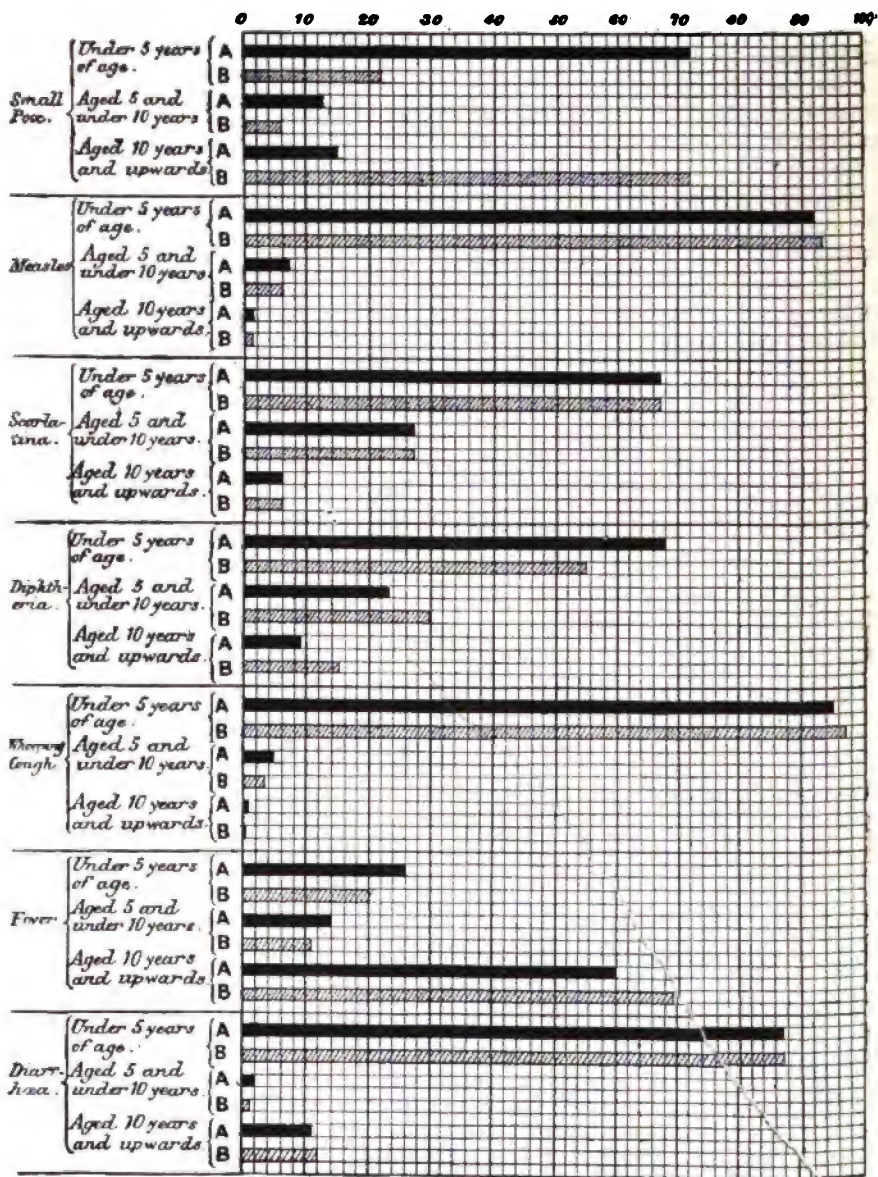
—	Brussels, 1888 (epidemic year)		Buda Pesth, 1876–81	
	Vaccinated	Unvaccinated	Vaccinated	Unvaccinated
Under 5 years	200	715	294	766
Over 5 „	800	285	706	234
Total	1,000	1,000	1,000	1,000

The manner in which small-pox age-incidence has altered, while the age-incidence of other comparable maladies has remained unchanged, is well illustrated by Dr. Barry in a diagram, of which a copy is annexed, giving the facts for Sheffield for two periods of ten years. The slight alteration which has occurred in the age-incidence of fever is explained by Dr. Barry as due in part at least to a change of nomenclature: remittent fever being included in the earlier period and excluded in the later.

¹ *Forty-third Annual Report, 1890.*

² Abbott, *op. cit.*

DIAGRAM showing the variations in the Age Incidence of the several fatal Infectious Diseases in the Borough of Sheffield as witnessed during A. the ten years 1861-70, B. the ten years 1876-83, 86-87.



NOTE. It will be observed that the lines indicating the age incidence at one and another time have no relation to the actual amount of disease prevalent. The total of the black line gives a constant 100 for the one period and the total of the shaded line a constant 100 for the other.

Again, on this subject, the following diagram, also Dr. Barry's, shows how the age-incidence of Sheffield small-pox has altered coincidently with the spread of vaccination :—

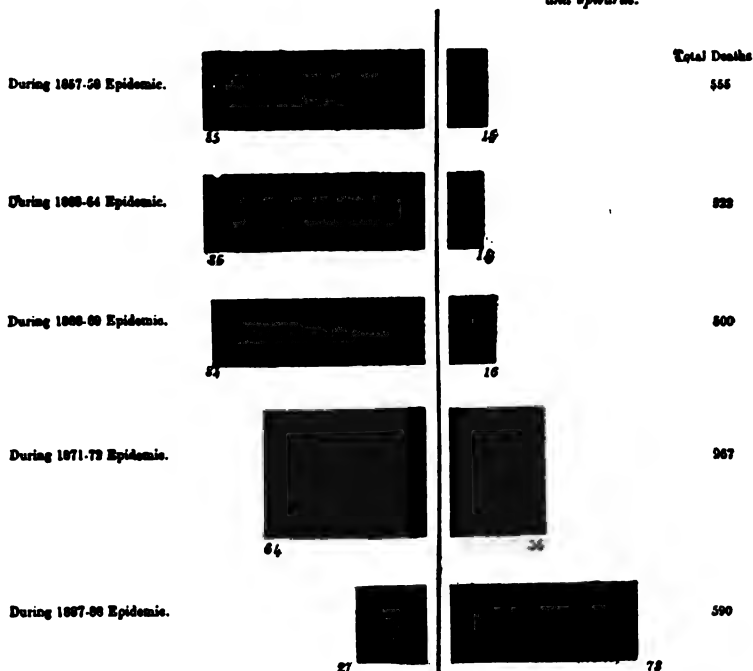
DIAGRAM showing for the Borough of Sheffield

- (a) The share, during successive Epidemics, of the total Small-pox mortality borne by children under 10 and by persons aged 10 years and upwards.
- (b) Similar facts, during 1887-88, for "Vaccinated" and "Unvaccinated" classes respectively.
- (Difference of width of horizontal bars denotes differing amounts of fatal Small-pox.)

(a.) Out of every 100 small-pox deaths among people of all classes in each of the five epidemics 1857-8, 1863-4, 1868-9, 1871-2, and 1887-8 :—

Share borne by Children under 10 years of age.

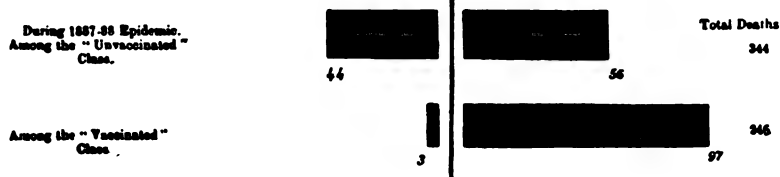
Share borne by Persons aged 10 years and upwards.



(b.) Out of every 100 small-pox deaths among people of all ages in the "vaccinated" and "unvaccinated" classes respectively during 1887-88 :—

Share borne by Children under 10 years of age.

Share borne by Persons aged 10 years and upwards.



Finally, in regard to age-incidence, there is in one of the Scottish Registrar-General's Reports an interesting comparison as to the small-pox deaths of children under one year old. From 1855 to 1864, when vaccination (in Scotland) was only optional, the number of deaths from small-pox in children under one year old was very nearly equally divided between the two halves of the year.* But when vaccination became compulsory at the age of six months (in Scotland) this grouping altered very much—about 79 per 100 being under six months and only 21 between six and twelve months. The inference was that vaccination made the difference. But a third fact was needed to complete the comparison—namely, the proportion in each of the two half years when vaccination was totally unknown. The statistics of Kilmarnock in the last century supplied the omission as follows :—

Rates of Deaths from Small-pox between Six and Twelve Months of Age, to 100 Deaths from Small-pox under Six Months, during three Selected Periods.

Age	Kilmarnock, 1738-1768 (vaccination unknown)	Scotland, 1855-1864 (vaccination optional)	Scotland, 1865-1879 (vaccination compulsory)
Under 6 months . . .	100	100	100
From 6 to 12 months . .	491	108	26

The first ratio may be taken as the normal one of natural small-pox. It is a disease with nearly five times the mortality in the second six months that it has in the first six months of life. But when modified in the second six months by optional vaccination, the mortality decreases very much—from 491 to 108. And when vaccination becomes compulsory, as in the third period, the 108 becomes reduced to 26. The second half year is (in Scotland) the first part of life in which the influence of vaccination is brought to bear on the prevalence of small-pox, and its effect is surely abundantly obvious. Surely, too, it is very obvious that it has not been sanitation which has caused the remarkable change in this altered relationship of babies towards death from small-pox.

The manner in which sanitation acts, or rather does not act, with reference to the age-incidence, is seen on looking at the statistics of the decline of fevers, a decline which, in so far as it is not a mere change of nomenclature, is universally accepted as due to sanitary measures. The following table was handed in to the Vaccination Commission by the Registrar-General (First Report, p. 114) :—

Mean Annual Deaths from Fever, at successive Life Periods, per Million living at each such Life Period, 1847-53, 1854-71 and 1872-87.

Period	All ages	0-5	5-10	10-15	15-25	25-45	45 and upwards
1847-53	1,189	1,512	1,118	911	1,108	910	1,888
1854-71	870	1,297	988	718	807	656	972
1872-87	867	426	879	844	431	327	828

The regularity of the influence of sanitation, as exhibited by these figures at every period of life, demonstrates beyond the possibility of doubt that it is not to sanitation that we owe the changes of age-incidence shown in the preceding tabular statements of reduction of small-pox mortality.

(8) *Effects of Re-vaccination.*—The most striking case is that of Prussia,

now the best vaccinated country in the world. Re-vaccination was made compulsory in 1874, the law taking effect in 1875. So long ago as 1834, re-vaccination had been compulsory in the army, with the result that the army rates have since been remarkably low. And in 1835 a regulation was issued which stated 'that teachers, masters, craftsmen, &c., would 'do well to satisfy themselves' that pupils, apprentices, &c., were vaccinated, while children receiving scholarships or other benefices had to show proof of vaccination. But there were no penalties unless in case of attack by small-pox, and then naturally the penalty was seldom inflicted. In fact, a chief result of this regulation appears to have been that when the pandemic of 1870-8 swept over Germany, the returns of small-pox cases as 'vaccinated' or 'unvaccinated' were rendered worthless by the parents making mis-statements to evade the penalty in question. But in 1874 all this was changed, re-vaccination being made compulsory and the law properly enforced. The consequence has been a most remarkable and sudden disappearance of small-pox from Prussia. The diagram on the next page by Dr. Arthur F. Hopkirk shows the facts.²

To show the additional protection afforded by re-vaccination over that conferred by infantile vaccination alone, Gerstäcker (quoted by Abbott, *loc. cit.*) compares London and Berlin. But in regard to the former the influence of the Metropolitan Small-pox Hospitals, already discussed, must be borne in mind. The figures are as follow :—

—	1875	1876	1877	1878	1879	1880	1881	1882	1883
London . . .	1.3	20.8	71.0	38.8	12.1	12.5	61.9	11.1	8.4
Berlin . . .	5.2	1.8	0.4	0.8	0.7	0.8	4.7	0.4	0.8

In the Army and Navy.—The value of re-vaccination is further shown by the experience of our own army and navy. Writing in 1819, Sir Gilbert Blane said : 'When there was no vaccination in our navy, one-fifth of all the men enlisted died of small-pox.'³

The Army.—In the army an order for the re-vaccination of all recruits was issued on September 21, 1858. But the short service system did not begin till 1873, so that the influence of the order would be very gradual, the number of unprotected soldiers decreasing year by year. For purposes of comparison, the experiences of the forces in the Colonies, India, and Egypt, are of comparatively little use, as the corresponding rates in the surrounding population are not easily obtainable. We had therefore best look for information to the army in the United Kingdom. The statistics for the forty-two years, 1847-88 inclusive, are given in the second Report of the Royal Commission, pp. 278-9. If these are divided into three periods of fourteen years each, the first will represent roughly—(1) a time when re-vaccination was not insisted on; (2) a time during which the army was being gradually transformed into a re-vaccinated force, and (3) a time in which the army was under the full benefit of the practice resulting from the regulation of 1859. It is not, however, to be assumed that every individual soldier has now the completest protection that re-vaccination can afford. Through occasional oversight, through occasional interference with the success of the operation by men sucking the lymph from the arm, and through operative failures, there is still an unprotected,

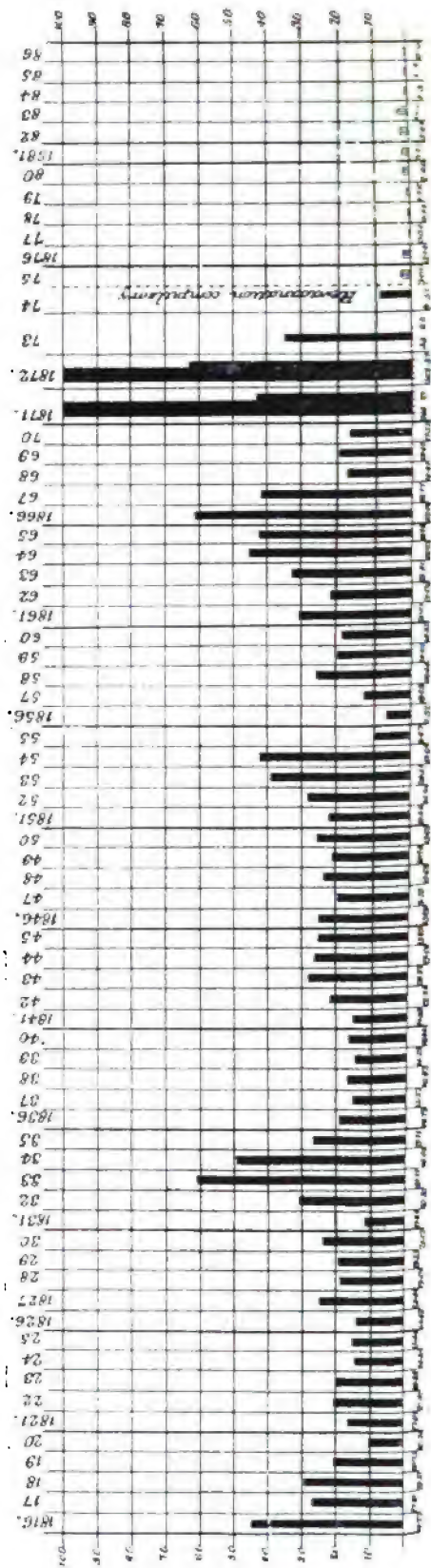
² *Royal Commission's Report*, Q. 6791, &c.

³ Diagram facing p. 282 of *Royal Commission's Second Report* (Appendix No. 2).

⁴ *Royal Commission's Report*, Q. 2644.

DIAGRAM SHOWING THE MORTALITY FROM SMALL-POX IN PRUSSIA FROM 1816 TO 1886 PER 100,000 INHABITANTS.

(The figures from 1816 to 1882 are taken from Dr. M. Schultz's "Impfung, Impfgeschichte und Impftechnik" Berlin 1888; from 1883 to 1886 the diagram is drawn according to the figures given on page 149 of Vol. I. "Statistisches Handbuch für den Preussischen Staat" Berlin, 1888.)



and especially an incompletely protected, residuum in the army. But granting all this, the following figures are striking enough :—

Table showing the Mean Admissions and Deaths per 100,000 per Annum from Small-pox amongst the Troops in the United Kingdom in three Successive Periods.

Period	Admissions	Deaths
1847-60	282	18
1861-74	84	6
1875-88	16	1

It is practically impossible to get for comparison with these rates an unvaccinated population similar in age and character to the army. But in this connection Dr. A. R. Wallace, an opponent of vaccination, has pointed out that in England and Wales in the twenty-three years 1860-82 the mean small-pox mortality of males aged from fifteen to fifty-five years was 17·6 per million. Thus practically the same rate (18 per 100,000) which had prevailed in the army before re-vaccination was enforced there continued to prevail (17·6 per 100,000) in the generally unrevaccinated adult male population of the country at large in later years, while the rate of the army under re-vaccination stationed in the same country fell to 6 in 1861-74, and to 1 in 1875-88.

The Navy.—In the Navy, compulsory re-vaccination dates only from March 7, 1871, while for foreigners—Kroomen, &c.—taken on at ports outside the United Kingdom, there appears to have been no compulsion until

Table showing the Deaths from Small-pox in the Home Force of the British Navy from 1859 to 1889.

Year	Mean strength	Cases of small-pox	Deaths	Ratio per 10,000 of strength	
				Cases	Deaths
1859	19,800	51	4	26·4	2·07
1860	23,500	84	12	35·7	5·10
1861	22,900	85	1	15·8	0·44
1862	20,760	8	1	3·8	0·48
1863	21,570	39	2	18·0	0·92
1864	19,630	199	9	101·3	4·58
1865	20,980	18	0	8·6	0·00
1866	21,200	29	0	13·7	0·00
1867	21,600	30	0	13·9	0·00
1868	23,200	16	0	6·9	0·00
1869	22,100	8	0	3·6	0·00
1870	21,000	24	0	11·4	0·00
1871	22,100	67	4	30·8	1·81
1872	23,000	62	9	26·9	3·91
1873	22,400	7	0	3·1	0·00
1874	22,500	2	0	0·9	0·00
1875	21,600	1	0	0·4	0·00
1876	20,800	0	0	0·0	0·00
1877	21,000	2	0	0·9	0·00
1878	19,000	4	0	2·1	0·00
1879	18,900	0	0	0·0	0·00
1880	22,370	0	0	0·0	0·00
1881	22,140	4	0	1·8	0·00
1882	19,160	8	1	1·6	0·50
1883	22,200	1	0	0·4	0·00
1884	18,570	2	0	1·0	0·00
1885	23,100	2	0	0·8	0·00
1886	21,800	0	0	0·0	0·00
1887	23,700	0	0	0·0	0·00
1888	24,000	4	0	1·6	0·00
1889	24,730	0	0	0·0	0·00

about 1879 or 1880.¹ Once more, therefore, the home force of the navy furnishes the most unequivocal statistical material. Keeping in mind that the order dates only from 1871—though, of course, there would be an increasing amount of re-vaccination before that date—the figures in the table tell a story which he who runs may read. The long rows of noughts in the columns of deaths show a state of protection which has been equalled only under the German law of 1874.

In 'Vaccination Vindicated,' the author wrote as follows regarding the army and navy statistics of the twenty-three years 1860–82 :—

'The year 1871 happens to stand out from the others in three ways. It was the year of chief epidemicity; it was the year of the new navy regulations; and it was the middle year of the twenty-three, there being eleven on each side of it. It may therefore be taken separately as in the following table :—

Mean Annual Small-pox Death-rate per Million living.

Years					1860–70	1871	1872–82
Army	105	210	44
Navy	218	260	62

'These figures can be reasonably interpreted only in accordance with a belief in the efficacy of re-vaccination. Thus between the earlier and the later eleven years a very decided decrease is observable in both services. But in the navy the decrease has been much greater than in the army, because (1) there was greater room for decrease, the absence of compulsion in the navy giving, in 1860–70, a mortality more than double that of the army, and (2) there was greater cause for decrease, the compulsory order intervening between the two periods in the former case and not in the latter. In the 1872–82 period, both being under compulsion, the difference is much less, though the lingering effect of the previous (comparative) laxity is still invisible in the navy returns.'

Small-pox Nurses.—Probably no class of the community is so much exposed to infection from zymotic disease as the nurses in fever and small-pox hospitals. Regarding typhus fever nurses, Dr. Collie, late Superintendent of Homerton Hospital, says that 'the only way in which nurses become seasoned against fever is by taking the disease, which they all do, unless they have had it before.' As to small-pox nurses, the case is very different.

A Vaccination Committee of the Epidemiological Society² reported on the facts regarding 784 nurses and attendants in the Metropolitan Asylums Boards Hospitals. Of the 784, there were 79 who had had small-pox previously, and none of these were infected; 645 had been re-vaccinated before entering on their duties, and the whole of these escaped the disease; the remaining 10 had not been re-vaccinated, and every one of them took small-pox. Surely no more perfect statistical evidence could be conceived of than is afforded by these figures, with their 645 experiments on the one side and their ten control experiments on the other. Commenting on these same facts before the Vaccination Commission, Dr. Thorne said: 'Now here you have people living on the same premises, getting the same food, wearing very much the same quality of clothing, having the same water-supply, the same sewerage, the same drainage, and the same housing. The small-pox absolutely passes over the 645 who had been re-vaccinated, and deliberately seizes upon every one of the ten who had not been re-vaccinated. Then, on the other

¹ Staff-Surgeon T. J. Preston, *Royal Commissioner's Second Report*.

² *Transactions*, vol. v. New Series.

hand, as regards typhus, which has been referred to, there are a number of instances in striking contrast with those as to small-pox. For example, here is a case from Newcastle-upon-Tyne, whence the Medical Officer of Health reports that during 1882 fourteen nurses were employed at the Newcastle-upon-Tyne Infectious Hospital in attendance on cases of typhus. Of the fourteen, nine contracted the disease and two of them died. In the adjacent pavilion nine other nurses were in attendance on small-pox patients. Of this nine, all but one, who had recently had small-pox, were re-vaccinated before coming on duty. None of them contracted small-pox, but one did contract typhus. Thus, where the prophylactic of vaccination was applied, it absolutely prevented the nurses from getting small-pox; where no such prophylactic could be applied, that is to say, in the case of typhus, one of the small-pox nurses, although in a different building, did contract this fever. Now here again you have people living under precisely the same sanitary circumstances, drawn from the same class of life, and comparable in every way but one—some were purposely protected against the disease with which they were in contact, the others were not. Then during the same epidemic the Gateshead Medical Officer of Health writes: "Every nurse who has been more than a fortnight in the typhus wards has suffered from typhus. On the other hand, the only officers who took small-pox were two kitchen girls whom I neglected to vaccinate." So that there again you have the same people living under the same sanitary circumstances; small-pox deliberately seizes upon the only ones whom the Medical Officer of Health had forgotten to vaccinate, the others passing through the epidemic scatheless. That sort of evidence, I may say, is to be found in many an Officer of Health's report from one end of the kingdom to the other. These are not exceptional incidents.'

(4) *The Modification of Small-pox by Vaccination.*—We come now to a different class of evidence. From various detailed sources of information Dr. Abbott¹ has compiled this table, showing that the fatality of small-pox is very much less among the vaccinated than among the unvaccinated. From

Places and dates of observation	Total number of cases observed	Death-rate per 100 cases	
		Among the unvaccinated	Among the vaccinated
France, 1816-41	16,897	16.1	1.0
Quebec, 1819-20	—	27.0	1.7
Philadelphia, 1825	140	60.0	—
Canton Vaud, 1825-29	5,898	24.0	2.2
Darkehmen, 1828-29	134	18.8	—
Verona, 1828-39	909	46.6	5.6
Milan, 1830-51	10,240	38.5	7.6
Breslau, 1831-33	220	58.8	2.1
Wurtemberg, 1831½-35½	1,442	27.3	7.1
Carniola, 1834-35	442	16.2	4.4
Vienna Hospital, 1834	360	51.2	12.5
Carinthia, 1834-35	1,628	14.5	0.5
Adriatic, 1835	1,002	15.2	2.8
Lower Austria, 1835	2,287	25.8	11.5
Bohemia, 1835-55	15,640	29.8	5.2
Galicia, 1836	1,059	23.5	5.1
Dalmatia, 1836	723	19.6	8.2
London Small-pox Hospital, 1836-56	9,000	35.0	7.0
Vienna Hospital, 1837-56	6,213	30.0	5.0
Kiel, 1852-53	218	32.0	6.0
Wurtemberg	6,253	38.9	3.5
Malta	7,570	21.1	4.2
Epidemiological Society Returns	4,624	23.0	2.9
Illinois	1,981	48.6	6.1

¹ *Op. cit.*

the official returns for Bohemia for the twenty-one years ending 1855 he gives another table showing for each year the vaccinated and unvaccinated population, the sick-rate, and the death-rate. Of the vaccinated the mean annual population was 148,122, the cases of small-pox 389, and the deaths 20, or 5·5 per cent. of the cases. Among the unvaccinated the corresponding figures were, population 4,221, cases 355, deaths 105, or 29·6 per cent.

The Influence of Multiple Marks.—No fields for the study of small-pox, and of its course as affected by vaccination, can be equal to those furnished by the great small-pox hospitals. Until 1871 there was only one small-pox hospital in London, at Highgate, the direct successor (though on a different site) of the original 'Hospital for Inoculation and for Small-pox.' Mr. J. F. Marson succeeded Dr. Gregory as physician to this institution, and his observations, extending from 1836–1867, were the means of establishing one of the most important facts within our knowledge regarding this subject. He proved that the value of vaccination as a mitigating agency in cases of attack by small-pox depends very largely on the thoroughness with which the operation has been performed, this thoroughness being gauged by the number, extent, and character of the cicatrices left by vaccination. Mr. Marson, however, was not the first to form this opinion. In a pamphlet published in Perth in 1818, and entitled 'A Few Observations on the Efficacy of Cow-pox,' by James Macfarlane, M.D., it is urged 'that to insure in the meantime as far as possible the full force of the cow-pox action, vaccination should always be performed with at least two if not with three or four punctures,' and that 'one only of the vesicles should be opened.'¹

The following table was handed in by Dr. Thorne in the course of his evidence before the Royal Commission. The table is founded (a) on information given in the 36th volume of the Medico-Chirurgical Society's Transactions, by Mr. Marson, as the result of his observations made during sixteen years 1836–1851, in 8,094 cases of post-vaccinal small-pox; (b) on data derived from Mr. Marson's evidence before the Vaccination Committee of 1871, based on a further experience of 10,661 such cases, and covering the sixteen years 1852–67:—

Cases of small-pox classified according to the vaccination marks borne by each patient respectively	Percentage of deaths in each class, respectively uncorrected*		Percentage of deaths in each class respectively corrected*	
	1836–51	1852–67	1836–51	1852–67
1. Stated to have been vaccinated but having no cicatrix	25·5	40·3	21·7	39·4
2. Having one vaccine cicatrix	9·2	14·8	7·6	13·8
3. Having two vaccine cicatrices	6·8	8·7	4·3†	7·7
4. Having three vaccine cicatrices	3·6	3·7	1·8	3·0
5. Having four or more vaccine cicatrices	1·1	1·9	0·7	0·9
Unvaccinated	37·5	35·7	35·5	34·9

* The terms uncorrected and corrected are used to signify the inclusion or exclusion of those fatal cases of small-pox in which the patient suffered some other disease superadded to the small-pox.

† Should be 4·13.

The figures speak for themselves. They show that the controlling power of vaccination over small-pox is in very close relationship to the number of vaccine marks on the patient's arm. The power of vaccination to protect against fatal small-pox depends on the amount of induced vaccinia, just as in old days the benefit from variolation was indicated by the extent of the

¹ This pamphlet is in the Faculty Library at Glasgow.

resulting eruption; and in practice the united area of four good vesicles (about half of a square inch) may be accepted as the full expression of the vaccinal process, just as a small or moderate amount of a discrete eruption was looked on as proving that variolation had done its prophylactic work.

It will be seen in these figures that the unvaccinated mortality has been practically the same in the two periods, while the vaccinated mortality of every class shows an increase in the later period. The explanation is to be found in the fact that, doubtless owing to a keener search for cicatrices in the latter period, Class 1 includes less than 8 per cent. of the total cases, against over 9 per cent. in the former period.¹ The difference (over 6 per cent. of all the cases) consists of very imperfectly marked cases which had, on the one hand, a lower mortality than the cases said to be vaccinated but without any cicatrix, and, on the other hand, a higher mortality than the definitely marked cases in Classes 2, 3, 4, and 5. The transference, in the second period, of these badly-marked cases from Class 1 to Classes 2-5 had therefore the curious effect of raising the percentage mortality, both of the class from which they were taken, and of the classes to which they were transferred. The change is entirely a statistical one, and does not represent actual increase of fatality in any of the classes.

In the extent of his experience of hospital small-pox in this country, Dr. William Gayton comes next to Mr. Marson. In the hospitals of the Metropolitan Asylums Board he has treated not less than 10,408 cases, and his results are condensed as follows.² The table, it will be observed, classifies the marks according to quality, not number, and is specially valuable as giving the facts for a series of ages. Gayton's evidence is entirely confirmatory of Marson's.

Ages	Vaccinated with good marks			Vaccinated with imperfect marks			Vaccinated but no marks visible			Not vaccinated		
Years	Cases	Deaths	Per cent.	Cases	Deaths	Per cent.	Cases	Deaths	Per cent.	Cases	Deaths	Per cent.
0-2	4	0	0	33	3	9	23	9	41	276	181	66
2-5	57	0	0	150	18	12	96	28	40	401	303	50
5-10	206	2	1	532	27	5	207	40	19	510	180	35
10-15	439	5	1	939	33	3	214	43	20	317	74	23
15-20	608	12	2	1,037	66	6	205	39	19	304	86	43
20-25	389	11	3	848	100	12	167	56	34	174	83	48
25-30	189	19	6	529	80	15	116	35	30	105	56	53
30-40	147	14	10	526	78	15	187	49	26	103	43	41
40-50	79	4	14	186	33	18	65	24	28	49	31	43
50	19	2	11	80	18	22½	46	20	43	30	13	43
All ages	2,065	62	3	4,854	455	9	1,295	352	27	2,169	933	43

Opponents of vaccination sometimes try to found an argument on the largeness of the number of the vaccinated, compared with the unvaccinated, that enter these hospitals. Obviously, however, the numbers have to be calculated as a percentage of the vaccinated and unvaccinated that are to be found in the communities or in the classes from which the hospitals draw their patients. In this table there are 8,234 cases with vaccination of some degree or other, real or nominal. These came from the vaccinated 95 per cent. of the population. The admittedly unvaccinated numbered 2,169, and they were sent in by the unvaccinated 5 per cent. of the population. Thus a twentieth part of the population furnished fully a fifth part of the patients, and,

¹ *Select Committee's Report*, 1871, pp. 236-7.

² *Vaccination Vindicated*, p. 88, also *Royal Commission's Second Report*, p. 245. The Report also contains Dr. Gayton's figures in a much fuller table.

taking only children under five, the unvaccinated twentieth of the community sent in 677 cases, while the vaccinated nineteen-twentieths sent in only 361 cases. Where, as at Highgate, there is a hospital admitting a large number of paying patients, belonging to the better classes, whose children have probably received some sort of vaccination up to a fraction of a unit per cent. of their number, the proportion of 'vaccinated' admissions is bound to be much greater than in a free hospital. This remark applies also to an outbreak occurring in any country—as Wurtemberg—where vaccination is almost universal.

The Small-pox Eruption modified by Vaccination.—The direction in which vaccination acts as a controlling agent in the severity and fatality of small-pox is chiefly in modifying the eruption. The death-rate from discrete small-pox is very much less than from confluent small-pox, and that from the malignant or hæmorrhagic form of the disease is heaviest of all. In 1872 Dr. Russell showed this by a diagram in his Report of the experience of the Glasgow hospital, and quite recently Dr. Barry has demonstrated the same facts in the Sheffield hospitals by means of diagrams similarly constructed, and of which one is here reproduced.¹ It will be noted that each square in the diagram contains 100 smaller squares, so that these latter represent percentages.

In a school census as to vaccination marks, taken in 1868 by Drs. Seaton and Buchanan, they found, among 50,000 children, that of every 1,000 children having no vaccine marks 860 were marked with small-pox; that of 1,000 with one vaccine cicatrix, 6·8 had marks of small-pox; with two cicatrices, 2·49; with three cicatrices, 1·42; and with four or more cicatrices, 0·67.²

The influence of good as compared with inefficient vaccination has been looked at by Dr. Cory from another standpoint.³ Among persons scarred with small-pox who came under his observation at St. Thomas's Hospital and elsewhere, he noted (1) whether there had been any previous vaccination, and (2) if so, what had been the interval between vaccination and small-pox. In 147 cases, 88 were admittedly unvaccinated. The 5 per cent. of the unvaccinated population had thus contributed over 56 per cent. of the persons scarred with small-pox. Next, it is to be noted that the average age of the 88 unvaccinated was 6·6 years. Of persons alleged to have been vaccinated, but having no trace left of the operation, there were nineteen. The average age of attack had in these cases been 11·4 years. With distinct vaccination marks there were 47 cases, and the age of these when attacked averaged from 17·1 to 19·7 years, according to the number of vaccination marks which they exhibited. Commenting on these figures, Dr. Thorne said before the Royal Commission: 'Here you have people drawn from the same class of life, living under much the same sanitary circumstances—for they were nearly all London people—and the difference between contracting small-pox at seven or at nineteen years of age was the difference of being either unvaccinated or having three or four vaccination scars.'

(5) *The Evidence of Small-pox Epidemics.*—The value of vaccination is to be learned, not only from the general absence of small-pox, but from its occasional presence. From epidemics it is possible to observe (a) the prevalence of the disease in countries under vaccination laws of varying degrees of stringency, and (b) the behaviour of the disease towards the vaccinated and unvaccinated in particular communities.

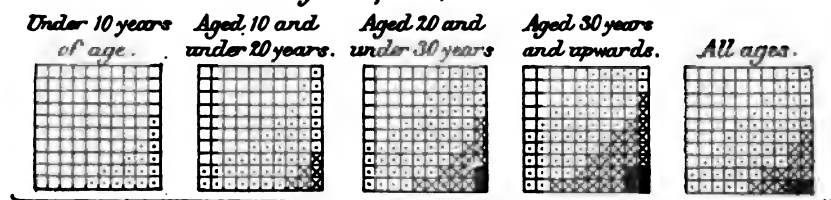
¹ Diag. K. Appendix 4, *Royal Commission's Second Report*.

² Parliamentary Paper No. 275, Session 1881.

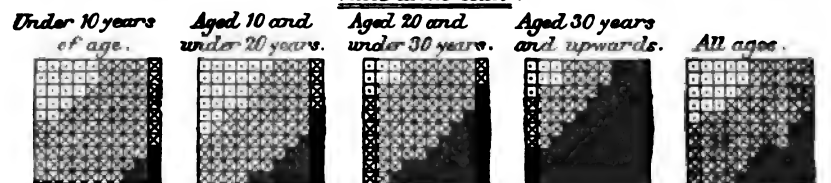
³ *Trans. Epidem. Soc.*, vol. viii. N. S.

DIAGRAM showing the proportion of cases of Small-pox of different types which occurred in persons of the "Vaccinated" and "Unvaccinated" classes respectively at all and certain specified ages in the Winter Street and Sheffield Union Workhouse Hospitals.

Borough Hospital, Winter Street.



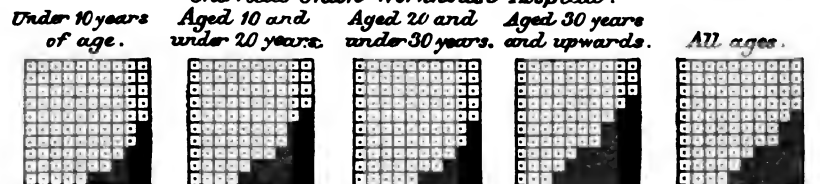
Vaccinated Class.



Unvaccinated Class.

■ *Confluent.* ☒ *Coherent.* □ *Discrete.* □ *Varioloid.*

Sheffield Union Workhouse Hospital.



Vaccinated Class.



Unvaccinated Class.

■ *Confluent (including Coherent.)*
 □ *Discrete (including varioloid.)*

NOTE. Cases with regard to which the Hospital records were incomplete have not been included in this diagram.

(a) *The Epidemic of 1870-73.*—The late Dr. Seaton, in the Report of the Medical Officer of the Privy Council and Local Government Board for 1874, deals with the epidemic of 1870-3. He makes the mortality of children under five years the basis of an interesting comparison. In Scotland, a compulsory law was not enacted till 1863, but it was, as regards the numbers who came under its sway, a very efficient law, so that in this respect Scotland was better off than England as to children under five years old at the time of the epidemic under consideration. In Prussia the law was very lax. The compulsory age was one year, but there was no penalty for disobedience, except in the event of subsequent small-pox, and, naturally, prosecutions were seldom undertaken then. The result was that in Berlin the epidemic found great numbers of unprotected children. Hamburg was still worse off, there being no compulsory law, the only enactment on the subject being that children without certificates of vaccination could not be admitted to the 'schools of the poor.' In Holland, there was, says Seaton, no vaccination law whatever, and in some only of the communes had children to be vaccinated before admission to the communal schools. These five populations, therefore—namely, Scotland, England, Berlin, Hamburg, and Holland—form descending series as regards vaccination; and, as regards small-pox, the following figures show that they form an ascending series, the two facts of vaccination and small-pox standing exactly in an inverse ratio to each other. The death-rates given are those under five years of age per million living at all ages; this standard, though not perfect, being the best available for the comparison:—

Scotland (chief towns), 1871-4	692
London, 1870-2	1,180
England, seventeen unions, most nearly corresponding with the seventeen chief towns, 1881-4	1,180
Berlin, 1871-2	3,448
Hamburg, 1871-2	5,717
Holland (chief towns), 1870-2	6,455

Somewhat similar comparisons for the years 1885-88 are given by Dr. Edwardes¹ for a number of Continental countries with varying vaccination laws.

(b) *The Montreal Epidemic of 1885.*—In Montreal in 1885 there occurred an epidemic whose story has been written by the late Dr. Tomkins, the Medical Officer of Health for Leicester.² The population was 167,000, and though, following on an epidemic in 1879-5, public vaccination had been provided since 1876, yet by 1885, 'from simple neglect on the part of large numbers of the people, who were really not opposed to the operation, a considerable portion of the children of Montreal were unvaccinated.' The first case (an imported one) occurred at the end of February, but the outbreak assumed no alarming proportions until June. By the end of the year 3,164 deaths had occurred, and at one time as many as 1,723 infected houses were under surveillance. To the hospitals, 1,382 cases were admitted, with a mortality of 418, or 31.3 per cent., as compared with the 16 or 17 per cent. which is now the rule in the London hospitals, where the great majority of the patients are vaccinated. But the Montreal hospital mortality was thus not far removed from that which prevailed in the last century in the old Small-pox Hospital of London.³ Of the total, 805 were unvaccinated, of

¹ *Vaccination and Small-pox*, by Edward J. Edwardes, M.D. Lond. 1892.

² *Lancet*, May 26, 1888.

³ Page 399, *ante*.

whom 815, or 89 per cent., died ; and 527 were vaccinated, of whom 108, or 19½ per cent., died. Again, of the 3,164 deaths which occurred in the city up till December 81, only 447 were over ten years old. Thus, of 1,000 deaths 859 were under ten years old. Once more, therefore, we have an approach to last century experiences. As has already been noted, in Geneva in pre-vaccination times 961 deaths in every 1,000 were of children under ten, while in London, in a recent year, only 86 per 1,000 of the vaccinated deaths were under that age. Montreal resembles much more nearly old Geneva than modern London. Once more, the small-pox death-rate in Montreal was nearly 19,000 per million living, which is just about the rate that was experienced once every four or five years in Kilmarnock in the last century. There is yet another resemblance. The total births of the year were 6,808, and the total deaths were 7,825 (or 46·7 per 1,000, as against 26·7 per 1,000 living in the previous five years), so that the deaths largely exceeded the births ; and in Kilmarnock in the last century we find the same thing, for in seven out of nine epidemic years there the deaths exceeded the births. Indeed, in regard to its small-pox experiences, Montreal in many respects carries us back into the last century.

Sheffield Epidemic, 1887-8.—The last epidemic to which reference need be made is that of Sheffield in 1887-8, so ably reported on by Dr. Barry. The first point to be noted is that Sheffield was, as regards primary vaccination, in a very good position. About 98 per cent. of the inhabitants had been once vaccinated. Now the fatality of modern small-pox is a mean between that of the two classes—the unvaccinated with a high fatality, and the vaccinated with a low fatality. In hospitals this often brings out a mean rate of about 16 to 18 per cent. About 79 per cent. of Dr. Gayton's cases were stated to have been vaccinated, though only 66 per cent. bore any marks of the operation, and the total mortality of his cases was 17·4 per cent. But in Sheffield, with its large percentage of vaccinated, the deaths, including those of the unvaccinated, were only 9·7 per cent. of the cases. Opponents of vaccination say that Sheffield was in an insanitary condition. They say the same of Montreal. Let this be granted, and we have two insanitary cities, one ill-vaccinated, with a fatality (in hospital) of 81·8 per cent., and the other comparatively well vaccinated, with a fatality (including both private and hospital cases) of 9·7 per cent.

Sir George Buchanan summarises some of the main facts regarding the enumerated population in the Sheffield epidemic as follows :—

(a) Children under ten years. Among the vaccinated the attack-rate per 1,000 children was 5 ; among the unvaccinated it was 101. The death-rate per 100 vaccinated was 0·09 ; among the unvaccinated it was 44. Therefore, 'for 100,000 vaccinated children the rate of small-pox mortality actually observed in Sheffield gives 9 deaths. For 100,000 unvaccinated children, the rate of small-pox mortality actually observed gives 4,400 deaths.' Among children living in houses actually invaded by small-pox the attack-rate per 1,000 of the vaccinated was 78, and of the unvaccinated 869 ; while the death-rates were 1 and 381 respectively.

(b) Among persons over ten years old, the differences, as was to be expected, were not so marked. The attack-rate per 1,000 of those twice vaccinated was 8, of those once vaccinated 19, and of those not vaccinated 94 ; while the corresponding death-rates were 0·08, 1, and 51. Therefore, 'for 100,000 persons over ten years old and twice vaccinated, the rate of small-pox mortality actually observed would give 8 deaths,' for those once vaccinated 100 deaths, and for those not vaccinated 5,100 deaths.

(c) Among people at all ages, the attack-rate per 1,000 of the vaccinated

was 280, and of the unvaccinated 750, while the death-rates were 11 and 872 exclusively.

(6) *Illustrations of the Influence of Vaccination.*—The above is a mere sample of the statistical evidence in favour of vaccination, and on such a subject statistics ought to have greater weight than any other kind of evidence. But medical men meet with persons who will not take the trouble to study large masses of figures, and who excuse themselves by the easy statement that 'statistics can prove anything.' For people 'who prefer illustrations to statistics,' Mr. Preston-Thomas writes as follows in the '*National Review*' for June 1889 :—'At Terrington the disease appeared in a house occupied by a man and his wife and seven children. According to the statement of Mr. Secombe, the medical man in attendance, the wife had had small-pox in childhood, the husband had been vaccinated when an infant, but was an ardent anti-vaccinator, and had allowed only one of his children to be vaccinated. All the family were attacked, with the single exception of this child, who escaped altogether, though sleeping in the same room and in the same bed with some of the others. The wife and two of the children died, the four unvaccinated children had the confluent form of the disease, and were much disfigured; the father had a very mild attack, and was able to nurse the rest. The report (December 1884) of a coroner's inquest in Shoreditch shows that small-pox broke out in the house of a determined anti-vaccinationist, and his three unvaccinated children all died. In St. George's, Hanover Square, in the same year, a report of the vaccination officer described the case of a family of four children of whom only one was vaccinated, and this one alone escaped. At Felling, in 1882, thirty-seven persons caught small-pox; thirty-two had been vaccinated, and all but one had the disease in a modified form and recovered, the exception being a woman, whose attack was complicated by bronchitis and pregnancy. The other five were unvaccinated, and four of them died. At Hull, in 1883, small-pox broke out in a house where there were nineteen lodgers. Of these, sixteen were re-vaccinated, one refused re-vaccination because he had been vaccinated in his youth, and two others also refused, though they had never been vaccinated. The last three caught small-pox, and the two who had never been vaccinated died. At Billericay, in 1888, three young men, one unvaccinated, the others vaccinated in infancy, left home to look for work. About eleven days after returning, the unvaccinated man fell ill with small-pox, which killed him. All three had occupied the same room the whole time. The vaccinated men escaped. The father and mother of the unvaccinated man refused vaccination, and both took small-pox, the former slightly, and the latter severely. At Chatham, in 1884, in a household consisting of father, mother, and three children, one child only was unvaccinated. It died of small-pox. At Kimblesworth, Durham, there was, in 1885, an outbreak of small-pox which attacked thirty-five vaccinated and three unvaccinated persons. The latter alone died. At Hyde, Cheshire, in 1886, there was an epidemic of small-pox, by which 122 persons were attacked. All those vaccinated recovered, eighty-five of them after very mild attacks, but "the five fatal cases were all without exception in undoubtedly unvaccinated persons." At Edenbridge, in 1886, four navvies were infected from a case so mild as not to be recognised as small-pox. Two of them had been vaccinated, two were unvaccinated. The former had very slight attacks, the latter died. At Rowley Regis, in 1888, there was an outbreak which extended to seventy-persons, of whom fifty-four were vaccinated and sixteen were unvaccinated. Among the fifty-four there were thirteen confluent cases, with one death. Among the sixteen, there were fourteen confluent cases, with five deaths. At

Claygate, Surrey, in 1885, small-pox broke out in a household consisting of father, mother, three sons, four daughters, and a lodger. The eldest son and the lodger had been re-vaccinated, and the second son had been once vaccinated. These three alone escaped, none of the other seven had been vaccinated; all took the disease in a severe form and one of them died.

'These instances (except the first three) have been taken almost at random from the reports of medical officers of health, and it would be easy to add to them to any extent from the same source.'

GENERAL CONCLUSIONS REGARDING DURATION OF VACCINAL PROTECTION

The following propositions appear to me to be deducible from the knowledge which has accumulated up to the present time regarding vaccination and small-pox :—

I. *Susceptibility to re-vaccination or variolation returns gradually, subsequently to vaccination.*

(i.) At first, after the completion of the vaccinal process, there is no reaction to local insertion of virus, either variolous or vaccinal.

(ii.) By-and-by a certain abortive or imperfect reaction is possible—a shortened cycle—a spurious pock.

(iii.) Still later, a perfect result is in many cases obtainable. In children of six years old, Layet, of Bordeaux, was successful in about 40 per cent. of his re-vaccinations.¹

II. *Susceptibility to attack by small-pox increases with length of interval since vaccination.*

Dr. Barry's Sheffield report, pages 181-2, contains the following figures :

Age	Attack per cent. of vaccinated inmates of houses in Sheffield invaded by small-pox *	Age	Attack per cent. of vaccinated inmates of houses in Sheffield invaded by small-pox *
0-5	5.6	15-20	41.3
5-10	9.9	20-30	38.7 †
10-15	25.2	30-	17.0 †

* The exposure must have been very real, as the great majority of the Sheffield houses were of very small size, over 81 per cent. having a rateable value not exceeding 112. 5s. annually, and less than 4 per cent. over £21. And among the unvaccinated persons similarly exposed 83 per cent. were attacked of those under five years, 92 per cent. of those from five to ten years, 98 per cent. of those from ten to fifteen years, and 86 per cent. of those from fifteen to twenty years.

† Probably including the bulk of re-vaccinated persons.

III. *Susceptibility to death by small-pox increases with length of interval since vaccination.*

From Dr. Barry's Sheffield report, pages 188-4, we take the following figures :—

Age	Deaths per cent. of vaccinated persons attacked by small-pox in Sheffield	Age	Deaths per cent. of vaccinated persons attacked by small-pox in Sheffield
0-5	0.8	15-20	1.9
5-10	2.2	20-30	5.4
10-15	1.7	30-	10.6

It is to be borne in mind that a large part of this increased death-rate is not due to lessening influence of vaccination, but to the natural law of small-pox fatality, by which the rate rises from the third quinquennium of life onwards, both in the vaccinated and the unvaccinated.

¹ *Rapport au Conseil Municipal sur le Service de Vaccinations et Revaccinations publiques pendant l'année 1884.* Par M. A. Plumeau. Bordeaux, 1884.

IV. *Resistance of the vaccinated to attack by small-pox outlasts resistance to re-vaccination or variolation.*

The data here are scanty, but the following figures may be compared: A, calculated from Layet's tables as given in the Bordeaux Report, and B, from Dr. Barry's Report, pages 181-2:—

A. Percentage success of re-vaccinations		B. Percentage success of small-pox attack under exposure*	
Age	Success	Age	Success
6-10	42	5-10	9.9
10-12	44	10-15	25.2

* See note to Proposition II.

V. *Resistance of the vaccinated to death by small-pox outlasts very considerably resistance to attack by small-pox.*

This is shown by a comparison of the figures already given under II. and III., relating respectively to susceptibility to attack and susceptibility to death by small-pox.

Percentages as given in II. and III.

Age	Attack	Death	Age	Attack	Death
0-5	5.6	0.8	15-20	41.8	1.9
5-10	9.9	2.2	20-30	88.7*	5.4
10-15	25.2	1.7	30-	17.0*	10.6

* Probably including the bulk of re-vaccinated persons.

VI. *The drift towards acceptance of attack and of death by small-pox is much slower in course and much less in ultimate amount in the well vaccinated than in the badly vaccinated.*

This is illustrated by Dr. Russell's Glasgow tables, showing the drift towards severity of type of small-pox, as indicated by the extent of the eruption.

In Persons with good Vaccination Marks.

Age	Eruption		
	Percentages		
	Rare	Copious	Confluent
0-9	86	14	0
10-19	78	18	4
20-29	72	24	4
30-39	53	41	6
* 40-	80	10	10

In Persons with bad Vaccination Marks.

Age	Eruption		
	Percentages		
	Rare	Copious	Confluent
0-9	84	8	8
10-19	44	42	14
20-29	38	40	27
30-39	18	35	47
† 40-	9	33	58

* Founded on only 11 cases at this age.

† Founded on only 12 cases at this age.

VII. *The rule that re-vaccination should be performed at ten to twelve years of age is not founded on any theory that primary vaccination has then lost its protective power.*

The facts are strongly against such a belief. The Sheffield evidence goes to show (1) that from five to ten years of age only 10 per cent. of vaccinated persons exposed to attack by small-pox are actually attacked, and from ten to fifteen years 25 per cent. (Proposition II.); and (2) that from five to twenty years of age only 2 per cent. of vaccinated persons die of the disease (Proposition III.) It is on account of the liability of the 25 per cent. to be attacked, and of the additional liability of 2 per cent. out of this 25 per cent. (i.e. 0.5 of the original 100 persons) to die, that re-vaccination is desirable at ten years of age. The power of infantile vaccination against attack by small-pox remains to perhaps at least one half of its original extent at twenty years of age (see Proposition II; at ages fifteen to twenty, 41 per cent. were attacked when living in invaded houses); and its power against death exists to a very considerable extent *all through life*, as abundantly shown by the statistics of the great small-pox hospitals.

ANTI-VACCINATION

Opposition to vaccination has been excused mainly by three allegations: (i.) That vaccination neither prevents nor modifies small-pox; (ii.) that it causes other diseases; and (iii.) that it is unnecessary, as small-pox is only slightly infectious and can be prevented by isolation in hospitals.

Anti-Compulsion.—Behind these objections, however, there is, in the case of many anti-vaccinationists, the political doctrine of the liberty of the subject, which has probably led them to take up a position regarding vaccination itself that their common sense would have shown them to be untenable, had the question not been for them resistance rather to compulsion than to vaccination. Their attitude towards compulsion is curiously illustrated in connection with what they call the 'Leicester system' of sending to hospital all small-pox cases. While they do not hesitate to point to Leicester for evidence that vaccination is unnecessary, they are as much opposed to compulsory hospital isolation as to compulsory vaccination, and assert that it is voluntary isolation and not compulsory that protects Leicester—as if it made any difference in regard to checking an outbreak whether cases go willingly or unwillingly, provided all of them do go to hospital, or as if optional removal to hospital would have any meaning at all in a place where the option resulted in unanimous refusal, instead of unanimous acceptance, of the isolation in question. Excepting, however, in so far as the incidence of the compulsory laws has been used to illustrate the value of the preventive itself, the political question is outside the scope of this article. But the contentions as above classified fall to be referred to here.

(i.) Evidence has already been given of the influence of vaccination in protecting against both attack and death by small-pox, and some objections have been incidentally dealt with. With reference to a contention that the diminution of small-pox which accompanied the introduction of vaccination was due to the gradual cessation of inoculation, it has been shown that in places where inoculation never flourished, the practice of vaccination had the same effect as elsewhere on small-pox mortality. And the difference, which we see in the present day, long after the abolition of variolous inoculation, between vaccinated and unvaccinated communities, between vaccinated and unvaccinated portions of the same community, and between vaccinated and unvaccinated members of the same families, show that, independently of inoculation, vaccination acts in opposition to small-pox.

Bearing on the assertion that the fall of small-pox is due to sanitation it has been shown that other diseases, like measles and whooping cough, mainly belonging, like pre-vaccination small-pox, to childhood, have been practically unaffected by sanitation; and that the group of agencies to which in the present day the adjective *sanitary* is usually applied, such as improved water supply, improved excrement removal, increased living room, and better ventilation, have had their effect less on zymotics as a whole than on particular diseases, like cholera, enteric fever, and typhus, whose causes lie in the particular evils which these agencies are fitted to combat. At the same time it has been agreed that all that makes for cleanliness in eating and drinking and breathing has a good influence over all diseases that trouble the human body, zymotic, constitutional, or local, and upon small-pox among the rest. Of the particular agencies which have been mentioned, increased living room and purity of air have the most effect against small-pox. But the ventilation which will extinguish a disease like typhus, whose virus cannot travel through more than a few feet of pure air, must have much less effect on variola, whose influence may extend a quarter of a mile or more from a hospital in which its cases are congregated. And the facts that have already been stated regarding the age-incidence of small-pox cannot be sufficiently explained on any theory of sanitary influence. It has been shown, too, that the age distribution of fever has been in no way altered in the course of the great decline of its mortality as a result of sanitation.

A contention has been made, based chiefly on Jurin's statistics, that small-pox hospital fatality is practically the same now as in the last century. But it has already been shown that Jurin's statistics have nothing to do with hospital practice, and that even for fatality rates outside of hospitals they are of little or no value, as they do not take into account the age of the patients, and do not include cases under two years old. It has also been shown that, as a matter of fact, small-pox hospital fatality has decreased very greatly since the last century.

It is alleged that the percentages of vaccinated and unvaccinated in modern small-pox statistics are unreliable, partly because doctors are dishonest; partly because the unvaccinated are in an undue proportion a weakly class, whose vaccination has in many cases been on that account postponed, who belong to the poorer part of the community, and who live under unsanitary conditions; and partly because it is impossible to distinguish vaccination marks on the arm of a patient suffering from confluent small-pox. The first reason need not detain us. Regarding the second, it may be pointed out that only about one per cent. of children have their vaccination postponed, that the postponement is usually only for two months, and that it may be due to the most trivial and temporary cause, such as a speck of eczematous or other eruption, a cold, or a passing derangement of the bowels. And in Sheffield Dr. Barry found that the fatality among the unvaccinated richer classes was no less than among the poorer people, living in the worst surroundings.

Regarding the last allegation, that vaccination marks cannot be distinguished in confluent cases, and that these are therefore set down as unvaccinated, Dr. Gayton told the Royal Commission that it was quite exceptional for cases to be received later than the third day of the eruption, and where it was by the end of that day so confluent as to obscure the marks, the cases were not entered as unvaccinated, but as 'vaccinated, without evidence.' In reply to a question on a similar point, Mr. Sweeting, late Medical Superintendent of the Fulham Hospital, said that among 2,584 patients under his care, in only three or four were the marks obscured, as cases usually came very early, in the papular stage. And finally, sup-

posing that in any published list of small-pox cases the 'doubtful' are added to the vaccinated, there still remains to them in regard to fatality an enormous advantage. In the 'Encyclopædia Britannica,' Dr. Creighton points to Dr. Vacher's cases in an epidemic at Birkenhead in 1877, where 80 per cent. of those entered, in regard to their vaccination, as 'unknown,' had been reported by parents or others to have been vaccinated in infancy. But if he had taken even the extreme course of adding the whole of the 'unknown' to the 'vaccinated,' he would yet have got only 40 deaths (or 9 per cent. among 448 vaccinated, against 58 deaths (or 78 per cent.) among 72 not vaccinated. And these figures had been published in that form¹ before the Encyclopædia article was written.

(ii.) *The Alleged Risks of Vaccination.*—The following list comprises most of the various and varied evil results which have been attributed to vaccination at one or another time and by one or another person:—

Cow-pox mange, cow-pox ulcer, cow-pox abscess, and cow-pox mortification; the transformation of the human countenance into the visage of a cow; physical degeneration of the species, the human race becoming small, thin, mean, bald-headed, and short-sighted; mental degeneration as shown in every walk of life—in literature, painting, and music; angioleucitis, atrophy and debility, blindness, boils, bronchitis, bullæ, cancer, cellulitis, cholera, convulsions, delirium, diarrhoea, diphtheria, dyscrasia, eczema, enteric fever, erysipelas, erythema gangrenosa, glandular disease, herpes, impetigo, increased severity of measles and phthisis, leprosy, lichen, marasmus, meningitis, paralysis, pityriasis, pneumonia, prurigo, pyæmia, pyrexia, rickets, scaldhead, scarlatina, scrofula, septicæmia, skin disease, syphilis, tabes mesenterica, tuberculosis, typhus fever, variola, whooping cough.

Since the Registration Acts came into force, they have been appealed to again and again for proof of the dangers of vaccination, and various statistical returns have for this purpose been obtained through members of Parliament. At no time has such inquiry been admitted by anti-vaccinators to be barren of results. The method, indeed, is such as could hardly fail. It is safe to assert that at any given time there will be found some diseases which show a rising mortality, and others which show a falling mortality. The *rise* can be attributed to vaccination, and the *fall* can be used to illustrate the argument that some diseases have diminished without any such aid as vaccination, and that so might small-pox. The weak point in the scheme is that maladies which at one time may be rising at another time may be falling. The public, however, have short memories, and hence we find that while enteric fever, cholera, and scarlatina are at one time exhibited as striking examples of diseases whose increase is due to vaccination, at a later date their decrease is triumphantly pointed to as proof that zymotics may be controlled independently of any Jennerian prophylactic. Recently, indeed, the allegation has taken a new shape. Decrease of enteric fever in this country is still held to show that vaccination for small-pox is needless, but at the same time a discovered increase of enteric fever as a registered cause of death in India is adduced to show that vaccination is mischievous.²

Syphilis.—But in an ocean of error there may be a droplet of truth, and so it is here. During the sittings in 1871 of the Commons' Select Committee on Vaccination, the attention of the profession was arrested first by one, and immediately afterwards by a second, series of cases of what no less an authority than Mr. Jonathan Hutchinson declared to be, unmistakably, vaccinal syphilis. So long ago as 1857 Mr. Hutchinson, in reply to a

¹ *Vaccination Vindicated*, p. 71.

² *Vaccination Inquirer*, 1892, p. 108.

series of questions issued by Sir John Simon on this subject, wrote: 'I believe that I have seen four or five instances in which local syphilitic affections (in some cases followed by general infection) were induced by vaccination.' But from that time till 1871 he did not meet with a single case, either at the Hospital for Diseases of the Skin or elsewhere, in which he had any reason to believe or suspect that syphilitic disease had been communicated by vaccination.¹ Again, since 1871, Mr. Hutchinson has been consulted about probably nearly every case of syphilis in England supposed to be due to vaccination, and in 1887 he stated that, 'though he had been diligently on the look-out for similar cases during the ten years' interval (since his last previous case), he had failed to meet with any.' He has altogether met with about six instances (including those of 1871) of children from whose arms vaccine lymph has been taken with the result of conveying syphilis to about twenty-six persons. Two or three other single cases have been published by others; and about eight years ago a medical man, after several unsuccessful attempts, succeeded in inoculating himself with syphilis from the vaccine vesicle of a child developing a syphilitic rash.

Abroad, cases would appear to be much commoner, and the smallness of the number that have occurred in Great Britain is doubtless largely due to a more careful practice of vaccination. But while the danger is infinitesimal it is real, and has to be guarded against with scrupulous care. At first it was believed by Mr. Hutchinson and others that blood must have been inoculated wherever syphilis was produced. But while blood is to be studiously avoided, it is now known that lymph apparently pure may convey the syphilitic poison. The precautions to be observed are those laid down by the Local Government Board, and already quoted. The important points are, a sufficient examination of the body of the vaccinifer, the use only of clear lymph that freely flows from the vesicles without visible admixture of blood, and the avoidance of first children as vaccinifers. Of course, in private vaccination the operator will usually have for additional guidance his knowledge of the family history. Where parents have scruples on the subject, calf-lymph can be used—as is universally done in Germany—without the faintest possibility of syphilitic contamination. The writer has elsewhere ('Edin. Med. Jour.' 1889-90) discussed at some length the theory put forth by Dr. Creighton, that none of the alleged cases are really syphilitic, it being impossible to transmit such a disease by vaccine lymph, and that the symptoms are due to an exhibition, on the part of the vaccine sores, of an 'unconscious memory' of the original cow-pox from which they had been derived perhaps seventy or eighty years before.

Statistics of Syphilis Mortality.—That the increase in the registration of syphilis as a cause of death, which the public records show to have been going on for many years, depends to any extent on the practice of vaccination, is a groundless supposition. It is true that the deaths registered from syphilis in children under one year old in England and Wales were only 255 in 1847, and were 1,733 in 1874, and it is true also that the greatest proportional increase was in 1854, or just the year after the Compulsory Vaccination Act came into force. But, as Mr. Preston-Thomas says,² 'The rise between 1853 and 1854 is easily explicable. In the analysis of the death certificates of 1854 (as will be seen on examination of the Registrar-General's reports), a new plan of tabulation was adopted, as the result of the Statistical Congress which had been recently held at Brussels; and a large number of deaths, which had before been unclassified, now began to be relegated to particular diseases. Thus we find that the deaths under the heading

¹ Report by Select Committee of 1871, Q. 8,212.

² National Review, June 1889.

'Causes not specified,' which had been 6,900 in 1853, declined to 5,663 in 1854, and that the difference of 1,237 was distributed over different headings. It was in this way that the figures in the column for 'Syphilis' were swollen. As to the gradual rise in registrations from infantile syphilis and its alleged relationship to the number of 'public vaccinations' performed in England and Wales, it is to be noted that, taking the years 1852-1882 (with the exception of 1872-8, which for certain reasons are not comparable), and comparing each year with its predecessor, on twelve occasions an upward or downward movement of vaccination was accompanied by a movement of similar direction on the part of syphilis, and on seventeen occasions the reverse was the case—when vaccinations rose syphilis fell, and when vaccinations fell syphilis rose.

The writer has elsewhere¹ shown from the Registrar-General's figures (1) that in Scotland, vaccination not being compulsory under six months, no less than 65 per cent. of the deaths from syphilis at all ages take place before the age of vaccination, and that in the second half year of life, or immediately following vaccination, the deaths fall to 11·6 per cent., or less than one-fifth of those in the pre-vaccination half year; (2) that the figures for England are almost identical, though the law enforces earlier vaccination than in Scotland; (3) that while the percentage under one year of age of the syphilis deaths at all ages in Scotland has increased from 70 previous to compulsory vaccination to 77 since compulsory vaccination, the whole excess of 7 per cent. is accounted for by infants under three months of age—the three months of non-vaccination. 'What, then, is the meaning of the increased registrations of syphilis as a cause of death? In elucidation of this question, I have to point out that there is one other period of life besides infancy in which deaths from syphilis show a great proportional increase. That period is advancing age. If we divide life into three epochs—(1) childhood; (2) adult life up to fifty-five years; and (3) all ages over fifty-five—we find that while in the last period the total syphilitic mortality is much smaller than in the others, yet in the last, as in the first period, deaths from syphilis have considerably increased. Obviously, vaccination can have nothing to do with this. And there is only one answer that will satisfy all the facts of the case, namely, that the change to a very large extent depends on improved knowledge of the disease by medical men. It is a question of diagnosis. The symptoms of primary and secondary syphilis in young adults were about as well known thirty years ago as they are now; not so the manifestations of congenital syphilis in children and the obscure tertiary affections of later life. One of the commonest results of congenital syphilis is premature birth. That fact is better understood than formerly, and some fraction of the enormous decrease (from 1,048 per 1,000,000 in 1850-4, to 476 per 1,000,000 in 1875-9) in deaths registered from this cause is doubtless due to the substitution of the term syphilis. So, too, congenital syphilis may cause brain disease ending in convulsions; and here, again, part of the diminution consists of a transference of deaths from convulsions to syphilis. These views are in exact accord with what we have already discovered—that it is in the first three months of life that the great bulk of increase has appeared.'²

Leprosy.—In this country, with leprosy almost unknown, the question as to whether it can be conveyed by vaccination is hardly a practical one. But in the West Indies the disease is largely on the increase, and in 1887 the Governor of Trinidad issued a circular to medical men there asking whether they believed it to be communicable in this way. In nearly every case the reply was in the negative. In a country where leprosy is so common, it is

¹ *Vaccination Vindicated*, pp. 136-8.

² *Op. cit.*, p. 139.

obvious that the sources of fallacy in regard to any alleged instance are likely to be numerous, while the theory that the cause of the disease is to be found in bad food appears fully to cover the facts of the case. In New Zealand, Sweden, Norway, and Iceland, leprosy has rapidly diminished coincidently with the spread of vaccination, and while the vaccination age in British Guiana is six months, the earliest age at which Dr. Cartor has seen the disease there is eight years.

Erysipelas.—Most practitioners who have had any large experiences of vaccination must have seen occasional cases in which the operation has been followed by erysipelas; and medical men who have only a slight acquaintance with the practice must yet be aware from their everyday knowledge of the healing of wounds, that even so small a lesion as that caused by vaccination may have this untoward complication. The usual causes having to do with the occurrence are faulty constitution, bad hygienic surroundings, foul lancets or points, dirty vaccination shields, and infection, directly or indirectly, with the poison of the disease. In Norwich in 1882 several cases occurred among the children vaccinated on one particular day by the public vaccinator, and four of them died; and of the fifty deaths that are annually registered, as due to 'cow-pox and other effects of vaccination,' the great majority have an attack of erysipelas for their immediate cause—an attack which in many of the cases might have come on, independently of the vaccination, after the next accidental scratch or abrasion of the skin. The likeliest way to prevent erysipelas from following vaccination is to adhere rigidly to the rules already quoted, as laid down by the Local Government Board.

(iii.) *Isolation.*—The last objection to vaccination which we have to consider is that it is needless, as in isolation a better preventive is to be found, without any of the disadvantages of the Jennerian prophylactic.

Leicester.—The argument is mainly founded on the experience of Leicester. But, as a matter of fact, Leicester is not an example of the effects of isolation as a substitute for vaccination. It can be cited only as an experiment in isolation, plus a certain amount of vaccination. The great bulk of the inhabitants of Leicester have been once vaccinated. It is the children only, or rather a great majority of them, who are unprotected. And when a case of small-pox occurs, the arrangements for its removal are under the superintendence of the Medical Officer of Health, himself a thorough believer in vaccination. Naturally, therefore, he sees that the escort which conveys the patient to the hospital outside the burgh is a vaccinated and re-vaccinated escort, and that in the hospital the patient has round him a cordon of vaccinated and re-vaccinated nurses and attendants; so far as possible, too, those who have been in contact with the case are also given the protection of vaccination. Indeed, isolation of infectious disease, whether scarlatina, typhus, or small-pox, is meaningless unless an administrative cordon of insusceptible persons is available. Not only so, but round the town of Leicester itself there is a wider cordon, consisting of the fairly vaccinated millions that inhabit England. And since the Leicester experiment was begun, it has not been subjected to the test of any general epidemic like that of 1870-8. After it has passed unscathed through such an ordeal, it will be time enough to talk of imitating its methods. But as regards the legislative aspects of the question, under compulsory isolation things would be no better than they are at present. To such a measure, as has already been noted, there would be as much objection as there is to compulsory vaccination, and the writers in the organ of the Anti-Vaccination Society do not hesitate to declare that they are antagonistic to the one compulsion as well as to the other.

And just as they base their opposition to vaccination largely on the Leicester experiment, so they would point to Keighley in justification of their opposition to isolation. For in that town there is neither vaccination nor isolation, and yet it has happened to remain as free from small-pox as Leicester. The fallacy of founding an anti-isolation argument on Keighley is no greater than that of founding an anti-vaccination argument on Leicester. In the same way it would be open to argue that because in a battle one particular soldier had, without evil consequences to himself, discarded his armour, offensive and defensive, therefore the whole army might safely do the same.

The Future of Small-pox and Vaccination.—The question has lately been raised by an opponent of vaccination, whether, independently of vaccination, small-pox is not now disappearing from this country just as the plague did about the end of the seventeenth century. The view that this may be so is largely based on the notion that small-pox is a foreign skin disease which took on infectivity as a secondary matter, and that it was practically unknown in England until three centuries ago or thereby. We have seen, however, that this opinion is entirely erroneous. And in past times, after every epidemic of plague, it might have been urged that the outbreak was simply the last flare of the candle, and that the disease had at length taken itself away for good and all. Nor could anyone have demolished such prophecies, for indeed the only demolition would consist in the reappearance of the plague.

But ultimately it did die. And it will not do to assume that throughout the world, from now till the end of time, small-pox will continue to rage epidemically unless prevented by universal vaccination. Just as leprosy has died out from this country, and is even now dying out from Norway and Sweden and Iceland, so may small-pox, or scarlatina, or measles, or any and every zymotic disease die out in the future from this and every other land. And, quite conceivably, small-pox, rather than either scarlatina or measles, might be the next in order of the maladies to disappear from the civilised world. But with the pandemic of 1870-78 so fresh in our memory, with such outbreaks as those of Montreal and Sheffield at a still more recent date, with the evidence we have of the undiminished virulence and fatality of small-pox wherever it gets fair hold of a population, and with the overwhelming proofs of the different behaviour of the disease in the present day as between vaccinated and unvaccinated countries, and between the vaccinated and unvaccinated in stricken towns, and in the wards of small-pox hospitals—with all these facts before us, it would be no less than madness to assume that the time has yet arrived when we can safely afford to loosen the bonds with which Jenner has taught us to restrain the ravages of one of the most foul and loathsome distempers that afflicts the human race in this era of history. When small-pox does go, the world will be well rid of it, but while the bane remains with us, so also must remain the antidote.

CHICKEN-POX

In the article on small-pox and vaccination, we have held that in the last century modified small-pox often presented such characters as, in presence of the then prevalent opinion that second small-pox was an impossibility, prevented the variolous nature of the attack from being recognised or accepted. It has been shown that the adjectives stone-, water-, wind-, crystalline, verrucose, &c., were applied to particular forms of pocks, according to the various modifications undergone by the variolous phenomena. Among other such names, we often meet with that of chicken-pox or varicella. It is indeed as old as Rhazes, and it is manifest that very often it has

been applied to variolous diseases. Indeed, it was not uncommon to blame small-pox after inoculation, on the use of varicella in mistake for variolous lymph for the protective operation. Chicken-pox so called, therefore, was an inoculable disease. Morton, the contemporary of Sydenham, thought it a milder kind of small-pox, and so late as 1792 Niedt, of Halle, held that it arose from the degenerated matter of that disease. Nor have there ever been wanting eminent physicians to assert the oneness of the maladies. In 1820, Thomson, of Edinburgh, argued with great ability, and after laborious research, that chicken-pox was simply one of the manifold forms of modified small-pox, and still later Hebra has again maintained the thesis of their identity. It may be surmised that the particular fact which must have formed the basis of all such views is that the outstanding characteristic of one of the forms of modified small-pox is its watery or vesicular eruption. And looking to the fixity of epidemic form which modifications of small-pox have occasionally assumed, the confusion which has existed is not to be wondered at.

Gee¹ states that Fuller, in 1730, was the first to insist on the specific difference between the two maladies, but it was Heberden in 1767 who really established the theory. Even Heberden, however, did not elicit the whole truth. He looked on chicken-pox as an inoculable disease, and it was left to Bryce, of Edinburgh, to demonstrate by a series of experiments that all attempts at its inoculation failed, 'without having produced even a vesicle on the arm where the ichor was inserted.' Trousseau similarly failed.

The period of incubation has been variously estimated. Gregory states it at from four to six days, and Trousseau at from fifteen to twenty-seven days. The question would bear further investigation, but recent opinion sets it down at about a fortnight.

The eruption appears without any previous sickness, or at the most with less than twenty-four hours' fever. It begins on any part of the body, and is added to irregularly by fresh crops for four or five days, during which time the constitutional symptoms are equally irregular, both as to severity and intermittence. The vesicles consist of a single cavity, with a very thin covering, and with little or no hardness or elevation of base. Their shape varies, is often oval, and they are not umbilicated. They increase in size, and on the second or third day the clear watery contents become more or less turbid. Within a week thin crusts form, and leave no cicatrix, unless sores have been caused through friction or other maltreatment. Chicken-pox and small-pox are not protective the one against the other. The characters of the disease are very constant, neither epidemics nor individual cases differing much from each other. It very rarely attacks adults, even if they have not suffered from an attack in childhood. In 727 cases at the Children's Hospital, 177 were in the first year of life, 187 in the second, 78 in the third, 100 in the fourth, and 96 in the fifth, after which the numbers rapidly diminished to seven in the twelfth year. (Gee.)

The Registrar-General annually records a few deaths from chicken-pox. Authors, on the other hand, say that it is seldom or never fatal unless from convulsions or concurrent disease. Dr. Ogle is inclined to set down many of its recorded deaths to small-pox. But there has probably been too great tendency to assume that whatever had been diagnosed as chicken-pox, and afterwards proved fatal, must have been small-pox. Very likely some—perhaps nearly all—of the deaths registered as chicken-pox were not due to that disease, but it would be rash to conclude that they were all, or nearly all, due to small-pox. About four years ago the writer saw in consultation a sudaminal eruption which was suspected to be either small-pox or chicken-

¹ *Reynolds' Medicine.*

pox, but which really depended on the free perspiration accompanying an attack of pneumonia. Unless where the appearance on the skin were sufficient to decide the question, then in every death registered from chicken-pox it would be necessary to make a *post-mortem* examination in search of local disease before, by exclusion, the fatality could be attributed to variola. It is not conceivable that confluent small-pox could be mistaken by any medical man for chicken-pox. It is only cases of varioloid, or of discrete small-pox, that could be confused in this way. Now the fatality of discrete small-pox is only about one per cent. in children, and varioloid is practically never fatal. If then we were to suppose that all deaths registered from chicken-pox were really due to small-pox, we would have to argue that for every death there had been a hundred cases. And as the deaths registered from chicken-pox in the year 1890 were 95, we would be forced to conclude that in that year there had been 9,500 cases of unrecognised small-pox, though in the same year only 16 deaths were registered from small-pox recognised as such.

The deaths from chicken-pox from 1855 to 1887 inclusive are given by the Registrar-General in the Vaccination Commission's first Report (p. 115). From these figures the rates per million have been calculated by Dr. Edwardes¹ as follows: 8, 8, 2, 8, 2, 2, 2, 2, 8, 8, 2, 2, 2, 2, 8, 8, 8, 8, 8, 5, 4, 5, 5, 4, 4, 5, 4, 8, 4, 8, 8, 8. The actual deaths in these years amount to 1,420, which, on the above supposition, would represent over 140,000 cases of undiagnosed small-pox. The fact that in the small-pox pandemic of 1870-8 only the average number of chicken-pox deaths were registered, seems to show that the former disease is not often set down as the latter. And if it be suggested that the altered age-incidence of small-pox depends in any degree on the registration of fatal infantile variola as varicella, it is sufficient to point to the fact that in these pandemic years the whole of the deaths from chicken-pox might be added to those from small-pox without materially affecting the age subdivisions of the latter disease.

As in small-pox, the length of infectivity of chicken-pox will depend on the falling of the crusts, which usually become detached in parts rather than entire. After exposure to infection, a quarantine of eighteen days before re-admission to school is insisted on by the Association of Medical Officers of Schools—this being based on the opinion that the incubation period is a fortnight.

¹ *Op. cit.*



VITAL STATISTICS

BY

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VITAL STATISTICS

VITAL statistics are the foundation of sanitary effort, and the basis of the work of medical officers of health.

It is desirable, therefore, that all persons interested in sanitary science should know what data are at their disposal, and how to use safely the various statistics placed before them.

Most of these figures are of interest mainly as tests of the sanitary condition of the populations to which they refer. They should relate, first, to their numbers; second, to the marriages, births, and deaths that take place amongst them; third, to their diseases, fatal or not fatal; fourth, the duration of their lives; fifth, their average size and strength; sixth, their social and mental condition.

It is further desirable that these statistics should relate both to the past and to the present time, in order that correct comparisons may be drawn between them.

On all these points, so far as the populations of the United Kingdom are concerned, we possess information of a more or less complete character. It will be necessary to indicate its sources under each of the above-named heads.

1. THE CENSUS

The numbers of the population of each locality in Great Britain, and the proportion of the sexes, have been ascertained every ten years since the year 1801, and at each enumeration attempts have been made to obtain some further information tending to throw light upon the social condition of the people.

The first census of 1801, in addition to the numbers of inhabitants of counties, hundreds, and parishes, gave the number of houses and of the *families* by which they were occupied, and also a rough statement of the occupations of the people under the three heads of persons engaged—1, in agriculture; 2, in trade, manufactures, or handicraft; 3, all other persons. It also included a return from parish registers, giving in each hundred or wapentake of England and Wales the number of baptisms and burials at every tenth year from 1700 to 1780, and in each year afterwards, and the number of marriages in every year since 1753.

In 1811 similar information was obtained, with the addition of the number of houses building and the number uninhabited.

In 1821 an attempt was made to ascertain the ages of the population in quinquennial periods up to twenty, and thence at decennial intervals, but the result was unsatisfactory.

In 1831, whilst the classification of 1811, of *families* engaged in three kinds of occupation, was retained, a further subdivision of all males over twenty years of age was made under ten distinct categories.

The inquiry as to ages was not repeated, beyond the distinction of males above and under twenty.

At this census the *area* of each parish and township was given for the first time.

At the census of 1841 a much closer classification of occupations was made, the whole population being distributed according to their various pursuits.

In 1851 still more details were obtained with regard to the various relationships and the civil condition of persons, and the number blind, deaf, or dumb. An inquiry was also made as to the accommodation afforded by the various churches and other places of public worship, and the number of persons generally frequenting them; also as to the existing educational establishments and the actual number of scholars under instruction.

In 1861 there were no returns of religious worship; otherwise no change was made in the form of the census.

In 1871, in addition to the above-mentioned details, a return was made of idiots and lunatics at large.

The census report for 1881 gave the age and sex distribution of each urban and rural sanitary district as constituted in that year, and the civil or conjugal condition of the population, of each of the forty-seven urban sanitary districts, with a population exceeding 50,000 persons.

In addition to the above-mentioned details, the census of 1891 will give the number of inhabited and uninhabited houses, and the number of houses containing less than five rooms—in other words, the amount of cottage property.

DEFECTS IN THE STATISTICS FURNISHED BY THE CENSUS

'It is necessary to bear in mind, with regard to all census statistics, that they are in England, at any rate, simply the tabulated results of facts furnished by householders in their schedules on the enumeration day. The imperfect education of a large proportion of householders in this country necessarily impairs the accuracy of much of the information collected, especially of that relating to occupations. Other causes lead to inaccuracies in the return of the ages and infirmities of the population.

'Occupation statistics furnished in the census schedules can, for instance, never be made to answer the purpose of a thorough industrial census. The difficulties arising from double and indefinite occupations, from the confusion between masters and journeymen, and between those actually engaged in, and those retired from, the various occupations must tend to depreciate the value of this branch of census statistics so long as these statistics are solely dependent upon information supplied in householders' schedules' (Mr. Noel A. Humphreys, in 'Introduction to Dr. Farr's Memorial Volume,' pp. 5 and 6).

As Dr. Rumsey has pointed out ('Fallacies of Statistics,' p. 240, under the head of WATCH AND CLOCK MAKERS, Order X. 7), no distinction is made between the employer and the employed: the occupation includes men moving in the higher circles of society, and enjoying every comfort of life, as well as artisans dragging on a weary existence in crowded and impure tenements in densely peopled town districts.

Again, as to the ages of the population, it is certain that both in the early ages and in advanced life errors are often made by the persons giving information. As Dr. Farr has pointed out, young children are often set down by their parents as a year older than they really are. Thus, when in their first, second, or third year, they were returned as one, two, or three years old; and the true number living *under* each age was misstated accordingly. In the first year of life, especially, the census gives much too low a figure, which of course does not correspond with the balance of births over deaths

in the register. In their old age, also, as old Fuller puts it, people are apt 'to set the clock of their age too fast.' Old people 'often grow ten years in a twelvemonth, and are presently fourscore—yea, within a year or two after, they climb up to one hundred.'

In middle life other sources of error on this point are apt to creep in; for instance, in the tendency of some women to understate their ages. In the census report for 1851 (vol. i. pp. xxiii-iv), Dr. Farr gives an instance of the mode in which this inaccuracy was detected. He there compares the figures of 1841 with those of 1851, and points out that persons of the age of twenty in 1851 must have been ten years old in 1841, and persons of the age of twenty-five in 1851 must have been fifteen in 1841; and as there is a certain loss from death, it is evident that, excluding the effects of emigration, the numbers at the age twenty to twenty-five in 1851 should be less than the numbers living at the ages ten to fifteen in 1841, of whom they (twenty to twenty-five) are the natural survivors. What, then, are the statements which the abstracts of ages express?

1841. The number of girls, age ten to fifteen, was	1,003,119
1851. The number of young women, age twenty to twenty-five, as stated in the returns, was	1,030,456

Now, as the first number would never have swollen in the ten years to the magnitude of the second, we are driven to the hypothesis that, in 1851, the heads of families returned several thousands of ladies of the higher ages at the age twenty to twenty-five.

These instances will suffice to show the difficulty of drawing conclusions in which the ages and the occupations of the population are involved.

Another serious source of error arises from the long interval of ten years that elapses between the enumerations of the people.

In some countries the census is taken more frequently: thus in Denmark and France it is quinquennial, in Prussia and Belgium triennial, and in Austria every year, and in some countries a more elaborate census is taken once in twelve years. In England, in the years intervening between the census, it is necessary to calculate the probable increase or decrease of the populations of the several towns by comparing the numbers of the most recent enumerations. The simplest method is to add to or subtract from the number of population given in the last census one-tenth of the difference between that number and that obtained at the previous census for each year that has elapsed since the last census; and as the census is taken in the first quarter of the year, and the population is required at the end of the second quarter, it is needful to make a further correction of one quarter's increase or decrease.

To those who understand the use of logarithms, and who possess the necessary tables, it is still easier to make the above calculations by the logarithms corresponding to the census numbers.¹

¹ This method is fully described in a leading article in the *Sanitary Record* for March 1879; but it may perhaps be of service to give an actual example, such as that of Norwich, given by Dr. Whitelegge in his work on *Hygiene and Public Health*, pp. 462-3. The population of this town in 1881 census was 87,848; to make the estimate for 1896 (1) find the logarithm for this number = 4.9437072, and (2) deduct from it the logarithm for 80,386, the population for 1871 = 4.9051804; this gives (3) 0.0385268, the logarithm of the decennial increase. (4) Dividing by 10, we get the logarithm of the annual increase = 0.0038527. (5) Again, dividing by 4, we have the logarithm of the quarterly increase = 0.0009632. (6) By adding together the logarithm of the 1881 population (1), and five times the logarithm of annual increase (4), and the quarterly increase (5), we get the

In some towns, such as Manchester, an attempt is made by the medical officer of health to estimate the population in each year by first determining the average number of inhabitants per house by the preceding census, and multiplying this by the number of occupied houses as ascertained from the rates-books: none of these methods are very trustworthy. Dr. Rumsey, in his work on the 'Fallacies of Statistics,' gives the following table, compiled by Mr. Presberg, comparing the *estimated* and the actual populations of sixteen cities and boroughs in the year 1871. The differences, which are usually in excess, are significant.

TABLE I.

Towns	Estimated population in middle of 1871	Enumerated popula- tion by census of 1871	Excess or defect of column 1
Portsmouth	125,464	113,569	+ 11,895
Norwich	81,787	80,886	+ 1,401
Bristol	178,364	182,552	- 9,188
Wolverhampton	74,438	68,291	+ 6,147
Birmingham	378,574	343,787	+ 34,787
Leicester	101,367	95,220	+ 6,147
Nottingham	90,480	86,621	+ 3,859
Liverpool	526,225	493,405	+ 32,820
Manchester	379,140	351,189	+ 27,951
Salford	123,851	125,801	- 950
Bradford	148,080	145,880	+ 2,200
Leeds	266,108	259,212	+ 6,896
Sheffield	255,247	239,946	+ 15,301
Hull	135,195	121,892	+ 13,303
Sunderland	103,087	98,242	+ 4,795
Newcastle	136,293	128,443	+ 7,850

Dr. Louis Parkes ('Some Aspects of Mortality Statistics,' 'Journal of Public Health,' p. 97) gives a table illustrating the same point. In the first three columns are given the census figures of each of eight London parishes in 1861, 1871, and 1881. In the fourth column is given the estimated population at the end of the first quarter of 1881, the calculation assuming the increase or decrease observed between 1861 and 1871 to have continued in the same ratio between 1871 and 1881.

TABLE II.

Parishes	Census returns			Estimation	Differences
	1861	1871	1881	1881	
Paddington .	75,784	96,813	107,218	123,677	+ 16,459
Kensington .	70,108	120,299	163,151	206,422	+ 43,271
Hampstead .	19,106	32,281	45,452	54,541	+ 9,089
St. George's, Hanover Sq. .	37,771	90,028	89,573	92,343	+ 2,770
Westminster .	67,890	66,050	59,926	64,260	+ 4,334
Bethnal Green .	105,101	120,104	126,961	137,249	+ 10,288
Whitechapel .	37,656	34,374	30,709	32,298	+ 1,589
St. George-in- the-East .	48,891	48,152	47,157	47,227	+ 70

Dr. Rumsey gives also an instance, in the case of the borough of Bradford, in which, in 1861, the calculation from the number of inhabited houses and the average number of their inhabitants resulted in an error of 24,000 in excess, in a population of 106,000; and another respecting

logarithm of the 1886 mid-year population = 4.9639339. On referring to the tables, the number corresponding to this logarithm is found to be 92,081, which is the estimated population at the middle of the year 1886.

Liverpool of an excess of 49,000 in a population of 444,000. Such errors as these are, as Dr. Trench, of Liverpool, remarked, 'large enough to falsify all former, and to render doubtful all future conclusions on our rates of mortality when calculated from estimates of population.'

The principle of these calculations really involves two distinct cases. The census being taken every ten years, the problem is, to calculate the population in years when there is no census. Our only resource is to assume that the rate of increase has continued uniformly. Now (1) in the *past*, where we have two censuses, one at each end of the ten-years interval, there is little danger of error. Thus in 1848 we assume that $\frac{7}{10}$ ths of the known total increase or decrease between 1841 and 1851 has taken place. But (2) in the *present*, where we have only *one* such limit at hand, the error may be very great. For example, for 1888 we assume (as before) that $\frac{7}{10}$ ths of the total increase between 1881 and 1891 will have taken place, which is, probably, nearly true. But, as we do not know what this total increase is, we have to call in another assumption—viz. that the rate of increase between 1881 and 1891 will be the same as that between 1871 and 1881. It is this double assumption that allows so much error to arise.

Another method of checking the official population may be derived from the birth-rate of a place. If we assume that this rate remains fairly constant for a series of years we may calculate the population by a simple rule-of-three sum. Dr. Newsholme ('Elements of Vital Statistics,' p. 9) gives the following example:—

In the Wandsworth district the average birth-rate for the ten years 1872-81 was 85·68 per 1,000, and the number of births in 1881 was 7,582; therefore the population in 1881, on the assumption that 85·68 was the birth-rate, was

$$\frac{7582 \times 1000}{85 \cdot 68} = 212500.$$

At the census of 1881 it was actually found to be 210,484, a difference of little over 2,000.

Although a medical officer of health is obliged to accept the estimates of the population, as given by the Registrar-General, if he wishes to be able to appeal to his authority, it would be well for him to check his results by one or other of the methods described above, both for his own satisfaction and for the information of his local board.

The only certain remedy for such errors as have been pointed out would be more frequent enumerations of the number of inhabitants.

The following table gives the results of each successive census since 1801, and shows the enormous increase in the population of England and Wales, and of London, in the present century:—

TABLE III.

Year of enumeration	Population in England and Wales and in London in each census		
	England and Wales	London	Increase in England and Wales
1801	8,892,536	958,863	—
1811	10,164,256	1,188,815	1,271,720
1821	12,000,236	1,878,947	1,857,980
1831	13,896,797	1,654,994	1,896,561
1841	15,914,148	1,948,417	2,017,351
1851	17,927,609	2,362,286	2,013,461
1861	20,066,324	2,808,989	2,138,615
1871	22,712,266	3,254,260	2,656,042
1881	25,968,286	3,814,571	3,256,020

It would be an important addition to the usefulness of the census if, in addition to the enumeration of the population, the proportion of the sick and infirm at each age could be ascertained, and the value of the results would be greatly enhanced by stating the numbers suffering from each kind of infirmity and sickness, not only the numbers of the blind, deaf and dumb, idiotic, and insane—which is partially obtained now—but also the diseases of the population at the time the census is taken.

This course was actually taken in the census for 1851 in Ireland, and the results were afterwards analysed with great skill and with much advantage by Surgeon Wilde.

It is highly desirable that at each decennial period these points should again be inquired into, not only in Ireland, but throughout the United Kingdom.

THEORIES OF POPULATION

The great increase that has taken place in the numbers of the population in the last eighty years opens up the question of the probable eventual overcrowding of the people on the limited area of the British Isles.

The doctrine of Malthus was, that population increases naturally in a geometrical progression, or as 1 . 2 . 4 . 8, while subsistence cannot increase at a faster ratio, in the same time, than is expressed by the arithmetical progression 1 . 2 . 3 . 4, . . . and the checks which repress the superior power of population, and keep its number on a level with the means of subsistence, are all resolvable into moral restraint, vice, and misery.

It is hardly likely that the instinct of humanity will be so dead in any man that he would propose to encourage the two latter 'checks' to population, but the dread of over-population may possibly render men lax in preventing a mortality that they think may perhaps be eventually beneficial to the country.

Dr. Farr, in successive letters to the Registrar-General and in census reports, was never tired of combating the fallacies involved in the Malthusian theory. It may be useful to medical officers of health in dealing with their respective boards to state briefly the heads of his argument:—

1. He shows that if the constituent roll of a nation is reduced too low 'it cannot struggle with success against other forms of life in nature; it cannot hold its own in the face of other powers; it can undertake no great concerted operations; the quantity of life is lessened on the earth, and the chance is diminished of the rise of men of genius, to whom the world owes progress in the sciences, discoveries in the useful arts, and triumphs in fine arts and literature. Ill effects of too many people willing to work can for centuries only be felt when they are blindly crowded in particular spots; when their labour is not organised; when their acquisitions are insecure; when their dwellings are dens; and when the supply of subsistence is not rendered continuous and, within due limits, equal by storage, by commerce, by skilful distribution, and by wise laws.'

2. The several propositions of Malthus were neither proved nor supported by argument, and they are contrary to experience; for a large part of subsistence is the product of industry, and increases in proportion to the numbers of civilised men. In Great Britain the means of subsistence have increased faster than the numbers of the people; for, while the population has doubled, the value of capital has quadrupled. Her produce, which in the present state of commerce is always convertible into the means of subsistence, has probably not increased at a lower rate; no one can pretend that the ratio has been arithmetical.

Again, the increase of the population is subject to no inexorable law, for it is controlled by many influences, such as the prudence of the citizens. Bad harvests and bad trade always diminish the number of marriages; education and the cultivation of the intellect diminish the rate of increase, and by encouraging higher ideals postpone the date of marriage. If necessary, legal enactments may intervene, as in Switzerland; but for the present there is no need to discourage the expansion of the population of England: her colonies are in need of more inhabitants, her industrial enterprises of more workpeople. 'Every master knows that good men, and every man that good masters, *are scarce*.'

'The theory is as misleading in practice as it is defective in statement. It assumes that the restraint of population is the corner-stone of policy. Had this principle been accepted by the people, the population of the kingdom, instead of amounting to 82,000,000, would have remained, as it was at the beginning of the century, 16,000,000. England, in the presence of the great Continental States, would have been now a second-rate power; her dependencies must have been lost; her colonies have remained unpeopled; her industry crippled for want of hands; her commerce limited for want of ships. And logically it leads to the policy of depopulation; for if increase causes misery, decrease, by parity of reasoning, causes happiness, this principle of population being the fewer the happier. It is a policy that diminishes the numbers of the wise and the good, but has no effect on the masses.'

2. STATISTICS OF MORTALITY AND REPRODUCTION

The next great group of vital statistics contains those drawn from the records of marriages, births, and deaths, and it is only in recent years that these have been in any sense of the term complete.

'In the earliest dawn of the nation, the English inquired into the causes of death, with a view to discovery and prevention. The protection of life was a fundamental principle of their laws. It was as much an object of their political organisation as national defence or war. And the dead body, it was held, called for the coroner's inquest whenever death was sudden, or violent, or in prison.' But this, it will be seen, was only a very partial and limited inquiry.

(a) BILLS OF MORTALITY

Towards the end of Queen Elizabeth's reign (1592) the London bills of mortality were commenced, and similar records were kept in all the parishes of the kingdom. These were abstracts of all the burials, baptisms, and marriages therein taken place. Persons were also appointed to view, before they were suffered to be buried, the bodies of all who died, and the probable disease of which each individual died was recorded and returned every week.

Captain Grant¹ thus describes this remarkable series of tables:—

'When anyone dies, then, either by tolling or ringing of a bell, or by bespeaking of a grave of the sexton, the same is known to the *searchers*, corresponding with the said *sexton*. The *searchers* hereupon (who are ancient matrons sworn to their office) repair to the place where the dead corpse lies, and by view of the same, and by other inquiries, they examine by what *disease* or *casualty* the corpse died. Hereupon they make their report to

¹ Quoted by Dr. Farr from *Natural and Political Observations upon the London Bills of Mortality*, 5th edition, 1676.

the *parish clerk*, and he, every Tuesday night, carries in an account of all the *burials* and christenings happening that week to the clerk of the hall. On Wednesday the general account is made up and printed, and on Thursday published and dispersed to the several families who will pay four shillings per annum for it.

The defects of these returns were that frequently baptisms did not take place; certain sects also only baptized adults. Dissenters' burials were not included; and the clerks of many parishes made no returns, or only made them irregularly. Even when complete, the bills gave no information about the population of the towns and counties of the whole kingdom.

Notwithstanding these shortcomings, certain of the returns were used for the purpose of compiling life tables for the use of insurance companies.

The most remarkable of these tables are those of Northampton and Carlisle. The former was compiled by Dr. Price, from the All Souls' bills of mortality, between the years 1785 and 1780. It comprised 4,689 deaths, but it takes no note of possible immigration into the town, and, for the cause above mentioned, the children of Baptists were not counted. Males and females are not distinguished.

Dr. Farr afterwards constructed a true life-table for Northampton, and shows that the result of the above errors was that the after lifetime of a person aged twenty was only reckoned at thirty-three years, whilst by the true table it would be forty years.

In calculating the value of an annuity of 1,000*l.*, at the age of thirty the price to be paid by the true table would be 19,081*l.*; by Dr. Price's table it would be only 16,911*l.*, a loss to the insurance company of 2,170*l.*

The Carlisle table was constructed by Mr. Milne from two enumerations of the population of the parishes of St. Mary and St. Cuthbert, Carlisle; the first in January 1780, when the inhabitants were 7,677, and the second in December 1787, when the inhabitants amounted to 8,677, namely, 3,864 males and 4,813 females. The deaths in the two parishes were 1,840—males 881, females 959—in the *nine* years 1779–1787.

This table was more correct than the other, but it was too limited in the numbers observed to be trustworthy, and it took no account of the large number of servants present amongst the population, most of whom emigrated from the town before death.

(b) THE REGISTRAR-GENERAL'S RETURNS

After the passing of the Registration Act in 1887, regular returns were made throughout the country of marriages, births, and deaths.

They are now collected by more than two thousand registrars and superintendent registrars, and after revision by the officials in the Registrar-General's office they are published in his annual report, quarterly and weekly reports of certain of the particulars of the returns being also published.

The returns of marriages contain, in addition to the names of the husband and wife, their ages and residence; and note is taken of the cases in which the necessary signature is made by 'mark,' a sort of test of educational capacity.

The returns of births give the sex, the date and place of birth, the number of births, their legitimacy or otherwise, and the residence of the parents.

The registers of deaths give the name and residence of the deceased, the date and place of death, and the primary or secondary causes of death, with occasionally the duration of the fatal disease.

From these data a multitude of tables are drawn up in the Registrar-

General's office, the details and comparisons given varying somewhat in each report.¹

Some statistics are also given relating to foreign countries for purposes of comparison.

NOMENCLATURE AND CLASSIFICATION OF DISEASES

The names given to the several causes of death, in the Registrar-General's tables, are as far as possible those adopted by the Royal College of Physicians of London, and revised by them in 1885; but in the grouping of these diseases they are tabulated as much as is practicable in reference to their probable source outside the body; thus there are eight chief divisions, each with sub-sections.

I. *Febrile or zymotic diseases*, including, 1,—miasmatic diseases, such as small-pox, chicken-pox, measles, rose-rash, scarlet fever, typhus, relapsing fever, influenza, whooping cough, mumps, diphtheria, cerebro-spinal fever, simple or ill-defined fever, enteric fever.

2. Diarrhoeal diseases—cholera, diarrhoea, dysentery.

3. Malarial diseases—remittent fever, ague.

4. Zoogenous diseases—hydrophobia, glanders, splenic fever, cow-pox, and other effects of vaccination.

5. Venereal diseases—syphilis, gonorrhoea, stricture of urethra.

6. Septic diseases—phagedæna, erysipelas, pyæmia, septicæmia, puerperal fever.

II. *Parasitic diseases*—thrush, hydatids, vegetable and animal parasites.

III. *Dietetic diseases*—starvation, want of breast milk, scurvy, intemperance, chronic delirium tremens.

IV. *Constitutional diseases*—rheumatic fever, rheumatism of heart, gout, rickets, cancer, tabes mesenterica, tubercular meningitis, phthisis, scrofula, other forms of tubercular disease, purpura, hæmorrhagic diathesis, anæmia, chlorosis, leucocythæmia, diabetes, or other constitutional diseases.

V. *Developmental diseases*—premature birth, atelectasis, cyanosis, spina bifida, imperforate anus, cleft palate, &c., old age.

VI. *Local diseases* of nervous system, of organs of special sense, circulating, respiratory, digestive, lymphatic, and urinary systems, of the organs of generation, parturition, organs of locomotion, integumentary system.

VII. *Violence*—accident or negligence, homicide, suicide, execution.

VIII. *Ill-defined and not specified causes*—dropsy, debility, atrophy, inanition, mortification, tumour, abscess, hæmorrhage, sudden death, causes unascertained, other ill-defined and not specified causes.

Whatever may be the shortcomings of this great series of statistics—and we shall presently see that they are many—few can doubt that they have proved of enormous value to the nation. They have constituted a kind of barometer of the various morbid influences weighing upon the people; they have aroused the nation to efforts to obviate or overcome those influences. They have produced nearly all the sanitary legislation we possess; most of the sanitary Acts bear the signature of her Majesty Queen Victoria. They have served as a mine of material from which statisticians have drawn valuable conclusions respecting the laws of population, the possibilities of extending human life, the causes of mortality, and the causes of disease.

'How the people of England live is one of the most important questions

¹ A statement of the particulars published from year to year in the Registrar-General's annual reports is given in the 38th Report.

that can be considered; how, and of what causes, and at what ages, they die is scarcely of less account; for it is the complement of the primary question, teaching men how to live a longer, healthier, and happier life.'¹

DEFECTS OF THE REGISTRAR-GENERAL'S STATISTICS

1. Until the year 1874 the certificate of the cause of death was not compulsory.

2. Still-births are not compulsorily registered.

3. A proportion of the deaths are not certified by legally qualified practitioners, and this proportion varies in different localities.

Dr. Farr calculated that in 17 per cent. of the total number of deaths registered in England and Wales no clue is given as to the cause of death. At one time in South Wales the registration books were in a state of hopeless confusion. At Llanbedr, out of 500 notices of death, 101 were certified, 899 were not; and in St. David's, out of the same number, only fifteen were certified.

4. Although in some few instances attempts are made to indicate the time of the commencement of the fatal illness, no idea is given in the death-certificate of the place where the disease originated.

5. The returns are often misleading on account of inaccuracy in the diagnosis of disease, on account of imperfections in the nomenclature used, either now or in former years; and lastly from misrepresentations as to the true cause of death. Alcoholism and syphilis are thus frequently omitted when they have caused death. To quote again from Dr. Farr, 'thousands of deaths occur without any scientific inquiry into the causes of death; and in thousands of other cases medical science seeks in vain to unravel the mystery which enshrouds the extinction of life.'²

As Mr. Humphreys remarks ('Vital Statistics,' memorial volume, p. 115), 'causes of death in the death register are necessarily little more than the more or less trustworthy guesses of a large body of more or less skilled observers, except in the small proportion of cases in which these guesses are corroborated or modified by *post-mortem* examinations. Statistics of cases of death should therefore be compiled with caution, and without any attempt at over-elaboration of detail. Still greater caution should be used in drawing inferences and deductions from a comparison of the results for a series of years; and changes of nomenclature and of classification add materially to the difficulties in the way of useful comparison of such statistics for different periods of years.'

(c) LOWE'S RETURNS

A Parliamentary paper was moved for in 1878 by Mr. Lowe, and hence is commonly called 'Lowe's Returns.' These returns constitute another important series of mortality tables. Their full title is 'Average Annual Proportion of Deaths from Specified Causes, at Specified Ages, in England generally, and in each Registration Division and Registration District, during the Decennial Period 1861-70.'

It gives the populations of these districts, and the deaths at all ages from all causes—from fever, diarrhoea, dysentery, cholera, scarlatina and diphtheria per 100,000 living. Then the deaths from all causes per 100,000 living at less than one year of age. The deaths are then given under five years of age, per 100,000 living, from all causes, and from the following

¹ Supplement to 35th Annual Report, p. 8.

² 27th Annual Report, p. 175.

diseases: diarrhoea, dysentery, and cholera, in one column; diseases of the respiratory organs, excluding phthisis; diseases of the brain, including hydrocephalus; small-pox, scarlatina, measles, whooping cough. Then, at ages between fifteen and twenty-five, similar returns of deaths of males and females separately, from phthisis pulmonalis and other diseases of the respiratory organs; and lastly, at ages between thirty-five and fifty-five, the deaths of males and females, of diseases of the brain.

By calculating each of these rates upon 100,000 of the population living at each age, the errors due to variety in the distribution of ages are eliminated.

3. STATISTICS OF SICKNESS

Constitute a very important addition to the material at the disposal of a medical officer of health.

The value of such returns has often been pointed out. Thus the metropolitan medical officers of health in 1858 state from their experience that 'the sickness table offers frequently a better test of the condition of the people in reference to their helplessness than even a table of deaths.' It is the amount and duration of sickness rather than the mortality that tell on the prosperity of a community, and the sanitary condition of a State cannot be adequately predicated from a simple enumeration of deaths.

Such returns at short intervals also show the influence of varying conditions of climate and season, of prosperity or distress, of the trades and manufactures, or of any other circumstances peculiar to a district; they direct the early attention of local authorities to the precise spot where each epidemic breaks out, and measure its progress. They are most valuable to the inhabitants, giving exact and timely intelligence of the presence of disease, and enable them promptly to deal with it.

The first successful effort to obtain a representative registration of disease was made in 1858 by the Manchester and Salford Sanitary Association, from weekly returns received from all the public institutions and poor-law medical practitioners in the town, thirty-nine in number. It was carried on with remarkable regularity for twenty years. Its example was followed at Preston, Newcastle-on-Tyne, and Birmingham, and it encouraged the British Medical Association in 1862 to agitate for a national registration of sickness.

Although this effort was not entirely successful, the value of returns of infectious disease only was recognised by many towns, and at the present time more than fifty towns have secured compulsory powers for obtaining from householders and medical men the early notification of cases of certain forms of infectious sickness.

Defects.—Even for the practical purpose of preventing epidemics, these returns are insufficient. Isolation and disinfection are not the only means by which these diseases may be met. We ought not only to seek to suppress them as they arise, but should try to prevent their origin.

In order to carry out this aim, it is necessary thoroughly to study their natural history, to know their habitats, the places most favourable to their germination, the conditions under which they can most readily assume the epidemic form; the relations to them of surrounding circumstances, of the weather, or of season; the condition of the soil, and even the kind and quality of provisions. The concurrence of other complaints should also be noted, and especially the predisposition to any specific disease occasioned by previous affections of the parts most attacked by the epidemic virus; the throat, for instance, in scarlet fever; the air-passages in measles and whooping cough, the bowels in enteric fever and cholera. We also need

more information as to the conditions that favour the recrudescence of epidemics—i.e. their rise again after a fall. All these are points that need investigation, if epidemics are to be prevented. Moreover, in course of time, it ought to be possible to predict the coming of an epidemic with at least as much certainty as we now receive tidings of a coming cyclonic storm, and its probable track should be marked out before its arrival.

But for all these purposes, it is essential that the returns obtained by means of the notification of disease should be made more serviceable than they are at present; not only should they be published *en masse*, as they are now, but they should be broken up and detailed, so as to show the incidence of the disease at different periods, in each district of the town. They ought also to be made comparable, so that it should be possible to compare one season with another season, and one district with another district.

This is not the case at present, for the proportion of the cases reported to the total number of the population is not known. It would not be difficult to ascertain this. In the weekly returns of the sanitary association a device was adopted, at the writer's suggestion, by which a very close approximation to this proportion could be obtained. This was to obtain, simultaneously with the disease returns, a record of the mortality occurring amongst the cases reported: this was then compared with the total mortality, and a very fair guess could be made as to the total number of cases occurring within the district.

The mortality returns were strictly confidential, and thus no invidious remarks could be based upon them. This device, if carried out in all the towns where notification of disease is obtained, would enable us to compare the records of one place with those of another, and would permit us to follow several of the lines of inquiry which we have suggested as desirable. But it is unfortunate that the registration of sickness cannot be carried further than it is at present. Thus it would be very important from time to time to ascertain the prevalence of puerperal and rheumatic fever, of pneumonia and diarrhoea, of cancer, syphilis, and goitre. Again, there are many diseases that, being neither epidemic nor often fatal, are commonly regarded without concern. Thus, as the Royal Sanitary Commissioners remark, 'ague is very rarely fatal; it counts for little on the register of deaths; yet ague, in all its various forms and its long abiding, is a great burden on the wealth of the nation, a burden which good sanitary arrangements could remove.' And so of phthisis, scrofula, diarrhoea, and many other disabling maladies. A more complete registration of sickness would show whether, and in what degree, legislation is needed for the prevention of diseases dependent on occupations and social habits. 'The effects of social vices and vicious habits of life, and whether any of them are sources of such sickness, poverty, and decay, as to justify, on public grounds, a considerable expenditure of money and executive force, cannot be known without registration.' Dr. Farr's remarks upon this subject in the supplement to the Registrar-General's 85th Annual Report are an important incentive to an effort to improve the statistics of non-fatal sickness. He says: 'The reports of the existing medical officers are of great practical value, and will become more valuable every day. What is wanted is a staff officer in every county or great city, with clerks, to enable him to analyse and publish the results of weekly returns of sickness, to be procured from every district, distinguishing, as the army returns do, the new cases, the recoveries, the deaths reported weekly, and the cases remaining in the several hospitals, dispensaries, and workhouses. These, compiled on a uniform plan, when consolidated in the metropolis, would be of national concern. It has been suggested that the returns of sickness

should, to save time, be sent to London, and there analysed on a uniform system, as the causes of death are. That, with the present postal arrangements, is quite practicable. The thing to aim at ultimately is a return of the cases of sickness in the civil population as complete as is now procured from the army in England. It will be an invaluable contribution to therapeutics as well as to hygiene; for it will enable the therapeutists to determine the duration and the fatality of all forms of disease, under the several existing systems of treatment, in the various sanitary and social conditions of the people. Illusion will be dispelled, quackery as completely as astrology suppressed, a science of therapeutics created, suffering diminished, life shielded from many dangers. The national returns of cases and of causes of death will be an arsenal which the genius of English healers cannot fail to turn to account.'

4. STATISTICS OF MEASUREMENTS

The last group of vital statistics contains data concerning the physical condition of the so-called healthy portions of the population. Again, we must quote from Dr. Farr.

'The stature, the weight, the strength, the working power, and the intelligence of the people are indications of health which should be explored in groups of the population at each age.' The first four of these conditions all admit of accurate measurement, and although it would not be possible to carry out this measure for a whole population, observations might be made from time to time upon certain numbers of the people at all ages.

It is difficult to over-estimate the value to a health authority of such information as is here demanded by Dr. Farr. By means of the returns of disease and death we are informed as to the actual losses sustained by the community from certain causes; but these figures tell us very little of the ultimate effect upon the physical power of the population. We cannot measure this influence by the simple record of the numbers directly affected. We have to learn their effects upon the following generation, and this can only be ascertained by such careful direct measurements as are here recommended.

Hitherto, the only measurements we possess have for the most part been made by private individuals; thus Dr. Ferguson, of Bolton, in his capacity of certifying surgeon, made a number of measurements of the young persons presenting themselves before him, and he came to some very adverse conclusions respecting the children of the working classes.

Mr. Charles Roberts made still more extended observations, and published them in his paper, read before the Statistical Society, on 'The Physical Requirements of Factory Children.' Dr. Beddoe, of Clifton, obtained measurements of groups of the population, in the different counties of England and Scotland and other parts, in relation to height, weight, girth, breadth of shoulders, and so on.

Most of these statistics are embodied in the report of the Anthropometric Committee of the British Association, and they are well worthy of study. They show distinctly the adverse influence of town life as compared with country life, and 'the check which growth receives as we descend lower and lower in the social scale.' A difference of 5 in. is found between 'the average statures of the best and worst nurtured classes of children of corresponding ages, and 8½ in. in adults.'

'The influence of town life and town occupations on the physique of the population, in districts in which the race differs little, and the climatic

conditions are the same, is seen by comparing the agricultural population of Ayrshire with that of Glasgow and Edinburgh, where the average difference in stature amounts to 4.15 in., and in weight to 36.1 lb. in favour of the country folk. A similar though not so great a difference exists in Yorkshire, where the fishermen of Flamborough exceed the artisans of Sheffield in stature by 2.91 in., and in weight by 24.3 lb.'

These observations are, however, insufficient for the purposes of public health. They refer only to one period of time, and hence no conclusions can be drawn from them as to the progress of the nation in health and strength. What is required is that there should be accurate measurements made every ten years of representative groups of the population of all classes and at certain ages.

GRAPHIC REPRESENTATIONS OF STATISTICS

Graphic representations of figures are often of great assistance to the student of vital statistics, and may be made in many ways. Thus by various degrees of shading upon maps, as in Boudin's '*Statistique Médicale*,' the extent of prevalence of various diseases in different parts of the country may be shown, or, again, colour may be employed in the same way as in Lombard's maps; and the varying intensity of the colour may be made to show the varying prevalence of the disease indicated by each colour. In Dr. Haviland's excellent maps, showing the distribution of heart-disease, phthisis, and cancer in England and Wales, two chief colours, blue and red, are employed, and each colour is given in three shades—the intensity of the blue colour showing the degree of prevalence, and the intensity of the red colour displaying the degree of immunity, enjoyed by different registration districts throughout the country.

Not unfrequently, also, maps are employed by medical officers of health, and the exact sites of the occurrence of certain diseases are indicated by crosses or other signs, printed in colour on the spots where these diseases originated or occurred.

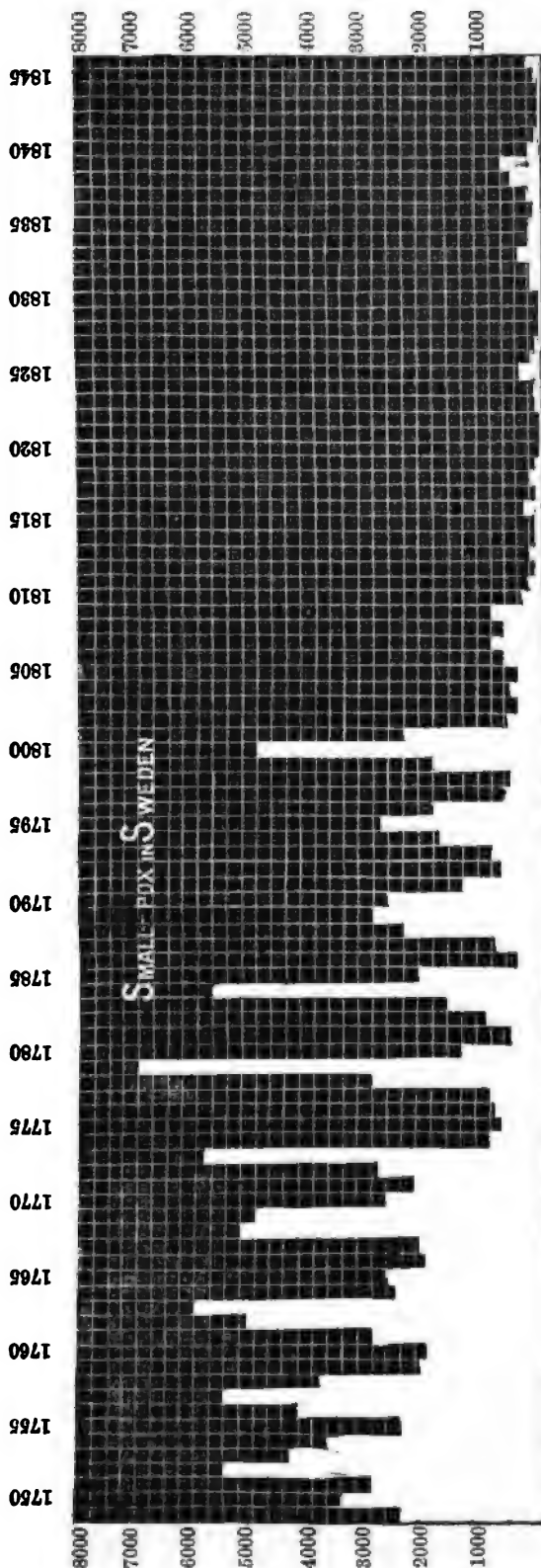
Masses of colour are sometimes used to show the relative proportion of different diseases taking place within certain definite periods of time, as in Dr. Farr's diagrams of the extent of lung disease and fever in the Crimean army. Lines of varying length are also made to show the relative proportion of disease and death, as in Dr. de Chaumont's work on State medicine.

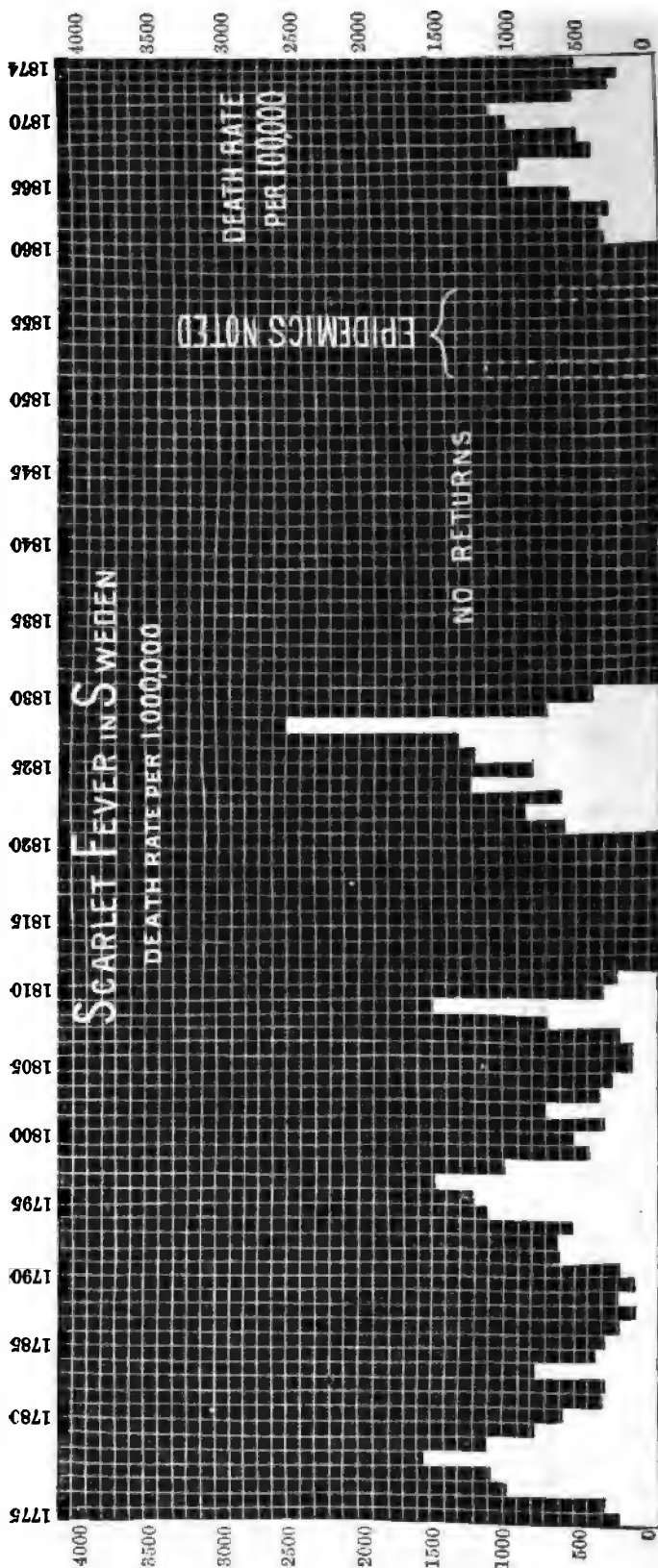
But perhaps the most useful of all devices for both showing the extent of sickness and the periods of time within which it occurred are the methods of curves or columns of disease.

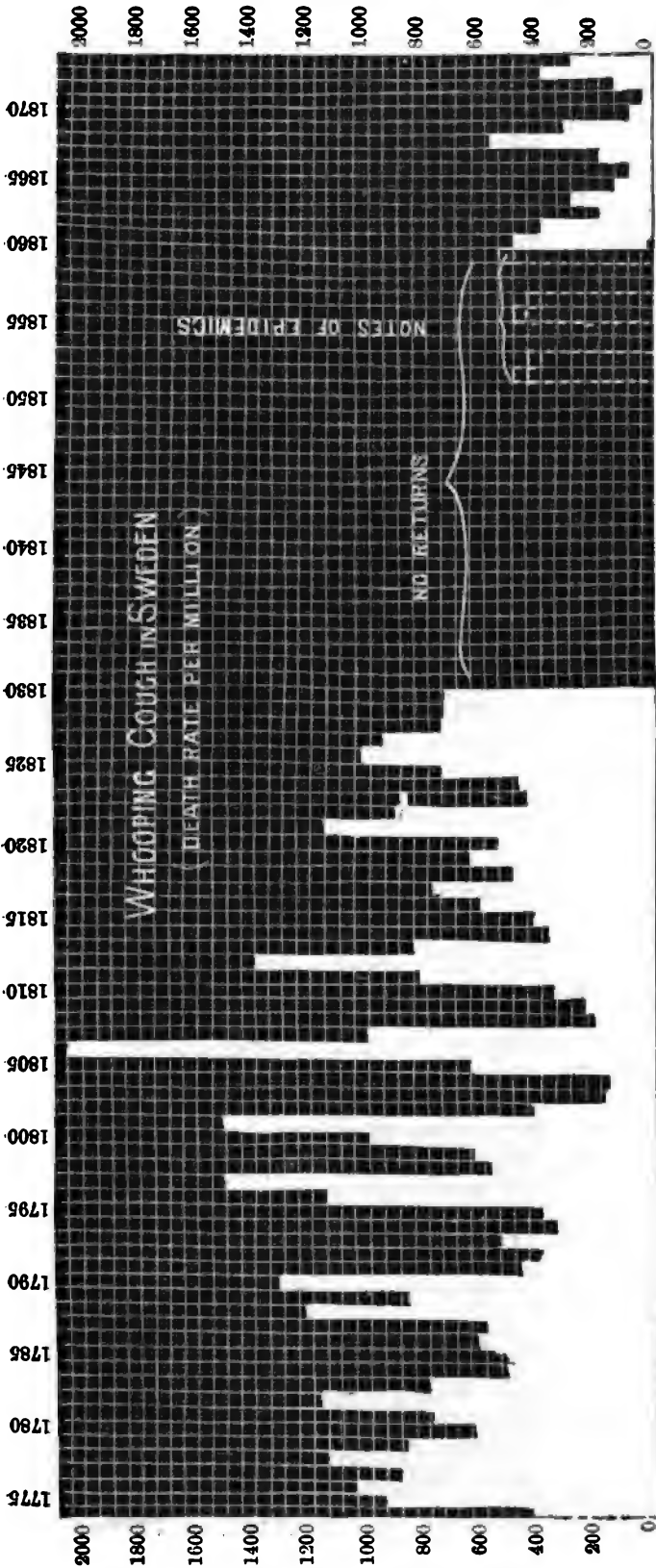
Periods of time—of days, weeks, months, or years—are denoted by the divisions of a horizontal line, and from these divisions vertical lines are drawn, their height above the base line marking the extent to which the disease prevailed during the period expressed by the divisions in the base line. Usually these divisions are of equal size, and the diagram is ruled in squares, so that the extent of the disease in units, tens, hundreds, or thousands can be indicated on the divisions at each side of the diagram.

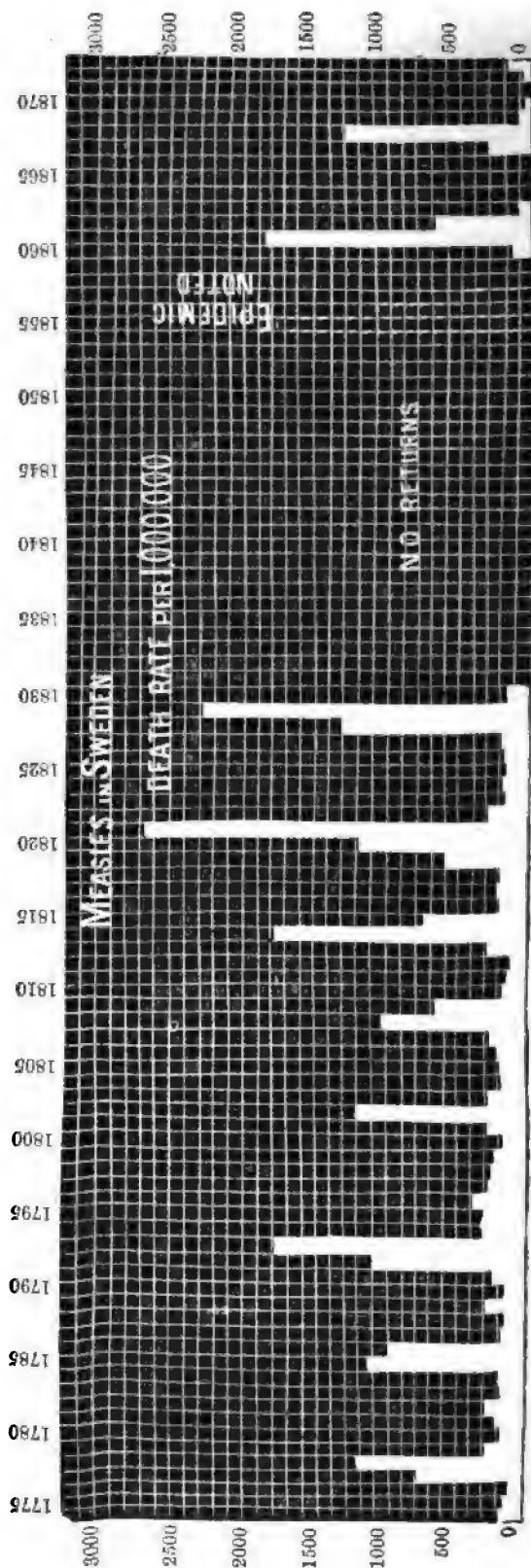
The accompanying figures, showing the rise and fall of various epidemics in Sweden for 100 years, are given as an illustration of this method, and also because of their intrinsic value. They are drawn from figures privately supplied to the writer by Dr. Berg, of Stockholm, and they serve to show the remarkable cycles in which small-pox, scarlet fever, measles, and whooping cough prevailed in that country.

Curves of disease are formed by drawing a continuous line through points









at the several heights on the vertical lines indicating the extent of the disease at certain fixed periods of time.

By this means not only may a direct comparison be at once made between the causes of different diseases, but they may be compared with other curves denoting atmospheric phenomena—heat and cold, humidity or dryness of air, barometric oscillations, and so on. The price of wheat, and other signs of prosperity or distress, may be in like manner brought into contrast with the curves of disease. Mortality returns at different ages may be represented in a somewhat similar manner. There are other graphic devices occasionally in use, as in Dr. Farr's diagrams to represent the degree of proximity of populations in relation to their rates of mortality (40th Report of the Registrar-General, p. 287).

These examples will, however, suffice to show the uses that may be made of graphic methods.

SOURCES OF FALLACY IN STATISTICS

We have now enumerated the several stores of statistical material at the command of a medical officer of health. But before proceeding to describe the various modes in which these figures may be employed, it will be desirable to indicate the limits within which they may be used without danger of error. Unless this is done, as Carlyle has said, the tables will be 'like cobwebs, or like the sieve of the Danaides, beautifully reticulated, orderly to look upon, but which will hold no conclusion.'

In an ideal series of statistics, (1) the facts must all be correctly observed. In some cases this condition is easily adhered to. It is thus simple enough to ascertain correctly the numbers of a population, and the proportion of births and deaths and marriages occurring amongst its members; hence the rates founded upon these figures during the year of census are pretty sure to be exact. But if more than this is attempted, if such details as the ages of the population, and the diseases from which they die, are required, then, as we have already seen, serious liability to error arises.

(2). In order that statistics may be of service for the purposes of the public health, it is important that they should admit of comparison, both with similar figures obtained at previous periods and with those of other places. But if correct deductions are to be drawn from these comparisons, the figures to be compared must be *homogeneous*—i.e. they must be of the same kind or order; and this condition must be fulfilled both in the facts that are grouped together, and in the standards against which they are placed. Take first the facts collected together.

a. There are comparatively few simple facts to be observed in vital statistics; for the most part they are complicated by an endless variety of differences—differences of degree and intensity—or by the influence of surrounding circumstances. In our enumeration of diseases, for instance, we may often be obliged to mass together deaths, or diseases of various organs, or all the deaths from particular diseases, such as cancer or consumption, without reference to their extent or duration, and much may be learnt even by such a classification as this, if care is taken to see that the statistics are comparable from one time to another, or that the surrounding circumstances at one place are not such as to prevent them from being fairly placed against those at another place. A comparison of hospital statistics is proverbially untrustworthy, on account of the extreme variation of the conditions that surround them.

b. Again, if such grouping of facts is to be of any real service in com-

paring the sanitary condition of towns, it is important that a number of facts, having an entirely different *causation*, should not be included together under one heading.

Thus it is not uncommon to see all the affections of the respiratory organs, with the single exception of phthisis, tabulated together, and this heading includes not merely catarrhal affections that might be supposed to have a common origin in atmospheric influences, but such diseases as acute pneumonia, pleurisy, or asthma, which frequently arise from quite other causes; and if it were not for the fact that catarrhal disorders are so common as to overwhelm the numbers of the other lung diseases, very little would be gained by this tabulation.

Again, if the rate of mortality from zymotic disease—the zymotic rate as it is called—is recorded without discrimination between the forms of zymotic disease—such as small-pox, scarlet fever, enteric fever, &c.—very little could be discovered as to the sanitary progress of a community in respect to vaccination, improved drainage and water-supply, and so on.

The facts enumerated must therefore be grouped together as much as possible in reference to some common cause.

c. Another bar to comparison is to be found in the varying virulence of certain diseases. It is thus unsafe to assume that a lessening of the death-rate from any form of epidemic disease is to be ascribed to sanitary measures, or to the particular kind of treatment that has been adopted.

Epidemics have a tendency to run a certain course, and then to subside for a certain number of years, and it is well known that they are much more virulent and fatal, in proportion to the number attacked, at their commencement than near their decline. The data, therefore, are not homogeneous, and do not admit of comparison from one period to another.

d. Similar remarks apply to the standard against which the facts are arranged so as to bring them into relation with those of some other place. Thus we have already seen that even the estimated population is an unsafe standard from which to derive a birth-rate or a death-rate; but in order that these latter statistics should be strictly comparable, even in the census year, it is needful that the populations of the places compared should be similar in at least two respects: first, as regards the proportion in them of persons at different ages; secondly, as regards their occupations and their migratory habits. These points will have to be presently more fully considered.

(8). The facts must be localised in regard to both the place and time of their occurrence.

It would thus be absurd to construct a phthisis-rate for Madeira or Mentone, from the deaths of persons occurring at those places from this disease; and the presence of the Brompton Hospital for Consumption presents a similar objection to the framing of a phthisis-rate for West London.

In some towns also the results of the coroner's inquests are returned to the Registrar-General fortnightly, or even monthly, and thus unduly affect the rates in his weekly return.

(4). The facts must be sufficiently numerous to give correct averages, and they must extend over sufficient lengths of time.

The simplest mode of ascertaining whether they are so or not is one given by Dr. Guy, and he gives an example of its application in an inquiry into the ages at death of the male members of the English aristocracy. He explains it as follows: 'The several facts were first arranged in groups of twenty-five each; two successive groups were then formed into groups of fifty; the groups of fifty in like manner into groups of a hundred, and so on

until the last totals in the table were obtained. The greatest and least averages from each group of facts were then selected and thrown into the following table :—

Number of facts	Average age at death		
	Maximum	Minimum	Range
25	69.40	50.64	18.76
50	66.44	55.20	11.24
100	63.70	56.85	6.85
200	62.98	57.61	4.77
400	61.10	58.24	2.86
800	60.84	59.67	1.17
1,600	60.25		

This example shows that, the smaller the total number of facts, the larger will be the relative percentage of discrepancy displayed by them, and hence arises the practical lesson to obtain all the homogeneous statistics possible, and the margin of error will diminish with the number collected.

The mathematical rule is that the error diminishes as the square root of the number of observations ; thus, if 1,000 observations have been collected, then, in order to obtain a result twice as accurate, four times the number of facts must be brought together.

Hence, as Dr. Guy remarks, ‘averages drawn from small numbers of facts stand in need of a confirmation which averages drawn from larger numbers of facts do not require, and in using the former we are bound to speak with a reserve proportioned to the scantiness of our materials.’

Extreme values ought also to be taken into account in the study of statistics. ‘As averages founded upon large numbers are numerical expressions of true *probabilities*, so extreme values are expressions in the same precise language of *possibilities* ; one use of them is to confirm and strengthen the conclusions drawn from averages, and another to test numerical theories.’

‘The same general principle which applies to averages applies also to the extremes, namely, that the value of the extremes increases with the number of observations from which they are selected.’

‘To obtain a correct mean, or a probable extreme, we must multiply our facts’ (article on Statistics, by Dr. Guy, ‘Encyclopædia of Anatomy and Physiology,’ 1852).

Another mode of testing the results of a statistical inquiry is by means of the ‘law of error,’ as it is called by mathematicians ; but it is seldom or never necessary to use this method in vital statistics, for it simply proves that the mean result is the most probable one ; and when there is only a single variable this mean is found by the familiar arithmetical process.

Professor Jevons gives the following rules for finding the probable error of a mean result :—

1. Draw the mean of all the observed results.
 2. Find the excess or defect, that is, the error of each result from the mean.
 3. Square each of these reputed errors.
 4. Add together all these squares of the errors.
 5. Take the square root of this sum.
 6. Divide the square root by the number of results.
 7. Multiply the quotient by 0.67449 (or approximately by 0.674, or even 0.67), a natural constant number derived from the law of error in a manner which is described in mathematical works upon the subject.
- Suppose we take four weekly death-rates of a town—say 32, 29, 20, 18.

We want to know the probable error of the mean, namely 25. Now the differences between this mean and the above numbers, paying no regard to direction, are 7, 4, 5, 7; their squares are 49, 16, 25, 49, and the sum of the squares is 139. Taking the nearest square root of this sum, 12, and dividing by 4, the number of observations, we have 3, which has only to be multiplied by .67 to yield us $2.01 =$ probable error. Thus the probability is one half, or the odds are equal, that the true death-rate lies between 23 and 27. As a matter of fact, it is 26.8.¹

These few hints as to the precautions to be observed in the use of statistics by no means exhaust the subject. The student of such figures has to be constantly on his guard against sources of fallacy such as must fatally vitiate his conclusions, and yet, on the other hand, he must not necessarily reject all materials that are not beyond suspicion. If a careful estimate be made of the possibility, or even the probability, of error, even faulty statistics may be used. As Mr. Humphreys remarks ('Vital Statistics,' p. 116), 'much valuable work may be done with defective materials if their defects be kept steadily in view, both in the calculation of results and in making deductions therefrom. The materials with which Dr. Farr had to work nearly fifty years ago were defective enough to have discouraged a less sanguine and indefatigable statistician; but while he was able from time to time to improve the quality of his materials few will venture to deny the value of the results he obtained with such defective material.' 'In vital statistics all students must learn to use imperfect materials, and yet to guard, as far as possible, against fallacious results.'

METHODS OF USING VITAL STATISTICS

1. *Life Tables*.—In considering the various modes in which the vital statistics which have been enumerated may be utilised, it will be convenient to take life tables first, as they display the incidence of mortality at each year of life, and thus serve to show the influence of varying proportions of persons at each age upon the death-toll paid by different communities.

Life tables are constructed from the registers of births and deaths by means of somewhat abstruse and difficult mathematical calculations, into which it is not now necessary to enter. The theory upon which they are founded is, however, a very simple one, and is stated by Dr. Farr as follows:—

'If 100,000 persons born at the same moment were followed through life, the numbers that died in each year of life noted, and the sum of their ages divided by 100,000, the average ages which they lived would be obtained.' In other words, the number of the survivors at each age would be ascertained, and then the mean duration of their lives. 'Say that it was found to be forty-one years at birth, if another 100,000 were taken in worse circumstances, dealt with in the same manner, and their average duration of life were found to be twenty-six years, you might infer that life is shortened fifteen years in the latter circumstances.'²

Dr. Farr considers that life tables supply the only scientific, thoroughly trustworthy, method for ascertaining the true import of rates of mortality in increasing or decreasing populations, whether such increase or decrease be due to the difference between birth-rates and death-rates, or to the effect of migration (see the 5th Annual Report of the Registrar-General, pp. 362-365).

¹ In actual working many more observations than four would be necessary to make such a calculation of any 'probable error'; but the above example may serve to show the untrustworthiness of weekly death-rates as a test of sanitary condition.

² Another fuller account of the formation of a life table is given by Dr. Farr in the *Transactions of the Royal Society*, 1859, pp. 838-41. Also in *Vital Statistics*, pp. 491-93.

He gives a short method of constructing life tables that may be useful to medical officers of health.¹

There are now four life tables for England and Wales—three calculated by Dr. Farr: No. 1, from statistics of population and mortality in 1841; No. 2, from the census enumerations of 1831 and 1841, and from the deaths of seven years (1838–1844); No. 3 from the census of the populations in 1841 and 1851, and upon 6,470,720 deaths registered in seventeen years, 1838–1854; No. 4, the new English life table, was constructed by Dr. Ogle, and was published in the supplement to the 4th Annual Report of the Registrar-General. Besides these tables we also have a healthy districts life table, constructed by Dr. Farr from statistics of sixty-three selected English districts.

TABLE IV.—*Showing the 'Expectation of Life' or Mean After Lifetime of Persons aged 0, 10, 20, 30, &c., according to the undermentioned Mortality Tables.*

Ages	English tables		Man- chester	Liverpool	Metro- polis	Surrey	North- ampton 1780	Carlisle 1816	17 officers' experience 1843	Institute of Actuaries 1869
	No. 2 1838–44	No. 4 1871–80								
0	40.36	43.56	34.2	26.0	37.0	45.0	—	—	—	—
10	47.47	49.24	40.6	41.0	45.0	49.0	39.78	48.83	48.36	50.29
20	39.99	40.53	33.3	34.0	38.0	42.0	33.43	41.40	41.49	40.06
30	33.21	33.25	26.6	27.0	30.0	35.0	28.27	34.34	34.43	34.68
40	26.46	26.38	20.6	21.0	24.0	28.0	23.08	27.61	27.38	27.40
50	19.87	19.80	15.2	16.0	18.0	21.0	17.99	21.11	20.18	20.31
60	13.60	13.69	10.3	—	—	—	13.21	14.34	13.77	13.83
70	8.55	8.61	6.8	—	—	—	8.60	9.18	8.54	8.50
80	5.00	4.99	4.6	—	—	—	4.75	5.51	4.78	4.72
90	—	—	3.2	—	—	—	2.41	3.28	2.11	2.36

Old and New English Life Tables, based respectively upon the Mortality in 1838–54 and in 1871–80. Also Liverpool Life Table, 1861–70, and Manchester, 1881–90.

Age	Of 1,000,000 born, the number surviving at the end of each quinquennial year					
	Males		Females		Liverpool persons	Manchester persons
	1838–54	1871–80	1838–54	1871–80	1861–70	1881–90 ²
0	1,000,000	1,000,000	1,000,000	1,000,000	1,000,000	1,000,000
5	723,716	734,068	750,550	762,622	539,630	698,140
10	689,857	708,990	715,769	738,382	498,621	664,390
15	672,776	696,419	696,917	724,956	482,892	648,180
20	651,903	680,038	671,119	707,949	462,768	634,040
25	624,221	657,077	644,342	684,858	434,497	614,440
30	595,089	630,088	612,774	658,418	—	587,060
35	564,441	598,860	579,908	628,842	360,344	552,230
40	531,657	568,077	545,844	596,118	—	510,960
45	495,770	522,374	510,403	560,174	275,193	463,860
50	455,727	476,980	473,245	520,901	—	410,870
55	409,460	424,677	438,381	477,440	184,224	350,420
60	356,330	365,011	388,974	422,835	—	282,810
65	294,588	297,156	324,165	356,165	97,635	210,020
70	223,490	222,056	253,161	277,225	—	138,860
75	148,076	144,960	174,800	190,566	34,155	77,380
80	80,343	77,354	100,394	108,935	—	35,010
85	32,979	30,785	44,419	47,631	6,003	12,220
90	9,321	8,015	13,802	14,225	—	3,160
95	1,628	1,183	2,704	2,533	417	590
100	154	82	295	225	9	80

The number dying under each age is readily ascertained by subtracting each figure from its predecessor.

¹ Extracts relating to this method are also given in Dr. Farr's collected works on 'Vital Statistics,' pp. 464, 465.

² Dr. Tatham's Manchester Life Table.

Life Insurance Tables.—Life insurance societies formerly used the Northampton and Carlisle tables, which have already been noticed as derived from bills of mortality. Dr. Farr considered that his life tables would afford a safer basis for the calculation of life insurance premiums and of the prices of annuities; but as these tables were derived from the rates of mortality of entire populations, and the clients of insurance companies are all selected lives, it is evident that the results would not be entirely satisfactory. Accordingly, actuaries have endeavoured to utilise their own experience. In the tables of the Equitable Assurance Society, published in 1825, and still more in the seventeen years' experience table, and in the healthy males table, they considered that they had obtained the needful accuracy. Even these statistics, however, are open to objection, because most of them are based upon the number of policies which became claims, and not upon the number of persons who were assured and died. As not a few persons insure in several offices, and as some are insured in over 100 different companies, their deaths would be counted several times over, and might injure the result if any of the deceased persons overstepped the average in any way.

2. '*Mean Duration of Life.*' '*Mean After Lifetime.*' '*Expectation of Life.*' '*Probable Duration of Life.*'

It will be seen that the three titles that come first are used as if they were synonymous, but as Dr. Farr remarks (8th Annual Report, pp. 279-80) '*expectation of life*' is an incorrect term; the time which it is *expected* a person will live is the time which it is an even chance he will live; it is the *vie probable* of the French, and is correctly expressed by '*probable lifetime.*' It is the age at which a given number of children born at the same time are reduced one-half; in other words, when the chance is equal of their dying before or after that age. The idea *intended* to be expressed by '*expectation of life*' is the *mean time* which a number of persons at any instant of age will live after that instant; it is the French *vie moyenne*, and this technical idea is strictly and shortly expressed by '*after lifetime.*' Upon referring to the fourth English life table it will be seen that the probable duration of life in England lies between 45 and 50 for males, and between 50 and 55 for females, whilst the mean after lifetime at birth for both sexes (Table IV.) is 48.56. This difference is to be accounted for by the fact that in an increasing population a certain number of persons at middle ages is added to it whose mortality is low. The mean after lifetime is calculated from one generation.

It has been shown by Dr. Farr that the mean duration of life, or mean after lifetime, may be approximately calculated from the birth-rate and death-rate by the following formula: Mean duration of life = $\left(\frac{2}{8} \times \frac{1}{D}\right) + \left(\frac{1}{8} \times \frac{1}{B}\right)$ when B = birth-rate per unit of population and D = death-rate per unit of population. Thus in Manchester with a birth-rate of 38 and death-rate of 29 the formula would be

$$\left(\frac{2}{8} \times \frac{100}{29}\right) + \left(\frac{1}{8} \times \frac{100}{38}\right) = 20 + 10 = 30 = \text{mean duration of life.}$$

Mean Age at Death.—This is the sum of the ages of a certain number of individuals at death divided by the number of deaths.

The method has been used in order to compare the viability of groups or classes of the population; but even when a sufficient number of deaths have

been used to obtain a correct average, it is only under certain severe restrictions that the comparison will be a sound one.

It can only be employed with safety, as a test or measure, in those cases in which the group or class to which it is applied is retained in a state of perfect uniformity, during the whole time comprised in the calculation. If, for instance, we compare the mean age at death of one class of the aristocracy with that of another, or the ages at death of different professions, the result will only be satisfactory if the individuals in question all start at the same age, and if they receive no accession in numbers during the time involved in the calculation.

If in any community there is no emigration or immigration, and the births and deaths are nearly equal for 100 years, the mean age at death will coincide with the mean future lifetime; but in England neither of these conditions are fulfilled, the births greatly exceed the deaths, and migrations are frequent: hence the mean age at death in England in 1842 was not 41 but 29. The error in the result by this method was twelve years in forty-one years.

Dr. Farr gives several examples of the extent of error possible by this method (6th Annual Report, p. 575), one of which may be given: 'Let us suppose,' he says, 'that in two contiguous parishes, equally healthy, A and B, all the children are born in A, and remain there up to the age of twenty, and if they die are registered in the parish register; that at twenty all the survivors emigrate, year by year, to B, and when they die are registered by the clergyman of B in his register. Now, if the mean age of death were taken in the parish A, it would be about four years (according to the present rate of mortality in England), while it would be sixty years in the parish B. The emigration of a part of the adult population from the country to towns produces an effect of precisely the same kind though to a less extent.'

Another example may be taken. An unhealthy military station, like Sierra Leone, might have its garrison constantly renewed by the arrival of fresh troops from England. These men might be killed off in a short time by the action of the deadly climate, and yet since the ages of these soldiers would not be less than twenty to thirty years, the average age at death would be very high, and would consequently indicate a very healthy condition as compared with English country districts.

The mean age at death gives information as to the ages of the dying, and *per contra* of the living, in different communities, but it does nothing more.

Standards of Comparison.—In order that statistics may be of service for the purposes of public health, it is important that they should be comparable. Thus, it is often necessary to compare the condition of a community at one period with its condition at another period; or, again, we may need to contrast its sanitary state with that of some other community.

Comparisons are essential to the sanitarian as tests of his progress, and as a means of determining the true causes of disease.

It is sometimes possible to make a direct comparison between the gross mortality of a place at one time with that at another time, or with that of another place; and the number of cases of, or the deaths from, certain diseases may be noted within a given period, say a week, month, year, or ten years, and the increase or diminution of these numbers at a subsequent period, in similar spans of time, may be a measure of the sanitary state of the community. But in order that deductions from these figures may be useful, it is imperative (1) that the rules already laid down should be observed, and (2) that the method should only be applied to places of the same number and class of inhabitants, or to the same place, unaltered in the constitution of its inhabitants at different periods of time.

It need hardly be said that these conditions are very seldom fulfilled, either in different towns, or in the same town at different periods of time. It is, moreover, frequently desirable to compare towns or communities having populations differing in numbers. It thus often becomes necessary to fix upon some common standard with which the number of births and deaths, or deaths from different diseases, may be compared in the first instance; in other words, to make an *indirect* comparison.

Comparison with Total Deaths.—By some statistis the number of deaths from certain classes of disease for every 1,000 *deaths from all causes* is taken as a basis for comparing the sanitary condition of different towns.

The latter method has been employed very legitimately by Dr. Farr in some of the returns of the Registrar-General. In his tables it serves to show the relative proportions in which the several morbid agencies affect the health of the nation at different times, and he does not attempt to draw any other conclusions from them. But it has been used with less caution, for instance, by Dr. Lombard in his '*Traité de Climatologie Médicale*.' It is here used to indicate the relative degree to which different nations or different towns are subject to certain classes of disease. Very fallacious results may, however, be drawn from such tables as these; and to prove this it will be sufficient to consider the result of the method in only one case. For the sake of simplicity, it shall be a supposititious one; but it would not be difficult to produce a parallel case in actual fact, if it were needful.

We will suppose, then, that in a certain town of 100,000 inhabitants the total number of annual deaths is 2,000, and that out of these, 500 are deaths from phthisis; in other words, that the rate of mortality is 20 per 1,000 of the population, and that the phthisis-rate is 5 per 1,000.

By the above-mentioned method, then, the proportion of phthisis deaths to the total deaths would be 250 per 1,000.

In another town of the same size the total number of deaths is 4,000 per annum, i.e. its rate of mortality is forty per 1,000 of population. In this town the number of deaths from phthisis is 1,000—twice as many as in the former place—but, according to Lombard's calculations, the proportion of phthisis deaths to the total mortality will be the same as before, as 1,000 to 4,000, or 250 per 1,000. It would thus appear that, so far as regards consumption and its causes, this town is in no worse condition than the other, whilst in reality their intensity is doubled. The source of the fallacy in this conclusion is sufficiently obvious; it lies in the want of homogeneity or similarity in the standard selected as common to the two places.

2. *Comparison of Deaths with Births.*—Similar objections would apply to taking the number of births as a standard with which to compare the deaths. The number of births per 1,000 of population varies greatly in different towns, owing to conditions quite independent of their sanitary state; such, for instance, as the prosperity or otherwise of the inhabitants, the price of corn, or the activity of trade, affecting the marriage-rate, and after this the birth-rate. It is thus quite unsafe to take the number of the births as a basis for comparison.

3. *The Registrar-General's Method.*—It is probably owing to the uncertainty of other standards that the deaths and other items of vital statistics are usually compared with each 1,000 or 100,000 of the population; and there can be no doubt that this is the most satisfactory method, if only proper precautions be taken. It is the method employed by the Registrar-General, and is very simple in its calculation.

The marriage-rate, birth-rate, and death-rate of any place are all found by a simple rule-of-three sum; thus, if the population of the place, determined by the results of the census, be 81,584, and the number of births

and deaths taking place during the year are respectively 1,192 and 549, then

$$(1) \frac{1192}{81,584} \times 1000 = 37.8 = \text{birth-rate.}$$

$$(2) \frac{549}{81,584} \times 1000 = 17.0 = \text{death-rate.}^1$$

The *marriage-rate* is of importance to the statesman as showing the relative prosperity of the mass of the population in different years, but it is also of interest to the sanitarian from its relation to the subject of the fecundity of marriage, and the bearing of this factor upon the numbers of the population at the different ages and their several rates of mortality. It may be stated that the marriage-rate per 1,000 of population has fallen in England and Wales from 17.6 in 1878 to 14.2 in 1887, and in Scotland the rates in these years were respectively 15.5 and 12.5. A similar reduction has been noted in other European countries. Owing to the higher wages to be obtained in towns, and to the preponderance of adults between twenty and forty years of age, it is always greater in urban than in rural districts. Many persons also resort to towns merely to be married, where their marriages are registered, and they subsequently return to the rural districts.

The age at marriage is not always accurately determined, but, on the whole, since 1878, when the prosperity of the country ceased to advance 'by leaps and bounds,' this age for bachelors has risen from 25.6 to 26.2, and for spinsters, from 24.2 to 24.7.

The *birth-rate* of the whole country has also diminished from 36.8 in 1876 to 31.4 in 1887, the highest rates taking place in industrial, the lowest in agricultural districts, partly owing to the above-mentioned causes of higher marriage-rates, and partly owing to the greater mortality of infants in towns. By shortening the period of suckling, this mortality, by a physiological law, diminishes also the intervals of child-bearing.

Quarterly, monthly, and weekly returns are calculated by dividing the estimated population by 365.24226, the number of days in a natural year; the dividend is then multiplied by 7, to give the weekly population.²

The *zymotic death-rate*, as it is called, is based on the total number of deaths occurring in a district during the year from seven zymotic diseases—small-pox, measles, scarlatina, diphtheria, whooping cough, fever (including typhus, enteric, and other forms of continued fevers), and diarrhoea. The objection to thus grouping all these disorders together has already been pointed out.

No valuable information as to the sources of mortality from any of them can thus be obtained; and if it is supposed that the extent of preventable mortality may be estimated from such a classification, the deduction is fallacious, seeing that many other diseases are as distinctly preventable as those enumerated. It would be far better to give the rates per 1,000 or 100,000 of

¹ By the use of a slide-rule, especially by means of Col. Fuller's machine, described by Dr. Paddock Bate in the *Transactions of the Society of Medical Officers of Health*, a very close approximation to these rates may be obtained without actual calculation. Dr. Longstaff states in *Studies in Statistics* that he uses M. Thomas' 'Arithmomètre' with much advantage.

² Dividing simply by 52.17747, the number of weeks, gives the same result, or by the number of days in a month or a quarter, in order to arrive at the population which may be applied to the births or the deaths in a month or a quarter, and the rates per 1,000 of such population are obtained in the manner just described. The result is a supposititious annual rate—an annual rate per 1,000 of population—on the supposition, that is, that the same proportion of births or deaths *would* occur throughout the year, as have actually occurred during the week, or month, or quarter, under consideration. Weekly rates are now published by the Registrar-General for large towns with an aggregate population of about nine millions.

the population for each disease separately, and then the special causes of excessive mortality would be pointed out, and might admit of remedy.

The *infantile death-rate* is not measured by the proportion of infants' deaths to the population; it is the proportion the deaths of children under one year of age bear to the births registered, and is thus expressed as so many deaths to every 1,000 births. In this way the enumerator avoids the possibility of error, due to the faults of the census which have been already mentioned.

The death-rates of children under five years of age are also not estimated upon the total population, but upon the number of children living under five years of age.

The infantile mortality also depends somewhat upon the proportion of male to female births, as the rate of mortality at birth is greatest amongst male children, the ratio being as 64 to 49.

The proportion varies somewhat from year to year, and in different countries. Thus in the ten years, 1888-47, 105 males were born to every hundred females; in 1868-77, 108·9 males were born to every hundred females.

In France the proportion is 106·5; in Russia, 108·9; in Holland and Belgium, 106·4; in Sweden, 104·6.

The proportionate mortality of legitimate and illegitimate children is not given in every return of the Registrar-General, but it was furnished by Dr. Farr in several of his letters. In the 38th Annual Report he shows that the mortality of children born out of wedlock is twice, or in some districts three times, as great as that of legitimate children, and thus indicates their unfavourable position: 'Who can doubt,' he says, 'that their bringing up is much harder and more difficult; that the existence of a class of men bound to society by few or no family ties is not a matter of indifference to the State? It is beyond doubt that fewer illegitimate children grow up to maturity; that they get through the world with more trouble than children born in wedlock; that more of them are poor; and that therefore more of them become criminals' (quotation from Bernouilli).

The percentage of illegitimate to total births in England in 1884 was 4·7. In 1875, in Liverpool, their death-rate was 418; in Preston, 448, the death-rates among the legitimate being 205 and 214 respectively. In Driffield the respective rates of legitimate and illegitimate were 168 and 596.

The following table gives the average rates of mortality in successive decades, and in 1885 and 1890 from all causes, and from the more important causes of death. It is desirable that those for the later years should be borne in mind, when a comparison of similar rates in any given district with those of the rest of the kingdom is needed.

TABLE V.—*Annual Mortality per Million living, at all Ages, in three Decennia, from several causes.*

	1851-60	1861-70	1871-80	1885	1890
All causes	22,165	22,416	21,272	19,010	19,548
Small-pox	221	168	236	100	1
Measles	412	440	378	530	439
Scarlet fever	876	972	716	230	242
Diphtheria	109	185	121	160	179
Whooping cough	508	527	512	480	478
Fever	908	885	484	200	197
Diarrhoeal diseases	1,080	1,076	935	500	606
Cancer	817	887	478	570	676
Phthisis	2,679	2,475	2,116	1,750	1,681
Diseases of respiratory organs	3,021	3,364	3,760	3,850	4,229
" nervous system	2,741	2,785	2,770	2,600	2,494
" heart, &c., including dropsy	1,247	1,849	1,477	1,600	1,757
Infants, under five, per million at those ages	67,602	68,305	68,116	52,900	—

Sources of Fallacy in Death-rates.—Before proceeding to point out the applications of the figures given by the Registrar-General, it will be necessary to inquire in some detail into the several sources of error that they contain, and which may interfere with their trustworthiness. Before they can be applied to the investigation of the relative healthiness of districts, the limits of possible error must be ascertained, and also how far erroneous conclusions may be obviated.

1. *Age and Sex Distribution.*—It will have been apparent from the tables already given that the mortality differs considerably in the two sexes, and at different ages; hence populations cannot fairly be compared with each other as regards their death-rates, unless the towns thus put into comparison show no material differences from each other in respect of the age and sex distribution of their populations. As the Registrar-General says in his annual summary for 1888, 'it is self-evident that if one town contains a much larger

TABLE VI.

Towns in the order of their corrected death-rates	Standard death-rate ¹	Factor for correction for sex and age distribution ²	Recorded death-rate	Corrected death-rate ³	Comparative mortality figure ⁴
	Column I.	Column II.	Column III.	Column IV.	Column V.
28 Towns	19.96	1.0657	21.57	22.99	1,178
Bristol	20.55	1.0851	17.90	18.53	949
Norwich	22.24	0.9565	19.64	18.79	963
Derby	20.45	1.0402	18.09	18.82	964
Portsmouth	20.65	1.0801	18.61	19.17	982
Brighton	20.66	1.0296	19.22	19.79	1,014
Bradford	19.26	1.1045	18.84	20.26	1,038
Leicester	20.81	1.0474	19.40	20.32	1,041
Plymouth	21.48	0.9908	20.86	20.66	1,058
Birkenhead	19.89	1.0695	19.86	21.24	1,088
London	20.04	1.0615	20.44	21.70	1,112
Halifax	19.58	1.0864	20.04	21.77	1,115
Wolverhampton	20.68	1.0811	21.27	21.93	1,123
Cardiff	19.60	1.0853	20.21	21.93	1,123
Nottingham	20.07	1.0599	21.26	22.53	1,154
Birmingham	19.95	1.0668	21.34	22.75	1,165
Huddersfield	19.37	1.0982	21.80	23.39	1,198
Hull	20.62	1.0816	22.80	23.52	1,205
Bolton	19.41	1.0959	21.68	23.76	1,217
Salford	19.54	1.0886	22.41	24.40	1,250
Oldham	19.17	1.1097	22.00	24.41	1,251
Sheffield	19.78	1.0754	22.93	24.66	1,263
Leeds	19.90	1.0689	23.26	24.86	1,274
Sunderland	20.43	1.0412	24.54	25.55	1,309
Blackburn	19.52	1.0898	24.50	26.70	1,368
Newcastle-on-Tyne	20.10	1.0583	25.46	26.94	1,380
Preston	19.59	1.0859	25.48	27.67	1,418
Liverpool	19.39	1.0971	26.69	29.28	1,500
Manchester	19.09	1.1148	27.64	30.80	1,578

¹ The standard death-rate signifies the death-rate at all ages, calculated on the hypothesis that the rate at each of twelve age periods in each town was the same as in England and Wales during the ten years 1871-80, the death-rate at all ages in England and Wales during that period being 21.37 per 1,000.

² The factor for correction is the figure by which the recorded death-rate should be multiplied in order to correct for variations of sex and age distribution.

³ The corrected death-rate is the recorded death-rate multiplied by the factor for correction.

⁴ The comparative mortality figure represents the corrected death-rate in each town compared with the recorded death-rate, at all ages, in England and Wales in 1888, taken as 1,000.

proportion either of very old persons or of very young children than another, its general death-rate will necessarily be higher than that of such other, although the two are on an exact equality as regards the death-rates at the several age periods; and so it will also be if the one town has a much larger proportion of males than the other, inasmuch as the male death-rate is

almost invariably higher than the female death-rate. Thus, taking the population of each of the twenty-eight towns, with the age and sex distribution shown at the last census, and applying to it the mean annual death-rate for each sex at each age period in England and Wales in 1871-80, we have a series of death-rates, which are given in Column I. of the table on page 495. It will at once be seen that these rates differ considerably from each other, and from the rate in England and Wales as a whole. The differences are caused simply and wholly by difference in sex and age distribution, the rates being those which would have been recorded in the towns as their general death-rates, had their male and female populations been equally healthy with the male and female population of the entire country at each separate age period. It will be convenient to speak of these hypothetical death-rates in Column I. as the *standard* rates. Before, therefore, the *recorded* death-rates in the towns can be fairly put into comparison with each other as measures of healthiness, they must be corrected for these inherent differences of the *standard* rates; and it is plain that the correction for 1871-80 will be duly made if the recorded rate in each town be multiplied by the number against the town in Column II., this number being obtained by dividing the annual death-rate in England and Wales in the decade (21.27) by the standard death-rate in Column I. The recorded death-rates in the several great towns in 1888 are given in Column III.; multiplying these by the factors, we obtain the corrected death-rates in Column IV. These are the death-rates that would have been recorded in each town had its population been identical, so far as age and sex distribution are concerned, with the population of England and Wales; and it will at once be seen, on comparing the recorded with the corrected rates, that the mortality of the towns as compared with that of the entire country, is, with but few exceptions, very much greater than would be concluded from the recorded general death-rates. The towns contain, as a rule, a much smaller proportion of aged persons, and a much higher proportion of persons in the prime of life, as also a much higher proportion of females, than does the country at large; and though these advantages are somewhat counterbalanced by an excess in the proportion of children, they are so to a limited extent only.'

By this method we are saved from the errors arising simply from differences in the age and sex distribution of the population, from whatever cause these may arise, whether from different birth-rates or the immigration or emigration of the people. A still simpler method of correcting the general death-rate may be adopted by taking advantage of the death-rates at each successive age period, which are given in the supplement to the 45th Annual Report of the Registrar-General, for each district in the kingdom. These death-rates in any one district may be applied by the rule of proportion to a population of 1,000 persons having the same age distribution as the population of England and Wales. The following table gives the relative numbers required:—

TABLE VII.—*Age Distribution of Population (Persons) of England and Wales (Mean of 1871 and 1881).*

All ages	Age periods									
	Under 5	5-	10-	15-	20-	25-	35-	45-	55-	65- 75 and upwards
1,000	186	190	107	97	89	147	118	86	59	33 18

We will take the town of Stockport as an example of the process. The figures given by Dr. Ogle in his supplement are as follows :—

Annual death-rate, all causes . . .	All ages	Under 5	5-	10-	15-	20-	25-	35-	45-	55-	65-	75 and upwards
Population . . .	107,433	13,794	12,006	11,338	10,908	10,013	16,506	12,793	9,878	6,893	3,116	891
Deaths in one year, all causes . . .	2,657	1,108	96	43	65	79	168	181	213	278	270	168
Corrected rate per 1,000 of popula- tion	25.2	10.9	0.9	0.4	0.6	0.7	1.4	1.6	1.8	2.5	2.8	1.7

The figures in the fourth row (corrected rates) are obtained by the rule of proportion, thus :

$$\frac{1108 \times 186}{18794} = \frac{150888}{18794} = 10.9.$$

If 13,794 children under five yield 1,108 deaths, 186 will yield 10.9.

2. *The Emigration of Persons from a Place shortly before Death.*—One important source of error remains unaffected, namely, the registration of deaths in places distinct from those in which the fatal disease originated. This occurrence takes place more frequently than is commonly supposed, and to an extent sufficient to greatly injure death-rates as a means of comparison for health purposes. It may take place in the case of workpeople who have been attracted from the country to a town by the prospect of employment, and whose health has been injured by it to the extent of contracting a fatal disease. They are obliged to leave their work, and return to their homes in the country to die amongst their friends.

But the cases in which this emigration to die are most common are those of domestic servants. When a servant is taken ill with some serious illness, in far the majority of instances, he or she is sent away to friends; and this fact is the chief cause of the abnormally low death-rates of the suburbs of towns. Many suburban districts show through a series of years impossible death-rates of 12 or 10, or even 8 per 1,000; and when it is considered that a death-rate of 10 per 1,000 means that, if the population were stationary, every person in the place would live on the average to 100 years, and that if any die under that age, others must live to a proportionate extent beyond that age, in order to keep up the average, it will be seen that the phrase 'an impossible death-rate' is not too strong. A mortality rate of only 12 per 1,000 means an average age at death of 88; and 15 per 1,000, an average age at death of 66. In many health resorts also there is the further probability that many visitors whose presence has been noted at the time of the census return home before death takes place. In no other way than by one of these two occurrences can we account for such a death-rate as that of Malvern, for instance, of 9.12 per 1,000, which is duly advertised in railway guides, and in other ways, as a proof of the extraordinary healthiness of the place.

This source of error not unfrequently injures the comparative value of even town death-rates, for one town may include more suburban districts than another; and where there are many well-to-do people resident within its borders—people who employ many domestic servants—its death-rate will be unduly lowered by the cause we have just mentioned, and the average of the whole town will be more favourable than one inhabited almost entirely by a working-class population.

8. Another source of error in death-rates is the presence in a town of

large hospitals, workhouses, and public institutions; and this is especially the case in county towns, into which people from the country are received, in many cases to die there. The Registrar-General, however, always notes the fact of the presence of these institutions, and thus calls attention to a probable source of fallacy.

4. Errors in connection with the schedules of disease, the causes of death. In the Registrar-General's returns the causes of death, or rather the names of the diseases which are supposed to have immediately preceded the deaths, are recorded under the heads of a carefully framed classification of diseases.

These nosological tables have been greatly improved since they were first brought out, and they are now apparently constructed with a view of obtaining some idea of the true causes of death and the various injurious conditions which have led to the diseases finally entered on the certificates of death.

The defects in these records are to be found, not in the arrangement of the statistical tables, but in the data upon which these are founded.

However perfect the grouping of diseases in separate classes and orders, the tables are of little value if the diseases which they include have been imperfectly observed or wrongly entered.

Unfortunately, owing to the defects enumerated on pp. 475 and 476, the evidence given by these elaborately arranged statistics is almost useless for the purpose of comparing one place with another, or one period of time with an earlier or later period.

Many of these defects would probably be greatly reduced in number and in their evil influence by the adoption of Dr. Farr's suggestion for the appointment of a Registration Medical Officer in each superintendent-registrar's district. But for the present it is necessary to warn those who consult the returns that no certain conclusions can be drawn from them.

Even at the best, as Mr. Humphreys has pointed out ('Vital Statistics,' p. 115), 'causes of death in the death register are necessarily little more than the more or less trustworthy guesses of a large body of more or less skilled observers.' 'Statistics of causes of death should therefore be compiled with caution, and without any attempt at over-elaboration of detail. Still greater caution should be used in drawing inferences and deductions from a comparison of the results for a series of years.'

The Distribution of Death and Disease

Some of the results of the study of vital statistics may now be given.

Dr. Headlam Greenhow in the year 1858 presented to the General Board of Health his 'Papers relating to the Sanitary State of the People of England,' the results of inquiry into the different proportions of death produced by certain diseases in different districts in England. These diseases were arranged in ten groups—1, Pulmonary affections; 2, contagious diseases; 3, alvine flux; 4, typhus and erysipelas; 5, croup, influenza, and ague; 6, strumous diseases; 7, nervous diseases of children; 8, apoplexy and paralysis; 9, rheumatic fever and rheumatism; 10, carbuncle and phlegmon. These papers were subsequently followed up by reports to the Privy Council by Dr. Greenhow and others on these and other subjects, such as industrial diseases, cholera, phthisis, excremental nuisances in relation to disease, small-pox, cancer, &c.

All these papers are of great interest and importance, but they must be studied in the official reports to the Privy Council and to the Local Govern-

ment Board, together with the remarks made upon them by Sir John Simon and Sir George Buchanan.

One of the most important deductions from Dr. Greenhow's researches was the demonstration that a large part of the mortality in England and Wales was preventable.

Preventable Mortality.—Sir John Simon remarked in his preface to Dr. Greenhow's papers that there were then 'strong *prima facie* grounds for believing that the local excesses of fatality are due to local circumstances of aggravation; that these aggravating local circumstances are such as it is fully possible to counteract; and that of the total mortality ascribed to these influences in England a very large share is preventable.

He afterwards infers that this preventable disease occasions 'at least a quarter of the mortality of England,' about 100,000 deaths at that time.

That this was not an over-estimate is now proved by the facts—1, that the then death-rate of 28·1 per 1,000 has been reduced to 19·5—8,500 per million—more than 90,000 at the present population; and 2, that many thousand preventable deaths still take place annually.

Such fulfilment of prophecy ought to give statesmen confidence both in the accuracy of forecast on the part of our leading sanitarians, and in the efficacy of the measures which they have recommended for the reduction of the death-toll, now and in past times, levied upon the nation.

As results of these researches on the part of Dr. Greenhow and his successors, many of the influences bearing upon the production of disease have been discovered.

Thus pulmonary affections have been found to be closely related to irritating dusts and fumes, to ill-ventilated rooms and workshops, to indoor employments and stooping postures when at work.

Consumption has been traced to the breathing of air charged with respiratory impurity, and to the influence of a damp, non-porous subsoil.

Epidemic diseases, notably scarlet fever and cholera, are found to affect especially certain districts, and the same is true of diarrhoea and dysentery.

Diarrhoea and enteric fever have been traced with certainty to the contamination of drinking water by faecal matter, either of a specific or non-specific character. Diphtheria, enteric fever, and scarlet fever have also been connected with polluted milk and with imperfect drainage; typhus fever with overcrowding.

Scrofula has been shown to be more fatal in country districts, and the nervous diseases of children, including convulsions, in manufacturing towns, especially where females work in factories, and also in Caernarvon.

Heart disease and rheumatism are most common in valleys, and in places protected from the influence of prevailing winds, and cancer 'in the sheltered and low-lying vales traversed by *fully formed* rivers, and having sites composed of the more recent geological formations' ('Haviland's Geography,' p. 86).

These results have been obtained, for the most part, by the use of the Registrar-General's returns; and, notwithstanding the inherent defects of these statistics they are well worthy of study, together with the other original researches promoted by the medical officers of the Privy Council and the Local Government Board. But they are chiefly important in regard to the question of the origin of disease.

More immediately connected with the statistical work of a medical officer of health are the facts ascertained concerning the distribution of death and disease in relation to—1, age; 2, density of population; 3, climate and season; 4, occupation.

1. Age.

In the Supplement to the Registrar-General's 35th Report, Dr. Farr describes 'the march of an English generation through life,' and, although space does not permit us to give it in full, it will be necessary to lay his main conclusions before the reader.

Age Nil to Five.—The first thing to observe is that the fatality children encounter is primarily due to the changes in themselves. Thus 1,000,000 children just born are alive, but some of them have been born prematurely; they are feeble; they are unfinished; the molecules and fibres of brain, muscle, bone, are loosely strung together; the heart and the blood, on which life depends, have undergone a complete revolution; the lungs are only just called into play. The baby is helpless; for his food and all his wants he depends on others.

It is not surprising, then, that a certain number of infants should die; but in England the actual deaths in the first year of age are 149,498, including premature births, and death by debility and atrophy; diseases of the nervous system 80,687, and of the respiratory organs 21,995. The total dying by miasmatic diseases is 81,266.

In the second year of life, pneumonia, bronchitis, and convulsions are still the prevalent and most fatal diseases; many also die then of measles, whooping cough, scarlatina, and diarrhoea. Scarlet fever asserts its supremacy in the second, third, fourth, and fifth years of age. Whooping cough is at its maximum in the first year, measles in the second, scarlatina in the third and fourth years.

The deaths from all causes under the age of five years are 268,182, in the healthy districts only 175,410; but in Liverpool at that date (1875) 460,870, or nearly half the number born, died in the five years following their birth.

Age Five to Ten.—The total deaths in the five years following are 84,809: 8,748 of them from scarlatina, the principal plague of this age; 1,864 from diphtheria, 4,086 from fever. More than half the deaths in this young age are from miasmatic disease, in all 19,256.

Age Ten to Fifteen.—But 702,509 survive and enter in this age, which culminates in puberty; and 684,568 pass through it into the next, for the deaths are fewer than at any other age. They amount to 17,946, of which 1,901 are by scarlatina, 2,842 by fever, 8,526 by phthisis.

Age Fifteen to Twenty.—Now the mortality increases, especially among women, of whom 2,268 die of consumption and 244 in childbirth, for at this age a few girls marry with some risk to their lives. The deaths of males by consumption are 8,811, by fever 1,368.

Age Twenty to Twenty-five.—At this age large numbers marry. The deaths are 28,705, of which nearly half, or no less than 18,785, are by phthisis; 1,100 women die in childbirth.

Age Twenty-five to Thirty-five.—Of the million, 684,045 attain the age of twenty-five, and 271,998 live to the age of thirty-five. Consumption is the most fatal disease of the age: it is the cause of 27,184 deaths, women suffering more than men. Fever is fatal to fewer lives than it was earlier; but it is by far the most fatal of the zymotic diseases, and slays its 4,197.

Age Thirty-five to Forty-five.—The losses are of 69,078 lives: 35,142 men, 33,936 women. Many of the structures now give way. Phthisis still predominates; fever still snatches its many victims; and the brain, heart, lungs, and bowels become more and more the seats of destructive disease: 564 persons commit suicide, 8,280 die violent deaths; 2,907 of them men and 878 women; 2,516 mothers die in childbirth.

While the deaths by fever are 8,777 out of 571,998 attaining this age in England, 14,822 people die of it in the Liverpool district out of 860,844. The lung diseases in the two sets of conditions are fatal to 7,452 and 18,967 lives. In the healthy districts the deaths by fever are 2,700; by diseases of the lungs, 5,261.

Age Forty-five to Fifty-five.—This age is the middle arch of life, for the million are reduced to half a million lives a few months after the age of forty-five. (*Mem.* This age is now extended to forty-nine.)

The deaths by all causes are 81,800: by fever, 8,749; diarrhoea, dysentery, and cholera, 1,944; by phthisis, 16,468; by lung diseases, 18,203; heart diseases and dropsy, 10,041; brain diseases, 9,818; bowel and liver diseases, 7,917. The centres of life are sources of death. Cancer, a formidable and dread disease that began to be fatal before, now destroys 4,588 lives: 1,140 men, 3,448 women.

Age Fifty-five to Sixty-five.—The number of males and females surviving becomes equal at the age of fifty-three, but at and after fifty-five the women exceed the men in number, as their mortality rate is lower ever after the age of thirty-nine.

While 421,115 of both sexes enter this stage of life, 809,029 live on to the next; 112,086 die, only 9,795 of fever, diarrhoea, dysentery, cholera, rheumatism, and other zymotic diseases. Cancer kills 5,998 persons, 4,085 of them women; consumption 10,445. The diseases most to be dreaded and guarded against, especially by men, are affections of the lungs and heart, of which 28,659 and 17,081 persons die. Diseases of the brain are fatal to 15,678; of the stomach and intestines and liver are fatal to 11,400.

Age Sixty-five to Seventy-five.—809,029 enter this age, and 161,124 leave it alive. The 147,905 dying in this period succumb to the same classes of disease as were fatal in the previous decenniad. The year of age seventy-two is that in which the greatest number of *men* die, and which may have led the Psalmist to say, 'The days of the years of our life are threescore years and ten;' but these are 'days passed away in Thy wrath,' in violation of the divine laws, and therefore are not necessarily the limit of healthy existence where the laws of life are observed.

Age Seventy-five to Eighty-five.—The numbers that enter this decenniad are 161,124, and the numbers that leave it alive are 88,565. The 122,559 that die of recognised diseases at this age die chiefly of lung, brain, heart, and other local diseases.

Age Eighty-five to the End.—Of the 88,565 who enter this age, only 2,158 live to the age of ninety-five, and 228 to 100. Finally, by this table, at the age of 108 one solitary life dies.

Such and so curious are the setting of 40,858,184 years of English life.

Influence of the Birth-rate upon Mortality

It has already been shown that the proportion of deaths to living persons is enormously great in the first five years of life,¹ and hence it might naturally be supposed that, apart from sanitary conditions, those populations would have the highest death-rates which contained the largest proportion of children under five years of age; in other words, that a large birth-rate necessarily increases the death-rate. But the problem is by no means so simple as this.

¹ In the 16th Annual Report of the Registrar-General, p. xiv, Dr. Farr remarks that 'the mortality (73 per 1,000) of children under five years of age is nearly three times the average rate of the whole population, while among boys and girls of the age ten to fifteen the mortality is at the rate of 5 per 1,000, only one-fourth or one-fifth of the general rate; and the mortality remains below the average until the age of fifty-five, but becomes after that age much above the average.'

It is quite true that in a stationary population, i.e. one in which the death-rate equals the birth-rate, or again in a population in which the only increase is from immigrants over five years of age, then the greater the number of children born, the higher will be the death-rate; but in an increasing population, in which the births exceed the deaths, a certain number of the children who are in excess survive to the healthier periods of life, and thus lower the death-rate in proportion.

A regular increase of the population of this kind has indeed the effect of increasing the proportion of children, under five years of age, who die off quickly; but it has also the effect of still further increasing the proportion of the living at ages from five to fifty-five, and of diminishing the proportion of old people, whose rate of mortality is also high.

The difficulty is to find the exact point at which these two opposing currents of influences counteract each other; but we may say, in general terms, that a high birth-rate can only lower the general death-rate when a sufficient number of the children thus born in excess survive to the healthy or low-rate ages to more than counterbalance the high mortality under five years of age.

A good example of the way in which the death-rate may be favourably affected by the birth-rate is afforded by Aston Manor,¹ a suburb of Birmingham, with a population in 1877 of 46,462, which had risen in 1888 to 64,920. Its birth-rate at the former period was 48·2, and had been higher; in 1888 it was only 31·7. The high birth-rate in the former period created an undue proportion of children during the succeeding years, a certain number of them surviving to the later period. The result is seen in the reduction of the death-rate from 18·4 in the ten years ending 1881, and in the seven following years it averaged 15·7.

This diminution was probably due (1) to the survival of a number of children to healthy ages, and (2) to the lowered proportion of infants in the later years.

Dr. May suggests that the marked decline in the general death-rate of the country may be partly explained in this way.

2. *The Relations between Density of Population and the Rate of Mortality*

It is to the late Dr. Farr that we owe any exact knowledge we possess of the relation between the density of populations in towns and their rates of mortality.

His first work upon the subject was published in the 5th Report of the Registrar-General for the year 1848.

In his letter to the Registrar-General, 'On the Causes of the High Mortality in Town Districts,' he showed that in the metropolis, in portions of the town inhabited by much the same classes of people, and with the sewerage and supply of water nearly the same, but in which the density of the population is different, the mortality varies directly as the density, although it does not increase in the same ratio.

He shows that this proposition is true not only of such poor districts as Whitechapel, Shoreditch, and Bethnal Green, but also of the wealthiest parts, such as St. James's (City of London), and St. George's (Hanover Square), and by grouping together the districts, and taking the mortality amongst females only, he constructed the following table:—

¹ *Public Health*, March 1889, p. 343. Communication from Dr. May.

—	Annual mortality of females	Density of population
	One in	Square yards to one person
Ten unhealthiest districts . .	36	32
Ten medium districts . . .	41	102
Ten healthiest districts . . .	49	202

From these and from other statistics he deduced the law that the mortality increased as the sixth root of the densities, and this ratio was so exact that if d and d' represent the density of the population in two places, and in m and m' their ratio of mortality, then

$$\frac{m'}{m} = \sqrt[6]{\frac{d'}{d}};$$

and, consequently, it became possible to express the mortality of one place, such as Whitechapel, in terms of the mortality of Bethnal Green, and the densities of Bethnal Green and Whitechapel,

$$m' : m :: \sqrt[6]{d'} : \sqrt[6]{d}$$

or,

$$m' = \sqrt[6]{\frac{d'}{d}} \cdot m.$$

Thus the mortality of Whitechapel being .02978 per cent., and the densities of the population being, in Whitechapel, 127,818 persons to a square mile, and in Bethnal Green 62,890, then

$$\sqrt[6]{\frac{62890}{127818}} \times .02978$$

gives .0264 per cent. as the rate of mortality of Bethnal Green, the actual observed mortality being .0262 per cent.; or, again, taking the numbers given in the above table—

$$\sqrt[6]{\frac{32}{202}} \times 49 = 36,$$

the number living to one death in the unhealthiest districts. So closely is this ratio followed in places differing only in their density of population that Dr. Farr even proposed that in any sanitary inquiry the influence of density should first be discovered by means of his formula, and that the influence of other conditions should be noted in any excess or deficiency in the resulting figures. As he remarks: 'The formula thus eliminates the element of density from the analysis of the causes of insalubrity.'

In his supplement to the 35th Report of the Registrar-General, Dr. Farr returns again to this subject, and though he slightly alters his formula, and now substitutes the power of 0.12 for the sixth roots of the densities, this expression is mathematically almost the same, and he repeats (p. clviii): 'The mortality of districts is nearly as the sixth root of the proximities.'

The following table gives the result of more recent statistics:—

Mean annual death-ratio	Density. Persons to a square mile	Births	Deaths	Annual increase of population
15 89	315	35.11	22.00	11.99
15 17	166	30.22	16.75	14.69
18 20	186	32.19	19.16	7.53
21 23	379	35.78	21.88	18.82
24 26	1,718	38.75	24.90	18.69
27 30	4,499	40.16	28.08	12.28
32 (Manchester)	12,375	37.33	32.49	8.22
39 (Liverpool)	65,823	37.57	38.62	—

It is not surprising that Dr. Farr, in his first attempts to unravel the complex conditions of society, should try to single out some *one* influence that in its variations should account for alterations in the rate of mortality. Accordingly, he passes in review the several sets of influences affecting the lives of men, such as a command of the necessities of life, soil and atmosphere, climate and seasons, winds, temperature, moisture, and electricity; but he shows that, with regard to the first named of these conditions, the dweller in the town is better off than the dweller in the country, and that all meteorological influences must fall very equally upon town and country districts.

There remains, however, another class of causes of disease to which these statements do not apply, and these are 'atmospheric impurities, organic matter undergoing decomposition, and the contagious particles of zymotic disease.'

It is to these causes, he says, 'that the high mortality of towns is to be ascribed.' 'The people live in an atmosphere charged with decomposing matter of vegetable and animal origin.'

It will be noticed that we have here simply given Dr. Farr's earliest views as to the cause of an increase of mortality with increase of density of population. He would, however, himself have admitted the existence of other factors towards this result. He has elsewhere pointed out the distinct deterioration of populations as they are more and more closely congregated together. The localities in which they live are the lowest, the property the worst built, and the surroundings the most unhealthy. The people themselves are, for the most part, those with the lowest standard of vitality, prone to hereditary diseases, such as congenital syphilis, tuberculosis, &c., and most careless about their health. Intemperance and immorality are rife amongst them, and they have few natural pleasures to raise them from their constant condition of nervous depression.

It may at first sight seem strange that out of such complexity of influences there could flow such uniformity of results, but the law of averages will accomplish a good deal, and we cannot resist the conclusion that all the conditions mentioned above take part in producing the effect upon the death-rate which we have been considering.

8. *The Influence of Atmospheric Elements upon Mortality*

The influence of season and weather upon mortality and sickness must be carefully taken into account in any estimate respecting the health of a community.

The relation of atmospheric changes to disease has indeed been recognised from very early times. Hippocrates devoted one of his works to 'Airs, Waters, and Places,' and his writings upon epidemics and his *aphorisms* abound with remarks upon the influence of various states of the weather upon the human frame. Since his time, Aretæus, Celsus, Sydenham, Boerhaave, Ramazzini, Baglivi, and many others have closely considered the subject. In modern times, also, important researches have been made, exact meteorological observations being compared with statistics both of mortality and disease. Amongst the former class we must place Scoresby-Jackson's and Buchan's contributions to 'Nature' on mortality in relation to weather (August 5, 1875), and in France those of Lombard.

Owing to the uncertain interval of time between the commencement of a fatal disease and its termination, these comparisons of weather and mortality necessarily have less value than those in which atmospheric conditions are connected at frequent intervals with records of disease. Nevertheless, the

influence in question is so potent in the case of certain diseases that it overrides this disadvantage, and the effect of season upon mortality is plainly visible, especially in the case of such diseases as bronchitis, diarrhoea, measles, and whooping cough, perhaps also in scarlet fever and enteric fever.

It may be sufficient to note here that as the result of Lombard's observations, confirmed by others, a high general mortality usually accompanies a cold and dry season, with great variations of temperature, especially in the winter, with little rain and dry northerly winds, and strong electrical tension.

Scoresby-Jackson also points out the unhealthiness of a small rainfall and great ranges of barometric pressure. Buchan found that in English towns spring was the season of least mortality; in Scotland, autumn was the healthiest, and summer the next in order. Both in England and Scotland there was an excess of mortality in the winter months. The high mortality of English towns in the summer is shown to be chiefly due to the prevalence of diarrhoea.

These conclusions may be compared with the remark of Celsus with regard to Italy: 'Saluberrimum ver est, proximè deinde ab hoc hiems, periculosior aestas, autumnum longè periculosissimum.' It is probable that the latter result was due to the prevalence of malarial diseases in Italy in the autumn.¹

The chief returns of sickness that have been compared with meteorological observations are those made (1) daily by the Provincial Medical Association in the years 1855-56.

(2) Ten years of weekly returns made in 1860-70 to the Manchester and Salford Sanitary Association, and to Dr. Whitmore, St. Marylebone, London; and

(3) Similar returns made to Dr. Ballard in Islington from 1857-68.

The results of the more extended series of these observations were given in papers published by Mr. Vernon and the writer in 1860, and Dr. Ballard's conclusions are contained in an elaborate report presented to the Medical Officer of the Privy Council, and published by him with his report in 1868.

It may be briefly stated that the conclusions drawn from the mortality returns are in the main confirmed by the registers of sickness. It was possible, however, to take note of the more minute effects of vicissitudes of the atmospheric elements.

Thus in the case of respiratory diseases, it was observed not only that they are greatly affected by the mean temperature, but that a high degree of humidity tends to increase the frequency of these disorders.

It was noted in 1860² that a high mean temperature of above 60° for the week has a powerful influence in predisposing to diarrhoea, and this exact observation was confirmed by Dr. Ballard, and more recently by Dr. Tomkins, of Leicester.

Measles in both sets of observations is noticed to be increased by a fall of temperature, together with a low degree of humidity and a dry season. Dr. Ballard says it was arrested by a rise of temperature over 60°.

Scarlet fever is essentially an autumnal disease, and was shown by one set of tables to be mainly affected by fluctuations of the barometer, low pressure being favourable to the disease; and by Dr. Ballard it is noted that a fall of mean weekly temperature below 58° checks it.

¹ In a recent work, *Studies in Statistics* (Stanford), Dr. Longstaff has also compared the mortality from certain diseases with season, and has skilfully used the directions from the average of such mortality to trace the relationship of such diseases as scarlatina and diphtheria, puerperal fever and rheumatism, laryngitis and croup, and shows their relationship to the rainfall. The connection between what is registered as 'simple continued fever' and typhus is also shown.

² *Influence of Atmospheric Changes upon Disease*, p. 8. *Proceedings of the Lit. and Phil. Soc. of Manchester*, 1861.

Whooping cough again resembles measles in its tendency to select winter and spring for its epidemic outbreaks.

4. *The Influence of Occupation upon Mortality*

The death-rates of persons employed in various kinds of work vary so greatly, that it is important to ascertain the composition of any population before drawing conclusions from its gross rate of mortality.

The subject has been studied since the days of Ramazzini, whose treatise, '*De Morbis Artific.*,' published in the year 1700, is a classic of State medicine. In England, Thackrah, Arlidge, Greenhow, B. W. Richardson, and others have done valuable work respecting different classes of workpeople. In the decennial Supplements to the 25th and 85th Reports of the Registrar-General attempts were also made to estimate the comparative mortality of males engaged in different professions and trades, but the most complete account of the subject is that prepared by Dr. William Ogle for the Supplement to the 45th Report of this series.

The inquiry in this paper, as in the others, was limited to males over fifteen years of age; and the tables that are presented in the report are the more valuable from the care with which they were drawn up, and for the cautions as to their use that accompany them. It is thus pointed out (1) that figures that relate to small trades, or to age periods when the numbers are few, must be received with due hesitation; (2) that allowance must be made for the vagueness with which the occupation is oftentimes stated both in the returns made for the census and in the death-registers, especially the latter. In the case of the various forms of miners, for instance, it was found necessary to divide them geographically. (3) There are many trades and occupations which require a considerable standard of muscular strength and vigour to be maintained by those who follow them; and so soon as from any cause the health and strength of a man fall below this standard, he must necessarily give up the occupation, and either take to some lighter kind of labour or retire altogether from work. (4) Conversely, the weaker individuals, and those whose health is failing them, are thus being constantly drafted out of each industrial occupation, and especially out of those which require much vigour; hence the death-rates of these latter are unduly lowered as compared with those of persons engaged in lighter work. (5) The several industries do not start on equal terms as regards the vitality of those who follow them. 'A weakling will hardly adopt the trade of a blacksmith, a miner, or a railway navvy,' but will prefer that of a tailor, a weaver, or a shopman.

It is plain, then, that much caution must be used in drawing inferences from the death-rates of different industries, but they may still furnish valuable indications of this comparative salubrity.

The following table is selected from that given by Dr. Ogle, and in the supplement it is followed by others giving the causes of death under thirteen headings.

It will be seen that, of all the occupations, that of a clergyman enjoys the lowest death-rate, its mortality figure being only 556. Then come gardeners and agricultural labourers (599, 701 respectively). Schoolmasters, grocers, and some kinds of shopkeepers come next; then barristers, various out-door workpeople, such as carpenters, sawyers, &c., and some miners, Cornish miners excepted. Tailors, chemists, and printers overpass the standard of 1,000; medical practitioners, law clerks, and butchers from 1,100 to 1,300.

TABLE VIII.—*Death-rates of Males, 25 to 65 Years of Age, in different Occupations. (Ogle)*

Occupation	Mean annual death-rates per 1,000 living				Comparative mortality figures 1880-1-3
	1861-1. 1871		1880-1-3		
	Years of age		Years of age		Years of age
	25-45	45-65	25-45	45-65	
All males	11.27	23.98	10.16	25.27	1,000
Occupied males	—	—	9.71	24.63	967
Unoccupied males	—	—	32.48	36.20	2,182
Males in selected healthy districts	—	—	8.47	19.74	804
Clergyman, minister, &c.	5.96	17.31	4.64	15.93	556
Gardener	6.74	17.54	5.52	16.19	599
Farmer	7.66	17.82	6.09	16.53	631
Labourer (agricultural)	—	—	7.18	17.68	701
Schoolmaster	9.82	23.56	6.41	19.98	719
Grocer	9.49	17.15	8.00	19.16	771
Fisherman	11.26	15.84	8.32	19.74	797
Carpenter	9.44	21.36	7.77	21.74	820
Bookseller	10.84	21.36	8.58	20.57	825
Lawyer	9.87	22.97	7.54	23.18	842
Draper	14.34	26.33	9.70	20.96	883
Groom, coachman	—	—	8.58	23.28	887
Coal miners (6 districts)	—	—	7.64	25.11	891
Plasterer	9.50	27.90	7.79	25.07	896
Watch and clock maker	10.78	24.90	9.26	22.64	908
Tanner	10.43	26.57	7.91	25.37	911
Shoemaker	10.39	22.30	9.31	23.86	921
Artist, sculptor, &c.	11.78	22.91	8.39	25.07	921
Commercial traveller	12.28	29.00	9.04	25.03	948
Baker, confectioner	10.72	26.39	8.70	26.12	958
Mason, bricklayer	11.43	27.16	9.25	25.59	969
Blacksmith	10.07	23.83	9.29	25.67	973
Tobacconist	13.19	21.76	11.14	23.46	1,000
Chemist, druggist	13.92	23.56	10.58	25.16	1,015
Tailor	12.92	24.79	10.78	26.47	1,051
Printer	13.02	29.38	11.12	26.60	1,071
Woollen manufacturer	—	—	9.71	27.50	1,082
Cotton, linen do.	—	—	9.99	29.44	1,088
Physician, surgeon	13.81	24.55	11.57	23.03	1,122
Law clerk	13.75	37.05	10.77	30.79	1,151
Butcher	13.19	28.37	12.16	29.08	1,170
Glass manufacturer	13.19	29.32	11.21	31.71	1,190
Plumber, painter, &c.	12.43	34.66	11.07	32.49	1,202
Cutler (scissors, needles, &c.)	11.88	32.74	11.71	34.42	1,278
Carter, carrier	—	—	12.52	33.00	1,275
Bargeman, &c.	14.99	30.78	14.25	31.18	1,305
Musician	13.94	34.76	13.78	32.39	1,314
Hairdresser	15.11	30.10	13.64	33.25	1,327
Brewer	19.26	36.86	13.90	34.25	1,361
Cabman, omnibus	15.94	35.28	15.39	36.33	1,432
Chimney sweep	17.53	42.37	13.78	41.54	1,519
Innkeeper, publican	13.01	34.14	13.02	33.68	1,521
Messenger, porter, &c.	—	—	17.07	37.37	1,565
Filemaker	16.27	42.30	15.29	45.14	1,667
Earthenware manufacturer	12.59	41.75	13.70	51.39	1,742
Miner (Cornwall)	11.94	41.73	14.77	53.69	1,839
Hawker, street-seller	20.09	37.82	20.26	45.33	1,879
Labourer (London)	13.35	40.64	20.62	50.35	2,020
Inn, hotel servant	21.91	42.19	22.63	55.30	2,205

Then follow the more unhealthy occupations of plumbers, hairdressers, cabmen, and cutlers, over 1,800; chimney sweeps, innkeepers, and publicans, over 1,500; file makers, over 1,600; costermongers and hawkers, and Cornish miners, over 1,800; and lastly, general labourers (London) and hotel servants and the list at over 2,000—more than four times the rate of clergymen.

Practical Applications of Vital Statistics by Medical Officers of Health

As the population of a place is the standard to which most of the other statistics must be applied in order that they may be appropriately compared with those of other districts, it is important that as correct an estimate as possible should be made of their number. To this end the number of persons living at the middle of the year whose data are being examined should first be ascertained from the last two censuses by the method mentioned on p. 469.

This estimate should then be checked by one of the methods detailed on pp. 469–471, and if the results by those several plans are found to differ materially amongst themselves, the fact must be noted and the possible extent of error must be calculated and laid before the local authority. Subject to this possibility, the figures, may, however, be employed to ascertain the marriage-, birth-, and death-rates of the place.

It is further important that the numbers of the population at the Registrar-General's twelve groups of ages should be obtained, especially in the case of an urban district, in order to apply to these figures the general death-rate of the country at these ages, and then to deduce from them a 'standard death-rate' free from error on the score of differences in age and sex distribution.

Even when this has been done, however, the medical officer will do well to remember Dr. Farr's caution. He cannot conclude that the several figures obtained are strictly comparable until he has made sure that there are not other important circumstances to be taken into account, such as differences in the constitution of the population, the amount of domestic service, the presence of public institutions, the migrations of large sections of the population, especially of those who have to leave service when seized with illness.

When due weight has been given to these perturbing conditions, he will be in a position to appreciate the death-toll exacted from the community, by the varying densities of the population in different parts of his district, by their social condition, and by their different occupations.

The medical officer of health may then turn his attention to the records of the causes of death inscribed by the district registrars, and he will be expected to lay before his board at stated intervals, quarterly or annually, tables showing the incidence of these causes upon the different classes of people under his charge. He must give the amount and causes of infant mortality under one year and under five years of age, in the first case referring the deaths to 1,000 of the numbers born, and in the other to the numbers living under five years of age per 1,000.

The deaths of these children under one year of age and under five may also be given per 1,000 of the total deaths, as recommended by the Society of Medical Officers of Health in 1888; but this method is open to the objection given on p. 492. The causes of all the deaths registered should then be tabulated according to the classification of the Registrar-General, or a selection may be made of the most important of these causes.

It is usual, for instance, to give the number of deaths from each of the miasmatic diseases, and they are sometimes, though without advantage, grouped together and their rate per 1,000 is given under the title of zymotic death-rates (*see* pp. 485, 486).

Diarrhoea, cancer, scrofula, tabes mesenterica, phthisis, hydrocephalus, and diseases of the nervous, circulatory, respiratory, digestive, urinary, and generative systems are usually given separately, and in the supplement to the 45th Report of the Registrar-General, childbirth, suicide, other violence, and other causes are recorded in the tables.

Such an arrangement as this will greatly assist the medical officer of health in his estimate of the sanitary condition of his district ; but he will have also to take account of many other surrounding conditions, such as the weather, the price of provisions, the state of trade, &c., before he can decide as to the full weight of morbid agencies causing preventable sickness, and before he can draw comparisons with the average rates, or with the rates of similar districts.

If the notification of infectious sickness is not already in action in his district, the medical officer of health will endeavour to secure its adoption, and he will also avail himself of all other means at his disposal for becoming acquainted with the general condition of the inhabitants of his district.

MARINE HYGIENE

BY

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MARINE HYGIENE

THE conditions affecting man's life and health at sea contrast remarkably with the corresponding conditions operating on shore. The atmosphere by which he is surrounded, the dwelling he occupies, and the medium on which it is supported, the accommodation at his disposal, and the food he eats are, at sea, very different from similar necessities on shore. If on the ocean he is exposed to privations and dangers, from which on *terra firma* he is free, on the other hand life afloat has many hygienic advantages over life on land.

Much of the highly valuable literature relating to marine hygiene is scattered over Blue Books and medical periodicals in the form of detached articles. The treatises on the general subject are comparatively few in number, and many of them deal with the question from one aspect only. Most of these refer to the Royal Navy only, leaving the equally important mercantile navy entirely out of consideration.

As far as is practicable in the space available, an attempt will be made in the following pages to treat of 'Marine Hygiene,' under various heads. viz. :—

- (A) The sea.
- (B) The ship.
- (C) Crews, passengers, &c.
- (D) Cargoes in relation to hygiene.
- (E) Port sanitation and special measures for the prevention of disease.
- (F) Bibliography of marine hygiene.

(A) THE SEA

Extent.—Two-thirds of the surface of the earth is covered by sea. The average depth of the sea has been variously computed by astronomers to be from twenty-six to eleven miles.¹ Deep-sea soundings show, however, an average of about five miles. The proportion of land to water in the northern and southern hemispheres differs greatly, being in the ratio of 1,000 to 419 in the former, and 1,000 to 129 in the latter. This partly accounts for the lower mean temperature of the former. The distribution of the water into the great oceans (Atlantic, Pacific, and Arctic), the varying depths of these, and their peculiar currents, are among the principal causes of the modifications of climate in the same and different parallels.

¹ Maury.

Analyses.—The sea is not uniform in chemical composition. Average ocean water contains ¹—

	Grains
Water	962.0
Sodium chloride	27.1
Magnesium chloride	5.4
Potassium "	0.4
Magnesium bromide	0.1
" sulphate	1.2
Calcium "	0.8
" carbonate	0.1
Residuum	2.9
	<hr/> 1000.0

The residue consists of organised animal matters, traces of iron, alumina, silica, &c.

The mean of seventy-seven analyses of ocean water, collected on the voyage of the 'Challenger,' yielded the following results: ²—

<i>In 100 Parts of total Salts</i>	
Chlorine	55.420
Deduct basic oxygen equivalent to this chlorine	12.503
Muriatic acid	42.917
Sulphuric acid	6.415
Lime	1.692
Magnesia	6.214
Potash	1.333
Soda	41.433
	<hr/> 100.004

The following have been proved to be present in sea-water—viz. Bromine, iodine, fluorine (in boiler crusts), phosphorus, nitrogen, carbon, silicon, boron, silver, lead, copper, zinc, cobalt, manganese, iron, nickel, aluminium, barium, strontium, arsenic, lithium, cesium, rubidium, and gold. Many of these bodies have been found in the ashes of sea plants and in boiler crusts.

The different constituents vary more or less in quantity according to the locality from which the sample is taken. The principal constituent, chloride of sodium, shows a considerable range in different places, intertropical seas containing, according to Maury, 15 per cent. more of salt than those in Arctic Regions. The water of the Gulf Stream has 4.4 per cent., and that of the Bay of Biscay 8.5 per cent. of salts.

Salinity.—The lowest degree of salinity in parts of total salts per 1,000 of sea-water (88.01) was found in samples taken from the southern part of the Indian Ocean, south of 66° lat., and the highest (87.87), in samples from the middle of the North Atlantic ('Report of the "Challenger" Expedition').

The salts of the sea have important functions to perform. They prevent the putrefaction of the organic matter in the water. They assist the penetration of solar heat, which would otherwise act only on the surface. They retard evaporation. The ocean teems with organisms, microscopic and other, which, but for the brine they are preserved in, would after death decompose and rapidly render the sea unbearable. The indirect action of the salts on the sea is to create circulation. Currents such as the Gulf Stream,

¹ Maury.

² *Report of Voyage of H.M.S. 'Challenger,' 1873-76; Physics*, by Prof. Dittmar.

and under-currents—e.g. from the Mediterranean to the Atlantic, according to Maury, owe their main strength to this agency.

Specific Gravity.—The freezing point of sea water is 27·2° F. The trade and other winds affect the specific gravity considerably by sucking up the water as vapour, and leaving the salts behind.

The sp. gr. of the South Atlantic	1·02676
" " " North "	1·02664
" " " South Pacific	1·02658
" " " North "	1·02548 (Buchan)

Heavy rains diminish density, and high winds increase it.

Temperature.—The temperature of the ocean is higher than that of ordinary water in the same region. It varies with latitude and is influenced by currents. In some parts of the Red Sea it sometimes reaches 95° F. The Gulf Stream is, perhaps, the most remarkable instance of variation in temperature of a current from that of the surrounding ocean. Its waters do not mix with those through which it passes, and in a distance of a few feet its winter temperature is sometimes 20 or 30 degrees above theirs. Between limits of 50° N. and 50° S.,

The mean temperature of the North Atlantic is	.	.	71·6° F.
" " " South "	.	.	66·7° "
" " " North Pacific	.	.	69·9° "
" " " South "	.	.	67·7° " (Buchan)

The Atlantic is the coldest ocean, and the Indian the warmest.

The following ocean temperatures are taken from 'The Voyage of the "Challenger"' (Sir C. Wyville Thompson):—

Extract from Tables of Temperatuers observed

Depth in fathoms	February and March, 1873 Between Tenerife and Sombbrero Island				June and July 1873		
	Stat. No. 1	Stat. No. 5	Stat. No. 10	Stat. No. 20	Near Bermudas Stat. No. 59	Near Azores Stat. No. 73	Near Madeira Stat. No. 83
	Lat. 27° 24' N. Long. 16° 55' W.	Lat. 24° 30' N. Long. 24° 28' W.	Lat. 23° 10' N. Long. 38° 42' W.	Lat. 18° 58' N. Long. 59° 35' W.	Lat. 32° 54' N. Long. 63° 22' W.	Lat. 38° 30' N. Long. 31° 14' W.	Lat. 38° 46' N. Long. 19° 17' W.
	18°·0 C.	20°·0 C.	22°·2 C.	23°·9 C.	21°·1 C.	20°·6 C.	21°·5 C.
Surface	18°·0 C.	20°·0 C.	22°·2 C.	23°·9 C.	21°·1 C.	20°·6 C.	21°·5 C.
100	16·5	17·0	19·5	20·0	17·4	15·4	14·8
200	18·2	18·9	14·8	15·6	16·8	13·8	12·0
300	10·9	11·2	11·5	9·7	14·3	12·6	10·4
400	9·3	9·4	8·7	7·0	11·2	9·8	10·6
500	8·0	7·7	6·7	5·8	7·2	7·8	9·6
600	5·1	5·2	4·6	4·0	3·7	4·7	5·7
1,000	4·0	4·0	3·5	3·3	3·3	3·7	3·8
Depth at bottom	1,890	2,740	2,720	2,975	2,875	1,000	2,400
Temperature at bottom	2°·0 C.	2°·0 C.	1°·9 C.	1°·6 C.	1°·8 C.	3°·7 C.	1°·8 C.

Madeira to Brazil (July-Sept. 1873)				Between Bahia and Cape of Good Hope		
Depth in fathoms	Lat. 20° 58' N. Long. 23° 57' W.	Lat. 3° 8' N. Long. 14° 49' W.	Lat. 7° 39' S. Long. 34° 12' W.	Lat. 35° 25' S. Long. 23° 40' W.	Lat. 36° 22' S. Long. 8° 15' E.	Lat. 38° 35' S. Long. 16° 9' E.
Surface	23°·3 C.	25°·6 C.	25°·3 C.	14°·4 C.	18°·4 C.	18°·4 C.
100	16·7	13·8	16·8	12·9	13·3	10·7
200	13·3	10·4	8·6	10·0	10·4	7·7
300	10·0	5·3	5·0	6·4	6·7	4·9
400	7·2	4·7	3·7	4·3	4·3	3·1
500	6·8	3·8	3·7	3·6	3·3	2·9
800	5·1	3·3
1,000	3·3
Depth at bottom in fathoms	2,400	2,450	1,650	2,050	2,650	2,325
Temperature at bottom	1°·7 C.	1°·7 C.	2°·3 C.	1°·1 C.	1°·0 C.	0°·5 C.

Organisms.—The ocean abounds in organic matter, living and dead. The animalculæ and microscopic forms in sea-water have been the wonder and astonishment of all observers. The phosphorescence of the sea is due to swarms of molluscs and zoophytes. Who is not familiar with the coral from the tropics? or the barnacle and acorn shells which form wherever wood, &c., is exposed to sea-water for any considerable time?

Movements of the Sea.—The waters of the sea have, besides tides, three different and characteristic forms of movement—viz. currents, swell, and waves. Currents are either of the nature of rivers in the ocean, or they may be in the form of undercurrents. Currents when warm always flow from equator to pole, and when cold, from pole to equator; 'and this they do, not by the force of the winds, but in spite of them and by the force of those very agencies that make the winds blow. They flow thus by virtue of those efforts which the sea is continually making to restore that equilibrium to its waters, which heat and cold, the forces of evaporation, and the secretion of its inhabitants are everlastingly destroying' (Maury). Certain currents are the result of wind, rain, and barometric pressure, and alter according to the degree of each of these agencies. The most remarkable of all these currents is the Gulf Stream, which is constant. Flowing from the Gulf of Mexico to Labrador, Greenland, Spitzbergen, the North of Scotland, Norway, North Cape, Nova Zembla; and also, by bifurcation, to Ireland, France, Spain, Portugal, and the West Coast of Africa, this mighty volume of water, at a high temperature, plays a most important part in modifying the climate of the shores against which it strikes, rendering the air both warmer and moister. The causes of this great stream have not been satisfactorily explained. Modern investigations 'seem to encourage the opinion . . . that it is due mainly to the constant difference produced by temperature and saltiness in the specific gravity of water in certain parts of the ocean' (Maury). Sir John Herschel considers it the result of the trade winds.

The *swell* of the sea, that peculiar undulation which does not break the surface, is chiefly noteworthy as a cause of sea-sickness, the direct result of the action of the swell on the ship's movements.

Waves are the immediate effect of the action of the wind on the surface of the water, especially when operating against the tide. They are chiefly of interest from a hygienic point of view from the casualties they give rise

to, and from the moisture dispersed from them, either by the direct force of the wind, or by their own impetus, against the ship.

Influence of the Sea on Climate.—A mass of water so immense as the ocean, with its chemical constitution, specific gravity, temperature, and currents, &c., necessarily has great influence on climate. The tropical seas have been likened to a boiler, and those in colder regions to condensers, constantly at work, causing a perpetual flow of warm water polewards, and the evaporation of moisture, which on its way toward the Arctic Regions is gradually changed into cloud, and ultimately deposited as rain. The irregular distribution of land and water over the surface of the globe renders this rainfall very unequal, both as regards amount in relation to distance from the equator, and actual deposit on particular coasts or regions. The warm waters of the sea carried to colder regions raise the temperature of the air beneath which they pass, and by the vapour rising from their surface moisten and soften it. Thus the Gulf Stream makes the climate of Ireland and the North of Scotland milder in winter than in regions further south, and much more so than that of corresponding latitudes in America. The west winds blowing across this current are robbed of their asperity; but the humidity and fogs of the British Isles are less agreeable effects of the same cause.

The vapour rising from the surface of the sea is one of the causes of winds.

The salts of the sea, by increasing its density, aid in the convection of the sun's heat from its surface downwards. They also doubtless retard evaporation, and thus indirectly limit rainfall. They are said also to influence electricity, but little of this being evolved on the evaporation of fresh, as compared with salt, water at normal temperatures (Maury).

Sea Air.—Lévy found that the average amount in volume of oxygen in the air of the Atlantic, taken 400 leagues from the shore, was 21·019 per cent., that of air taken at Paris being 20·960. In the former case the amount of nitrogen was 78·94, and in the latter 79·19; and of carbonic acid ·080 per cent. in the former, and ·048 per cent. in the latter.

Marine air contains a considerable amount of saline matters, drawn from the sea. These are, of course, more abundant when the atmosphere is laden with spray, as during high winds.

The air of the ocean is more moist than that of the land, partly from direct evaporation, and partly also from the deliquescent nature of the saline matters it contains. This humidity is more uniformly diffused at sea than on land, where the varying elevation and general character of the surface (sand, rock, earth, foliage, mountain, plain, water, &c.) attract or repel moisture with greater or less force, according to circumstances.

The electric tension of sea air is generally greater than that of land air. There is also comparatively little or no pollution of the former—as e.g. by smoke, noxious effluvia, or human exhalations.

The comparative uniformity of sea level over the globe makes barometric pressure on the ocean more regular, *cæteris paribus*, than on land.

Extract from 'Voyage of the "Challenger"'

THE GULF STREAM.

Wet and Dry Bulb observations made in crossing and recrossing the Gulf Stream.

Date and Position	Temperature of air	
	Dry bulb	Wet bulb
	Deg. Cent.	Deg. Cent.
April 23, 1873. Off Bermudas.		
Noon . .	20.6	17.8
Midnight .	20.0	19.4
April 26. Lat. 32° 4' N., Long. 66° 33' W.		
Noon . .	23.1	20.8
Midnight .	20.6	19.2
April 29. Lat. 36° 5' N., Long. 69° 54' W.		
Noon . .	19.7	18.0
Midnight .	17.5	15.8
May 3. Lat. 38° 34' N., Long. 72° 10' W.		
Noon . .	8.9	8.9
Midnight .	5.8	5.0
May 6. Lat. 40° 17' N., Long. 66° 48' W.		
Noon . .	8.3	7.2
Midnight .	5.6	5.0
May 9. At Halifax.		
Noon . .	15.0	9.4
Midnight .	8.9	8.3
May 21. Lat. 42° 19' N., Long. 63° 30' W.		
Noon . .	9.4	8.3
Midnight .	8.9	7.9
May 25. Lat. 37° 9' N., Long. 62° 30' W.		
Noon . .	21.7	21.4
Midnight .	21.7	21.4
May 29. Off Bermudas.		
Noon . .	24.1	23.4
Midnight .	23.3	23.3

The temperature of sea air varies in different regions, as on land. The general effect of the sea is to modify and render more equable the temperature of the air above it. Thus, according to Humboldt, the maritime atmosphere of the tropics is 2° C. below that on the land (25.5° against 27.7°). The daily mean range of temperature, which near the equator is, on the continents, from 5° to 6° C., seldom exceeds 2° in marine air. Between the parallels of 25° and 50° N. latitude, the range is frequently beyond 15° on

land, whilst at sea it scarcely attains 8° (Boudin). It is stated that on clipper passenger steamships, during a passage to Australia of six or seven weeks' duration, the mean weekly range of temperature has only been 5° F., the highest range being 9·9°, and the lowest 1·6°.

The air from malarious coasts is often extremely fatal to the crews of ships moored to leeward and within a short distance from land.

Sea Life.—In contrasting the conditions of life at sea with those on land, distinction may be made between the various circumstances proper and peculiar to living on shipboard and those due to geographical and geological considerations, either as regards mere life at sea-level, or as regards life on land as compared with life on the water. On board ship the sailor is liable to the ailments due to increased hygrometric state and barometric pressure, such as may occur at low levels, either at sea or on shore. If these have their disadvantages, he has the compensation of being exempt from many of the dangers of living on land, such as, e.g., the risk of inhalation of sewer air. This advantage may, however, be but partial, inasmuch as the effluvia from the bilges and drains of a ship are often quite as offensive and injurious as those from ordinary house-drains. The effect of frequent exposure to cold and damp, so commonly the lot of the sailor, is to give rise to disorders of which rheumatism and bronchitis are the types. This disadvantage is not a necessity, but only an accompaniment of some phases of sea life, for it is claimed that on certain voyages, e.g. between England and Australia, the constant humidity of the atmosphere acts as a protection against chills. Rapid changes from one climate to another, whilst in many cases pleasurable and exciting, are often very trying to health.

Life on ship-board is one thing to the sailor and another to the passenger. With both it differs considerably in relation to the type of vessel, duration, route or season of voyage, &c., &c. Much of the advantage of such an existence is due to the tonic effect of the prolonged stay in the open air, rather than to the specific virtues of the marine atmosphere. The motion of the vessel through the air is itself highly stimulating and exhilarating, and especially so in the case of the swifter steamships.

Not the least remarkable feature of sea life is the constant motion of the vessel. This motion is of various kinds—viz. the direct progress of the ship through the water, rolling (or lateral rotation), pitching (or plunging), and (in the case of steamships), vibration.

The peculiar and specific effect of the combined action of rolling and pitching on the passenger is well known. The familiar gait of the seaman on land is the result of his habitual attempts to maintain his equilibrium on the deck of a rolling ship, the inclination of which will sometimes in rough weather amount to 20 degrees on each side (Wilson).

The effect of the mere vibration of steamships on the occupants, though doubtless considerable, is but little understood.

Pitching and rolling are greater in small ships than in large ones, and consequently sea-sickness is more common in the former than in the latter. The treatment most effectual in this disorder is to induce the sufferer to swallow some food or drink, to keep on deck near the middle part, and to lie flat on the back with the head low and a pad on the abdomen.

The size of ships has other important relations to hygiene than in the matter of sea-sickness only—but acting in the opposite way than in regard to that disease. It was the observation of Lind that 'small and well-aired ships are always the most healthy.' The explanation of this lies in the greater capability of ventilating such vessels than those of larger size.

The route of the voyage is most important from a hygienic point of view.

Some voyages are proverbially healthy, and others the reverse. Hygienically the voyage affects the voyager in regard to whether it is

Foreign or coasting,
Long or short,
Arctic, tropical, temperate, &c.

Each of the above is influenced—especially in regard of time—by the kind of vessel (sailing or steam). Service in many of the short-voyage steamers of Great Britain is attended with a special disadvantage to the health of the crews. This arises from a defect in the principle adopted in fixing the wage of the men. In the class of steamships known as 'weekly boats,' i.e. such as make short runs, it is the common practice to pay the seamen, firemen, &c., a certain sum per week and leave them to look after their own victuals. The result of this arrangement is that the men, through ignorance or negligence, frequently do not provide themselves with sufficient or proper food. Often the food taken by sailors on board such boats is kept in the forecables, there being no suitable store-room for it, and so becomes unwholesome.

The principal dangers to health in long voyages, from temperate to hot climates, arise from errors as to diet, drink, and clothing.

The necessity for reducing the amount of animal food and stimulants in southern voyages is insisted on by Lind, and most authorities since his time. Errors on this point cause many diseases. Inattention to clothing in passing to warmer latitudes is also to be credited with many evil consequences. The amount, material, and colour of the dresses are each hygienically important, by reason of the relation these bear to the absorption and convection of heat, moisture, &c.

The dangers to health arising from errors of diet or clothing on Arctic expeditions are of an opposite kind to those above named. Here the necessity for an increase of animal food rich in fats, and extra covering for the body to prevent the radiation of its heat, are among the prime necessities of existence.

On voyages of any considerable length—whether to high or low latitudes, but especially the former—one of the greatest wants is that of fresh vegetables, which are rarely to be procured at sea. The absence of these occasions the scourge of sea life—scurvy—of which a description will be found elsewhere. By means of preserved vegetables, such as are now to be had in greater variety and more palatable condition than in former times, owing to improved methods of preservation, together with the daily use of lime-juice as a substitute for such vegetables as cannot be so prepared, the evils of scurvy have been much reduced, and might, under proper care, be entirely removed. The disease, however, still makes its appearance in seamen's hospitals from time to time, and recent Polar expeditions have been attended with much suffering and loss through neglect on this score alone.

(B) THE SHIP

The term 'ship' is defined in the Merchant Shipping Acts as including 'every description of vessel used in navigation not propelled by oars.' On this, Boyd observes that 'the criterion as to whether a vessel falls under the category of ship, is whether the vessel be one whose real habitual business is to go to sea: if so, though propelled by oars as well as sails, it is a ship within the meaning of the Act. If she does not go to sea at all she is not a ship in this sense.' Such a definition as the above allows the word 'ship' to apply to a large variety of craft, of which the following is an account:—

KINDS OF SHIPS

Ships are classed in various ways, e.g. according to—

1. *Mode of propulsion* (sailing or steam). The different descriptions of sailing vessels are given below. Steamships are divided into paddle, screw, twin-screw, mail, cargo, passenger, &c.

2. *Rig*.¹—Sailing vessels are of various kinds, commonly classed according to their rig. Those in the merchant service comprise: *Full-rigged Ship*, viz.—A three-masted vessel, each mast fitted with topmast, topgallant mast, and royal mast, all being square-rigged (i.e. with yards and square sails). There are also *four-masted vessels*, having one or more (but not all) of their masts square-rigged, and the others with topmast only and gaffsails.

Barque.—A three-masted vessel, the two foremost masts being square-rigged as above, the hindmost, or mizzen, having only topmast with gaffsail. The barquentine resembles the foregoing in having three masts, one only of which (the foremast) is square-rigged.

Brig.—A vessel with two masts, both square-rigged. The brigantine has two masts, one only (foremast) being square-rigged, the aftermast carrying a mainsail (or boomsail) and topmast, and gaff topsail.

Topsail Schooner.—A two-masted vessel with long lower masts, the foremast having a loose square foresail, the aftermast having mainsail and topmast, &c., as in the brigantine. A three-masted topsail schooner has a foremast as in the foregoing, the two aftermasts having mainsails and gaff-topsails as above.

Common Schooner (fore and aft).—Two-masted, the lower masts being long, with short topmasts and no yards, and carrying mainsails and gaff-topsails only.

Chasse-Marée.—A small three-masted vessel with foremast, mainmast, and jiggermast, on all of which lug-sails are carried.

Luuger.—A large size of the above.

Sloop.—Having one mast with topmast and standing bowsprit, and carrying mainsail, gaff-topsail, stay-foresail and jibs.

Cutter.—One-masted, with running bowsprit, with sails, &c., as above.

To these may be added the *dandy*, a small vessel with one mast, and a jiggermast over the stern.

3. *Build* (decks).—Vessels are classed as one-, two-, or three-decked, according to number of decks.

Spar-decked.—When lighter than, but otherwise resembling, a three-decked vessel, and with generally two decks laid and caulked.

Awning-decked (or partially awning-decked).—Having a superstructure above the main deck.

Shelter-decked.—With exposed (or weather) decks, covered and closed in at top and sides.

Shade-decked.—With weather-deck not enclosed at sides above bulwarks.

Flush-decked.—Having a continuous upper deck, without poop, bridge-house, or forecastle.

Well-decked.—Having poops or raised quarter-deck, bridge, and topgallant-forecastle, the space between these forming the well (fig. 2).

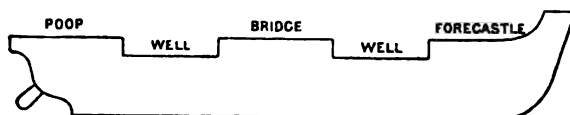


FIG. 2.

¹ Abbreviated from Captain Paasch's *From Keel to Truck*.

Ship with Hurricane-deck—Having a light platform over the erections on the upper deck, for promenade, &c.

Ships are also single- or double-bottomed, clincher- or carvel-built (i.e. with outside planks overlapping or flush-laid), clipper (with sharp fine lines), or bluff (full), &c., &c.

4. Ships are also classed according to the *purpose* they are intended to serve, e.g. :—

Trading ships.	Cattle ships.
Passenger ships.	Ore ships.
Emigrant ships.	Colliers.
Liners (large or small).	
Trawling and fishing vessels (steam, line, and net).	
Sea-going screw wherry, ¹ hopper, 'ketch,' 'shooter,' &c.	

5. *Trade* (route, &c.).—Foreign, coasting, East Indian, Australian, Mediterranean, North or South American, &c.

6. *River craft*, &c. : e.g. tug-boat, keel, dredger, wherry, passenger, ferry-boat, barge, canal-boat, towing-hopper.

7. *Royal Navy*.—The fleets of war vessels of England and other countries comprise :—

Battle-ships.	Cruisers.
Coast defence vessels.	Torpedo vessels and torpedo boats.

To which may be added transport ships.

With regard to the relation between the different classes of ships and their general salubrity, there is much to be said both in favour of and against many of the varieties above named, so that it is almost impossible to form a decided judgment. A comparison of the hygienic advantages of sailing ships as compared with steamers involves questions of size, speed, material, construction, &c., and not the mere matter of motive power which, *per se*, has little to do directly with sanitation.

Sailing ships are probably more liable than other vessels to accidents, such as loss of men overboard, from the nature of the duties to be performed. On the other hand, accidents connected with machinery, scalding, &c., the ailments resulting from working under high temperature, &c., are characteristic of steamships. Again, in steam vessels, especially those used for purposes of war, there is likely to be more unhealthiness from insufficiency of cubic space than in sailing ships, on account of the room taken up by the machinery and coal to work it. Steamships have, however, counterbalancing characteristics. The introduction of the screw propeller in place of the paddle gave a great impetus to naval warfare. De Méricourt states that in 1859 a flotilla of eighteen French and English vessels (thirteen screw steamers and five sailing ships) set out from Europe for China with 12,000 men. The results on the lives of the sailors and soldiers were as follow :—

—	No. of Ships	Total Equipment	Deaths	Rate of Mortality per 1,000
Sailing	5	3,765	45	11.9
Screw steamers . .	13	8,117	64	7.9
Total	18	11,882	109	9.3

¹ This differs from the river wherry in size (100 tons, &c.), in being decked over, and having a mast or masts for use in case of the engines breaking down, &c.

The lower rate of mortality in the screw steamers is attributed to the superior speed, and consequently quicker voyage, of these vessels, and to the greater number of calls they made at different ports; whereas the sailing ships anchored once only on their voyage, which was much longer.

The ships of the Royal Navy have, as compared with those of the merchant service, both advantages and disadvantages from a hygienic point of view. Though men-of-war's crews are in most respects exceedingly well cared for, the proportionately large number of men on board and the general construction of the ships almost imply crowding and defective ventilation. In trading ships the tendency, especially of late years, has been in the direction of undermanning, and attendant increase of cubic space per head. The fore-castles of such vessels are in general more easily, and perhaps better, ventilated than battle-ships. Again, cargo ships are not fairly comparable, say, with those in the passenger trade, in respect of sanitation; for cargoes are of all kinds, from the most innocuous, such as bricks, metal, coal, cotton, timber, grass, &c., to the most unhealthy, such as rotten plums or potatoes, manure, putrid hides or fish, infected rags, &c. On the other hand, passenger ships vary in equal degree, from the magnificent liner with ample accommodation and every possible luxury, to the dark, dirty, overcrowded emigrant vessel, with its prevailing characteristics of cheerlessness and discomfort.

Perhaps among the most insanitary of the larger vessels, fortunately not very numerous, are those carrying both emigrants and cattle. Such ships are often exceedingly offensive and unhealthy on account of the accumulation and decomposition of manure and urine from the quadrupeds, which there is usually no means of removing until after debarkation. Of the same character are certain kinds of transport ship for cavalry, such as described by Fonssagrives, in which the urine flowed down and mixed with the bilge-water, causing a most disagreeable stench.

The *rig* of ships has no relation beyond that of general size to hygienic character. *Build* has, on the contrary, a great deal to do with this. The fewer decks, the more airy and sweeter the vessel. Ventilation becomes more complex and more difficult in proportion to the compartments into which the ship is divided either longitudinally or transversely, vertically or horizontally. 'Well-decked' vessels, on account of liability in stormy weather to ship heavy seas, which lodge in the 'well' and flood the ship, are dangerous.

Among fresh-water craft the canal boat is the most worthy of attention as regards the health of those on board, for it is the house and home of a large population, and this often under circumstances most adverse to sanitation. Overcrowding, dirt, foul air, defective ventilation and disease, are frequent on such boats, and but for the system of inspection under which they are placed would be much more common than they are.

MATERIAL OF SHIPS

Ships are constituted of—(1) Wood; (2) iron; (3) steel; or they are (4) composite, i.e. composed partly of wood and partly of metal.

Wooden Ships.—Although wood has been until recent times almost the sole material of which the hulls of ships is made, few wooden ships are now being built. The large number of such vessels afloat, however, makes the consideration of this constructive material important from hygienic and other points of view.

Health.—It was an observation of Lind, that new-built ships were unwholesome. He says: 'The freshness of a ship's timbers is sometimes a general cause of sickness. A vapour constantly exhaling from the wood may

be felt and is often seen by candlelight in a well-illuminated ship. . . . It produces irregular fevers accompanied with a flux and dangerous symptoms.' Lévy refers to the notorious insalubrity of hastily-built ships of humid wood as a cause of scurvy among crews.

Wooden ships are warmer in cold climates, and cooler in hot ones than those of iron. In the latter a great deal of condensation of vapour goes on, causing dampness, as will be shown further on (p. 526). Wooden vessels have frequently become saturated with the infection, e.g., of yellow fever, and in consequence have had to be sunk in the sea for a length of time for purposes of purification.

Kinds of Wood employed in Shipbuilding.—The principal woods used are oak (British and Dantzic), teak, elm, and fir. Oak was first employed for this purpose by the Veneti (Cæsar, 'De Bello Gallico') lib. iii. cap. 13. The frames of ships are now generally constructed of it or of other hard-grained woods. Mr. Leonard Wray¹ speaks very highly of many of the woods of Honduras, viz. greenheart, live oak (Bignonia), mahoc, bullet tree, ironwood, locust, for ships' planking, &c.; also dogwood, red- and pitch-pines, cedar; the morra (for keels, timbers, and beams), and its branches for knees. The sap of the oak contains tannic and gallic acids, which act on iron rapidly, hence the use of iron nails and fastenings for that wood is objectionable. Teak and many of the Honduras woods are very valuable on account of their strength, resistance, and durability, partly due to their oily and resinous nature, by which they resist moisture and dry-rot better than oak. Teak, however, like other timbers, varies much in quality according to the soil on which it is grown. Elm decays rapidly when alternately wet and dry, but is very durable when kept constantly immersed in the water; hence it is chiefly used in making keels, &c.

The ancient Egyptians, whose sculptures and paintings show their knowledge of sawn planks of timber in boat-building, are said to have used acacia and black pine on account of their durability.

Cedar, pine, and other light woods, strongly clamped with iron or brass, were used by the Romans for building their *naves liburnæ*, or despatch ships.

Paasch² names as many as sixty-three varieties of woods employed in shipbuilding and the fittings of ships, cabins, &c.

Durability of Ship Timber.—This and strength determines the choice of woods in most cases. The kinds of timber lasting best are those containing most resin, gums, and oily matter. These resist the action of moisture and repel the ravages of worms. The circumstances affecting durability may be considered under the following head, viz. :—

Decay of Wood

When masses of timber lying in close contact become more or less moist, and are exposed to an elevated temperature without the free circulation of air, fermentation and decomposition soon set in. This is due to the action of bacteria and other fungi. Imperfect seasoning is one of the most common causes of *dry-rot*, a destructive fungoid disease of timber in houses on shore and in ships at sea. According to Lloyd's rules, ships of the best and most thoroughly seasoned timber, and built in the open air, are classed for twelve years only. If built under cover they may be classed for an additional year, showing the importance attached to the exclusion of external moisture. Un-

¹ Quoted in *Brit. Encycl.*, 1860.

² *From Keel to Truck.*

seasoned oak is especially liable to dry-rot. The age of wood and the season of the year when it was felled have each an important bearing on its preservation; young timber containing less mineral matter (lignin) in its cells than old, and summer-felled trees being more sappy than those cut down in winter. The parts of the ship most liable to decay from wood-rot are the internal, and from the causes above named. Of the external parts, those alternately exposed to air and water suffer most in this respect, those constantly submerged being least affected. The ravages of dry-rot under favourable conditions (moisture, absence of ventilation, and a certain degree of warmth) are such as few without actual experience would credit.

The writer has known timbers a foot in thickness in the floors of a house completely eaten through, and made into touchwood by it in the course of two years. It appears that this alteration of wood does not result from the development of various species of fungus. Thus in the ship 'Queen Charlotte,' Knowles observed the *Boletus hybridus*, *B. medulla panis*, *B. lachrymans*, *Xylostroma giganteum*, and *Auricularia pulverulenta*.

Wood is also liable to destruction by animal parasites, among which Fonssagrives names the *Lymexylon navale* (Coleoptera), the *Lymnoria terebrans* (Crustacea, order Isopoda), *Lymnoria xylophaga*, *Teredo navalis*, and others.

Iron nails and fastenings rust, and thus injure timber, especially oak.

Preservation of Wood

Various means have been adopted at different periods for the preservation of wood. The following are given in the 'Encyclopædia Britannica' and elsewhere:—

(1) The first, or natural mode, is that of weather seasoning, which requires two years or more to complete the evaporation of the sap, which is the principal medium of decomposition. Immersion timber for masts, &c., is submerged in sea-water or mud for protection. Wood so preserved is not liable to crack.

(2) Charring in a sandbath is said to have been in vogue at the beginning of this century ('Encycl. Britannica').

(3) Desiccation in a stove, by current of dry hot air, and exposure to the fumes of sawdust or tar (Guibert). A modification of this is the method of Gavini, who proposed to withdraw the sap by an air-pump, and to inject under high pressure (Fonssagrives). Desiccation in a current of air at a temperature of from 115° to 120° F. is employed to preserve other woods than English, which become fissured at a heat of above 105°. Desiccated wood is said to be hygrometric.

(4) Steaming and pickling (Machonochie's method). This consists of (a) steaming the wood in a closed tank; (b) condensing the steam so as to form a partial vacuum, to remove the natural juices; (c) submerging the timber in oil from teak, &c.

(5) Injection of solutions of metallic salts (a) perchloride of mercury (Kyan's process, or Kyanizing); (b) chloride of zinc (Burnett's process). Various other salts have been used, but none with success.

(6) Creosoting. After abstracting the air and sap *in vacuo*, creosote under pressure of 150 lb. to the square inch is forced into the wood, and pressure kept up for forty-eight hours or more. This method is chiefly employed for the protection of pine. Yellow pine should absorb 11 lb. of creosote per cubic foot. The process prevents decay and the development of worms.

(7) Carbonization of the internal surface of the ship's timbers, e.g. by means of the flame of ordinary coal-gas, after brushing with tar to render the wood more inflammable.

(8) Dr. Boucherie's process (applicable to straight-grained, porous woods). This consists of forcing out the sap by a pressure of 15–20 lb. to the square inch, by means of a raised tank containing a solution of sulphate of copper. The pressure is applied at one end of the block of wood to be operated on. The natural juices are replaced by the copper solution.

Strength of Timber

Timber is valued according to its cohesive strength, its resistance to pressure, and its transverse strength.

The cohesive strength of different woods per square inch in lbs. is stated by Barlow¹ to be as under :—

	Lbs.
Ash	17,000
Teak	15,000
Fir	12,000
Beech	11,500
Oak	10,000
Pear	9,800
Mahogany	8,000

Professor Hodgkinson places the following woods in inverse order of resistance to pressure :—Yellow pine, cedar, red deal, birch, sycamore, Spanish mahogany, ash, dry English oak, box. Unseasoned timber has less resistance than seasoned.

Barlow gives the transverse strength of the various woods in the following order :—Teak, English oak, Canadian oak, Dantzic oak, Adriatic oak, ash, beech, elm, pitch-pine, red-pine, New England fir, Riga fir, Mar Forest fir, larch.

Iron, Steel, and Composite Ships.—At present almost all ships are being built of iron, steel, or a combination of those with wood. Such vessels are in general of much larger size than those of wood. The masts and standing rigging, and occasionally even the yards, are of iron, as is often the house amidships and sometimes the boats. Metal as a material for ships has hygienically both advantages and disadvantages in comparison with wood. Thus in tropical latitudes the decks, cabins, engine-rooms, fore-castles, &c., of an iron ship, especially the first of these, acquire a high temperature, which, from the angle at which the sun's rays fall on the metal, together with the fact that the dark unpainted surface greatly promotes absorption of the heat, becomes often insufferably high. In cold weather the reverse is experienced. Another evil of iron ships is the condensation of aqueous vapour, or 'sweating' as it is called, in fore-castles, cabins, &c. This, where a number of men are housed together, makes the quarters constantly wet, and unhealthy. It is obviated by lining the decks, plates, or stringers with wood, or covering the surface with paint or varnish, and whilst this is still moist, applying a thick coat of coarsely granular cork.

On the other hand, iron vessels are free from the danger of saturation to which the older wooden ships were so liable. Paint, which is necessary to prevent iron from rusting, is, *ceteris paribus*, sanitary. White paint (zinc or silicate) is preferable to that in other colours, both as regards its appearance and its reflection of the rays of heat.

OTHER METALS, ETC., USED IN SHIPBUILDING

Copper is used for ships' sheathing below water-line, as a protection against sea water and the ravages of marine animals. It is used alone for pipes, funnels, &c., or, in the form of brass, for handles, locks, casings, and numerous other things.

¹ *Strength and Stress of Timber.*

Lead appears to have been used as a sheathing for ships in the time of the Romans. A ship of Trajan's, raised after many centuries of submersion, was found to have such a sheathing fixed with copper nails. It is used for certain purposes on shipboard at present, e.g. as a protection for steps and for piping, &c. In French steamships lead was formerly employed largely in the form of pipes to convey water for drinking purposes, which, together with the free use of red and white lead in the machinery, &c. gave rise to a very large amount of seriously fatal lead colic among the crews, and especially the firemen and engineers. The disease remained unrecognised for a considerable length of time. At length the discovery of its true nature and source led to the stoppage of the evil. The reader will find a description of this 'colique végétale ou colique sèche' in the 'Traité de Géographie et de Statistique Médicales' of Boudin, who did not distinguish the disease from ordinary lead poisoning, and in the works of Fonssagrives and De Méricourt, who did. The latter shows that much diversity of view existed as to this disease, and that under different theories, all erroneous, a multiplicity of names were given to it. Many years appear to have elapsed before M. Lefevre directed attention to the state of the patient's gums, which showed the blue line, and established the character of the complaint. Instituting special inquiries, M. Lefevre discovered, in the pipes for conveying drinking water from the tanks, in the tubes for the distilled water from the condensing apparatus, and in various other appliances, ample explanation of 'the apparently capricious occurrence of cases of dry colic on board French men-of-war, whilst ships of other nations enjoyed a perfect immunity.'

Among other material entering into the construction or used for the protection of ships, may be mentioned canvas, hemp (in sails, rope, &c.), tar, limewash, grease, &c. The two former require merely a passing reference here from their being frequently stored in the forecastles of schooners, &c., and thus encroaching on the already limited crew space. Tar and limewash are among the most hygienic, whilst grease—from its smell and share in the constitution of bilge-water—is one of the most unhygienic matters on shipboard.

Construction.—The skeleton or 'framing' of a wooden ship consists principally of the following parts:—(1) The keel, running the entire length of the bottom of the ship, and corresponding to the backbone of a vertebrate animal. The keel in front terminates in the (2) stem or foremost part of the ship, with which it forms an angle of from 90 to 100 degrees or thereabouts; and behind in (3) the sternpost or aftermost part of the ship proper, to which is attached the rudder. To the keel at regular intervals throughout its entire length, and curving outwards not unlike the ribs from the human spine, are (4) the timbers. Those in the bow or front part of the ship are termed cant timbers; those aft of the cant timbers are the frames. The sternpost supports (5) the stern, which curves underneath as the counter. The planking (6) is fixed external to the frames, and runs horizontally the entire length of the ship; the main rail (7) fixes in position the upper ends of the frames. The curved part of the outside and bottom of the ship, below the water-line is the bilge.¹ (8) The highest part of the side, above the level of the upper deck, is the bulwark (9), perforated by mooring pipes. The upper part of the planking near the stem is perforated on each side by a hawsepipe, through which passes the anchor chain.

The framing is lined inside with planks. This and the planking on the

¹ The term 'bilge' (derived from 'bulge') is applied by shipbuilders to this part, and by sailors to the cavity in which offensive liquid, known as bilge-water, collects.

outside of the frames are called the 'skins,' the whole forming the wall of the ship. Between the skins in the intervals of the frames is a space in the wall, closed in at the top by the covering board, and extending downward to the bottom of the vessel, where it ends in the limber or bilge, a longitudinal channel parallel to, and one on each side of, the keelson—a kind of inner or upper keel.

A mid-ship section of a wooden three-decked vessel is shown diagrammatically in fig. 8. The parts of hygienic interest in connection with it are (1) the *decks* (upper, main, and lower, in a merchant vessel, and spar, gun, and orlop, in a man-of-war), each resting on beams extending transversely from one side to the other, and supported on shelf-pieces or projections from the inner skin; these beams are planked on the upper surface, which is slightly curved, especially on the upper deck, so as to throw off water to the side, where it comes in contact with the waterway, a projection from the inner planking *above* deck, corresponding in position, &c., to the shelf-beam *below* deck; (2) the *hold*, or principal receptacle, situated below the lower deck; (3) the limbers or bilges are shown in section 1 on each side of the main keelson, with covering or limber board, and forming by means of water-courses through the floor timbers, a continuous longitudinal canal; (4) the ventilating channel (longitudinal) in the inner skin below each deck may, as shown in the diagram, serve either for an outlet from the 'tween decks or an inlet for foul air from the bilge.

From the foregoing description it will be seen that water falling on the upper deck (sea, rain, washings, &c.) will flow to the waterways, where it is discharged overboard by means of the scuppers or outlets through the bulwarks. Liquids and drainage from cargoes falling on the lower decks and floor of the hold, pass into the bilge. Leakage from the sea through the seams of the outer planking finds its way between the skins into the bilge, and thence to the well or sink, where the pumps are placed to discharge it overboard. In steamships the water on the floor of the engine-room and drippings of oil from machinery, &c., flow to the bilges.

Next to the bilges, perhaps, the most insanitary parts of the ship are the (5) *peaks* (fore and after), or spaces right forward and right aft. These are separated from the hold, &c., by bulkheads. The peaks are frequently in a foul condition, and in the case of small ships they affect the air of the lower forecastles, where the crew are housed.

Forecastles are of two kinds, according to situation. The upper or top-gallant-forecastle is placed upon the upper deck, right forward; it has side-lights, and is entered by a doorway. Lower forecastles are found only on schooners and small vessels. They are below deck, and are entered by a hatch or opening in the deck, measuring, perhaps, $2\frac{1}{2}$ feet square, sometimes covered with a scuttle, but often left open as the chief means of light and ventilation. Leading from the hatch into this forecastle is a ladder or flight of steep steps.¹ Lower forecastles, as crew-spaces, are, usually speaking, very unhealthy.

Much superior to forecastles as crew-spaces are the *deck-houses*. These are found on most Norwegian and Danish merchantmen—even small schooners. They are also met with on many high-class ships, British and foreign. Deck-houses are placed amidships. They have the sanitary advantages of light and air, which lower forecastles do not possess, and which even top-gallant-forecastles have imperfectly. They are easy of access,

¹ Owing to the small size of the hatch, and the nature of these ladders or steps (which often consist only of a few cross-pieces of wood nailed to the bulkhead), the removal of sick sailors is difficult, and (in case of enteric fever) dangerous.

and allow of more convenience and comfort for the men. It is difficult to understand why they are not more general in the merchant service. The testimony, both of masters and crews of vessels, in their favour, appears to be unanimous. The masters of vessels provided with them state that they do not interfere with navigation. The space occupied by crews in forecastles would be much more advantageously used for storage.

In steam vessels the engine-room and boilers are usually placed amidships, extending downwards to the floor, and separated from the hold, 'tween

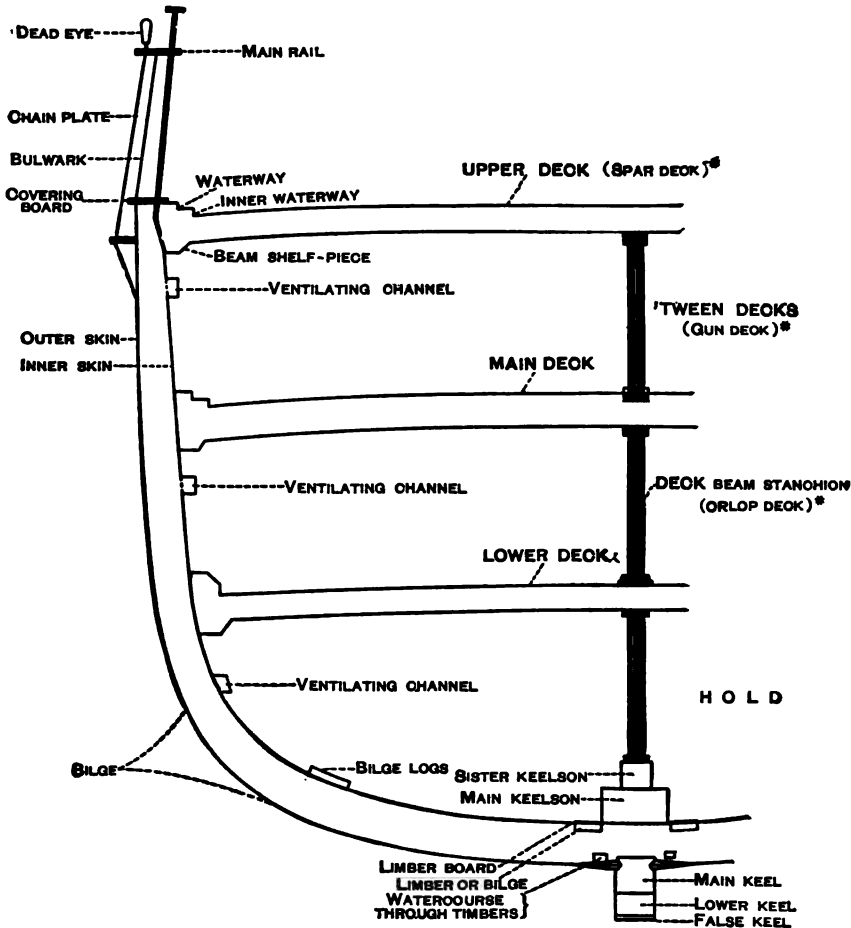


FIG. 3.—Midship Section of Wooden Ship.

* Names of Decks of a Warship.

decks, &c., by bulkheads, thus preventing the free circulation of air from end to end below deck.

An important part of such ships—from a sanitary aspect—is the *stoke-holes*. These are the places from which the furnaces are coaled. They are situated at the bottom of the ship, deeply below the water. In the smaller class of vessels there is only one stokehole to each steamer, but large ships have two or more. They are generally provided with wide cowed shafts, for the delivery of cold fresh air from the upper deck, the hot and consequently light air being allowed to escape upwards. For economy of space, the stokehole is usually made no wider than is absolutely necessary—say 8 feet—

and the width and depth being those of the ship. The stokers, therefore, work in a deep well, and are exposed to great heat from the furnaces, from which they are unable to withdraw whilst on duty. The air at the bottom of the stokeholes tends to become very foul by reason of the escape of the various gases of combustion from the fires. These gases, from their greater density, are not carried upwards with the warmed air, and the supply of fresh air by the cowed shafts before mentioned does not counteract their evil effects. In some ships of war (torpedo-boats, &c.), where the stokeholes are much enclosed, fresh air is of necessity supplied by special apparatus, such as fan-blasts, &c. The effect of the stokehole on the stoker is detrimental to a high degree. Firemen are, proverbially, unhealthy. The peculiar ailments from which they suffer will be described under the proper section (Health, Disease, &c., p. 574).

Iron ships have keel, stem, stern and sternpost, frames and beams, &c., as in wooden ships, but all are made of metal. The planks of the latter are in the former replaced by iron plates; the decks and transverse partitions (bulkheads) dividing the vessel into compartments also consist of iron plates. The longitudinal bars inside the frames (stringers) have plates (stringer plates) riveted to them, forming the inner skin. Iron masts, being hollow, are often used as ventilators. Boats also are sometimes made of the same material. They are said to be very buoyant, even more so than those of wood.

Iron ships are frequently provided with cellular double bottoms or water ballast tanks; or the double bottom may be placed above the ordinary floor of the vessel. These are great safeguards against accident. The need of them is apparent in war ships, where a shothole below water line would quickly sink a vessel. Moreover, the bottom of any iron vessel may accidentally be pierced by an unseen agent, with similar result. A danger in connection with these double bottoms has been pointed out by Macdonald,¹ who says that the constant chipping and scraping off of old paint and putting on of new is trying to the health of the men engaged on this duty. The paint is commonly formed of lead as a basis, and gives off lead vapour. In addition to this the space of the cells is confined and unhealthy to work in, on account of the small size of the manholes and the want of ventilation.² It is customary to pump fresh air down to the workmen and to supply acidulated drinks whilst the scraping is going on. In the Queen's Regulations and Admiralty Instructions, 1887, p. 465, precautions are laid down to prevent accident from this cause, including the use of the air-fan before and during the progress of the work; the testing of the air in the cell by candle-light; the keeping up of communication between the men in the inner compartment and those in the outer air, &c.

In vessels of war the construction is arranged to subserve to this special purpose. The upper deck has the top-gallant-forecastle right forward as in a merchant ship, but used for guns or for purposes other than housing the men. At the after part of this deck is the quarter-deck, with or without a poop-deck over part of it. Below the upper deck is the gun-deck, used also as sleeping quarters for the crew, and having officers' accommodation aft. Next comes the orlop-deck, in which certain stores are kept, and beneath this the hold. The orlop is separated from the sides of the ship by narrow passages (wings), which serve as means of access for repair during action, &c., and also to keep the stores from the side of the vessel. The hold is the general magazine for ammunition, spirit, water, provisions, &c. The orlop-deck contains the cockpit or surgery, and some accommodation

¹ *Naval Hygiene*.

² The special ailments to which these workmen are liable will be found at p. 579.

for officers. The ventilation of this deck, as from its position below water-line may be imagined, is defective. That of the hold is still more so. This part of the ship is not used for crew space.

Passenger ships are, by the Passenger Acts, provided with 'space properly divided off . . . to be used exclusively as a hospital or hospitals for the passengers. This space shall be under the poop, or in the round-house, or in any deck-house . . . properly built and secured . . . or on the upper passenger deck, and not elsewhere, and shall in no case be less than eighteen clear superficial feet for every fifty passengers.'¹ Between emigrant vessels and passenger ships of the highest class, there is a wide range in the extent and character of the accommodation provided for those on board. In the former there is often but little convenience and no comfort. The latter are constructed and fitted up to administer to the wants and fancies of the most exacting and capricious tastes. Electric lighting, arrangements for carrying off the smoke, smell, and heat from the engine-rooms above the passengers' heads, elaborate refrigerators for preserving fresh provisions, spacious apartments, punkahs, promenades, &c., combine to render a sojourn in one of these 'floating hotels' exceedingly agreeable, and as little like a sea voyage as it is possible to imagine. Some of the 'liners' are built of steel, and are 450 or 500 feet in length, with 50 or more feet of beam, and upwards of 6,000 registered tonnage. By means of half-a-dozen boilers and triple expansion engines, 8,000 horse-power and a speed of more than 400 knots in the twenty-four hours is attained. They are fitted with double bottoms in separate watertight compartments, and there is usually an arrangement for dividing the upper part of the vessel into several parts by means of sliding doors, &c., as a protection against fire.

STATISTICS OF SHIPPING

The following figures show respectively the particulars of vessels in the Royal Navy of England, the merchant navies of different countries in trade with the United Kingdom, and the fishing boats of the United Kingdom, canal boats, &c. The statistics of the crews, passengers, &c., will be found under the proper heading (pp. 581, &c.).

Royal Navy.—An Admiralty² return showing the fleets of different countries up to April 1888, gives the following information as regards England :—

(a) Battle-ships, 49, ranging from 4,010 to 11,940 tons displacement, of which seven of the largest were completing or building.

(b) Coast defence vessels, 18, ranging from 1,230 to 4,910 tons displacement.

(c) Cruisers (completed), armoured 6, ranging from 5,390 to 8,400 tons displacement; unarmoured, 58, ranging from 1,420 to 5,780 tons displacement; (completing and building), armoured, 6, each 5,600 tons displacement; unarmoured, 22, ranging from 1,580 to 9,000 tons displacement.

The unarmoured cruisers of the largest size are estimated to attain a speed of 22 knots.

(d) Armoured torpedo ram, 2,600 tons displacement.

(e) Torpedo vessels (completed or uncompleted), 14, from 245 to 735 tons displacement, and having a speed of from 19 to 21 knots; torpedo storeships, 2; torpedo-boats (built, and to be built), 86 first-class, speed from 15.5 to 23 knots; 61 second-class; wood (completing), 12.

¹ Board of Trade 'Notice to Passengers,' 1887. Clause xxiv.

² Navies (England and other countries).

Italy has the largest number of battle-ships (21), of which no less than 18 range from 11,000 to 18,890 tons displacement. Among other vessels of war, that country possesses, according to the return in question, 95 first-class torpedo-boats, of a speed from 20 to 22·5 knots.

Merchant Shipping.—Year 1891, vessels actually employed (classed according to tonnage) in the home and foreign trade :¹—

	Sailing		Steam	
	Vessels	Tons	Vessels	Tons
Under 50 tons . . .	8,940	188,862	847	19,730
Of 50 and under 100 tons	4,007	286,255	492	33,616
" 100 " 200 "	1,087	157,592	333	49,216
" 200 " 300 "	268	65,561	230	57,780
" 300 " 400 "	124	42,960	230	80,347
" 400 " 500 "	96	44,691	275	123,768
" 500 " 600 "	88	44,448	240	131,637
" 600 " 700 "	88	57,790	229	149,390
" 700 " 800 "	113	85,852	257	192,414
" 800 " 1,000 "	139	169,005	522	468,354
" 1,000 " 1,200 "	232	257,725	567	621,477
" 1,200 " 1,500 "	362	485,227	761	1,028,337
" 1,500 " 2,000 "	858	609,419	667	1,141,835
" 2,000 " 2,500 "	143	314,229	266	589,119
" 2,500 " 3,000 "	29	79,472	139	379,038
" 3,000 tons and above .	8	9,418	74	250,998
Total . . .	11,114	2,847,501	6,129	5,317,040

MERCHANT SHIPPING²

Extracts from Board of Trade Returns to the House of Commons

United Kingdom (foreign trade), tonnage of sailing and steam vessels with cargoes and in ballast, entered and cleared at ports in the United Kingdom in 1891 :—

	Tons	Including Steam (Tons)
British ships . . .	53,957,485	49,371,398
Foreign ships . . .	20,855,185	13,593,686
Total . . .	74,812,620	62,965,084

Principal Maritime Countries (foreign trade).—Tonnage of sailing and steam vessels entered and cleared in the principal maritime countries of Europe and the United States, with cargoes and in ballast, in 1890 :—

Countries	Tons
United Kingdom	53,973,112
Russia (in Europe)	958,258
Norway	3,468,397
Sweden	8,624,301
Denmark	4,693,308
Germany	9,275,710
Holland	3,122,125
France	9,254,879
Portugal	746,175
Spain	10,473,542
Italy	3,473,148
United States	8,149,878

¹ Extracted from the Annual Statement of Navigation and Shipping of the United Kingdom for the year 1891. By the Board of Trade.

² Tables showing progress of British merchant shipping. Board of Trade return to the Order of the House of Commons, June 1892.

Principal Maritime Countries (Merchant Navies)

Countries	Tonnage in the year 1890 Tons
British Empire (including United Kingdom)	9,688,088
United Kingdom	7,945,071
Russia	—
Finland	—
Norway	1,705,699
Sweden	510,947
Denmark	302,194
Hamburg	588,229
Bremen	378,088
Total German Empire	1,433,418
Holland	255,711
Belgium	75,946
France	944,013
Italy	820,716
Austro-Hungarian Empire	204,214
Greece	271,886
United States, registered for over-sea (foreign trade)	946,695
United States, enrolled and licensed, including lake and river steamers of home trade . . .	8,477,802

Boats Registered under the Sea Fisheries Act, 1868.¹

Boats	England		Scotland		Ireland		Total United Kingdom	
	No.	Tons	No.	Tons	No.	Tons	No.	Tons
<i>First Class.</i> 15 tons and upwards . . .	3,878	183,421	3,705	77,533	441	11,742	8,019	272,696
<i>Second Class.</i> (Less than 15 tons navigated otherwise than by oars only). Tonnage known	4,055	23,848	6,582	26,228	2,899	9,744	13,536	50,820
„ unknown	20	—	—	—	—	—	20	—
<i>Third Class.</i> (Navigated by oars only). Tonnage known	115	246	1,245	2,535	3,658	6,277	5,018	9,058
„ unknown	—	—	—	—	—	—	—	—
Total { Boats, the tonnage of which is known . . . Boats, the tonnage of which is unknown . . .	8,043	207,515	11,532	106,296	6,998	27,763	26,573	341,574
	20	—	—	—	—	—	20	—
Total . . .	8,063	—	11,532	—	6,998	27,763	26,593	—

N.B.—Undecked fishing boats, not going outside the distance of three miles from low-water mark, or in the bays ten miles wide, are exempted from registration. This exemption does not apply to Scotland or Ireland.

¹ From *Annual Statement (Board of Trade) Navigation and Shipping of the United Kingdom, 1891.*

In addition to the above, there are in the Isle of Man and Channel Islands 686 boats.

Canal Boats, Barges, &c.—From the report of Mr. Brydone, Inspector under the Canal Boats Acts, it appears that 'the number of boats on the registers is much larger than that of the boats actually employed, and it may be estimated that while the aggregate returns of the various authorities show 11,827 boats on the registers in 1891 as against 11,487 in 1890, 10,000 will fully represent the total number of boats used as dwellings on the various canals and other waterways of the country.'¹ The number of barges in this country cannot be given even approximately. According to Collingridge, no less than 9,500 barges are employed in the Port of London alone. About 500 canal boats ply on the river in addition to these.

AIR AND VENTILATION

The *air* of ships, though fresh and sweet above deck, is very liable to be foul below, and that from several causes. Bilge-water is the essence of nastiness, and its effluvia one of the most serious nuisances on board. The bilge is the sink of the ship, and the exhalations from it are much more offensive and dangerous than the 'ground-air' of a house to which Blyth compares it. It is, in fact, more like sewer air. The perpetual motion of the ship, although available for purposes of ventilation, only tends to increase the evolution of gases from below by the constant agitation of the bilge-water. The sea-water in the bilges concentrates, and, under the action of organic matters of various kinds mixed with it, becomes decomposed, giving off sulphide of ammonium, sulphuretted hydrogen, and other gaseous vapours and compounds, which are diffused through the ship. Fonssagrives² quotes from Duhamel du Monceau a remarkable instance of the evil effects of inhaling the vapour from a cask of putrid sea-water. A sailor who unbunged the cask dropped dead; six of his comrades, at some distance, fell down in convulsions and lost consciousness, as did the ship's surgeon. Blood was discharged from the mouth, nose, and ears of the corpse, which decomposed rapidly. One of the most abominable of all the many stench of bilge-water, as testified by various observers, is that resulting from the drippings of a sugar cargo into the bilges, as sugar cargo is liable to heat and give off offensive effluvia (*see* 'Cargoes,' p. 581).

Another cause of foulness of the air of ships is the cargo itself, which may consist of guano, manure, fish, cattle, &c. One of the most noxious articles of cargo in the experience of the writer is compressed fuel, the vapour from which permeates the small vessels laden with it. Gas tar, paraffin, &c., from leaking casks in cargoes, also make their presence disagreeably felt. In cattle steamers the accumulation of dung, &c., pollutes the air of ships very greatly.

Frequently the evil effects of cargoes on the air of ships would be much less than they are, were it not from defect in the structure of the vessel and want of proper ventilation. Thus the writer has perceived the smell of artificial manure from the hold coming through defective seams in the floor of the lower fore-castle of a schooner; and a similar nuisance from open bulk-head between hold and fore-castle in a brigantine. Paint, paraffin, and other

¹ *Annual Report of the Local Government Board, 1891-92.*

² *Traité d'Hygiène Navale.*

offensive stores are often kept near the forecabin, and sometimes within it, even in ships of considerable size. The 'convenience' (water-closet or privy) is generally much too near the entrance to the sailors' quarters. Occasionally it abuts upon them and, from defective bulkhead, is offensive to the men in their berths.

The air of forecabin is also rendered impure by the respiration of the occupants, the combustion of fuel, candles or oil, mould, mustiness, dirt, &c. Instances are occasionally met with of serious accidents from the use of charcoal for fuel without proper means for access of fresh air and exit for the escape of the fumes.

In the year 1886, the Indian s.s. 'Nederlands en Oranje,' from Bombay *via* Odessa, with seventy-three hands (Indian crew), came to the Tyne for repairs. Three of the men had died at sea from inhaling the fumes of charcoal burnt in their forecabin during cold weather. Four died whilst the ship remained in the Tyne Port (from uræmia and dropsy, the result of paralysis of the kidneys), and several others suffered more or less severely.

The Board of Trade 'Instructions as to Survey'¹ provide under Section 54, 'that every place occupied by seamen shall, as far as practicable, be shut off and protected from effluvia which may be caused by cargo or bilgewater; the surveyor must, therefore, see that the bulkheads, sides, and decks of the crew spaces are so fitted, laid, and caulked, and are of such thickness that this provision is complied with, &c.'

In passenger ships, under the Act of 1888, s. 8, it is provided that animals shall not be carried on any deck below that on which passengers are berthed, 'nor in any compartment in which passengers are berthed, nor in any adjoining compartment, except in a ship built of iron, and of which the compartments are divided off by watertight bulkheads extending to the upper deck.'²

Passenger ships of less than 500 tons register may not carry more than two head of large cattle, nor, in larger vessels, more than one additional head for every additional two hundred tons. Not more than six dogs, and no pigs or male goats, may be conveyed as cargo in any passenger ship.²

Ventilation is one of the most important sanitary matters to attend to on shipboard, for aëration is the proper and principal means of counteracting what De Méricourt³ terms 'les deux plus grands dangers de la vie nautique,' viz. foul emanations from holds or bilges, and the exhalations resulting from the crowding together of human beings in a confined space. The larger the vessel the more complex does the question of ventilation become. The crews of war vessels are worse off than those of merchantmen, in regard of the purity of the air they breathe, owing to the larger number of hands and the difficulty of ventilating properly their quarters, all or most of which are of necessity below deck. The means recommended by the advisers of the Governments of this and other countries for the ventilation of the different parts of ships of war, several of which will be presently described, display a great deal of inventive faculty. But it is evident that from the mere number and variety of these, ventilation in the Royal Navy is felt to be a very difficult and, perhaps, still unsolved problem. Turner,⁴ Medical Director of the U.S. Navy, compares the ventilation of ships to that of an uncorked bottle. On merchant vessels, the structure is on the whole simpler; the crew-space, in

¹ 1892.

² Board of Trade Notice to Passengers, 1887.

³ *Rapport sur le Progrès de l'Hygiène Navale.*

⁴ *Buck's Hygiene and Public Health.*

all but the smaller ships, is above deck, and more easily dealt with; the officers and men to be accommodated are also fewer. But the hold and other parts in which the cargo is stowed are specially to be considered. Speaking generally, however, the ventilation of trading ships and canal boats is not well attended to. The means and appliances are not always there, and, when provided, their use is often neglected. In lower forecastles the hatch is sometimes the only inlet and outlet for air, and in stormy weather this is closed. Occasionally this hatch has a scuttle or companion, with ventilating openings at the side, quite insufficient for the object aimed at. The majority of such forecastles have a hole of about six inches diameter through the roof, fitted with a cowl or mushroom (fig. 4) ventilator, often closed. In some of the steam line-fishing boats, the ventilation is very defective. In one instance coming under the notice of the writer, the engineer's quarters opened directly into the engine-room, and there was no ventilation except by the companion. In this case matters were grossly aggravated by a horrible stench pervading the boat, owing to the foul state of the timbers from fish drainings left decomposing in them since the previous season. Others of these boats have cabins ventilated by hinged skylight cowls, &c., and the engineers are accommodated there, or in state-rooms adjoining.



FIG. 4.—Mush-room Ventilator.

The top-gallant-forecastles of steamers and the larger sailing vessels have ports opening to the outer air, in addition to other means of ventilation. The firemen's quarters of steam-ships, when opening into the engine-room, are apt to be very close and stuffy. Frequently in such cases the vertical shafts employed to carry off the foul air, instead of acting as upcasts, by reason of the greater density of the outer air in comparison with that from the warm engine-room, induce down draughts, and lead to the closing of the apertures. In cattle steamers, &c., where a large amount of fresh air is required, the ventilation of the different decks and hold is often effected partly by ordinary circular-cowled tubes, and partly by large flat-sided shafts (fig. 5), the tops of which are provided with hinged flaps, to direct the air currents downwards.

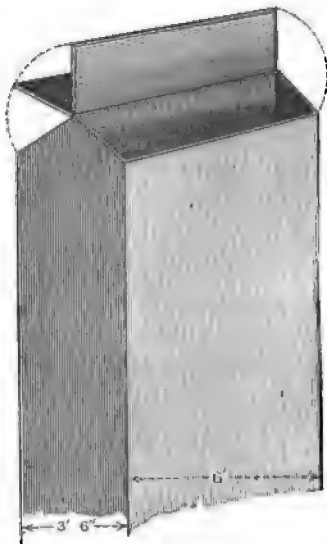


FIG. 5.

In the best class of coasting steamers, trading between British ports with passengers and general cargo, good ventilation is often met with, both in sailors' and passengers' quarters. A favourable example of this kind is afforded in the 'Earl of Aberdeen,' trading between Aberdeen and Newcastle. In this vessel the crew-space is right forward, and of

superior quality. The height from floor to ceiling is 6 feet 6 inches. Air and light are freely admitted by ports. There are also two ventilating shafts, one through the deck and another through the stair-head. As usual, the sailors are quartered at one side of the forecastle and the firemen on the other, the respective compartments being separated by a bulkhead. Over this bulkhead, ventilating tubes are carried from the one set of quarters across the ceiling of the other, and discharges by ports on the opposite side of the

ship. The engine-rooms of steam vessels and the cabins and saloons of passenger ships are ventilated by hinged skylights.

The propulsion of fresh air into the parts to be ventilated, the 'plenum' method, is that most commonly adopted on shipboard, being readily available, by means of the ship's motion, and being also well adapted to keep fresh the holds, 'tween decks, &c., where general cargoes are kept.

Holds are generally ventilated by cowl ventilators, the height of which is regulated by the height of the structure in front of them, but in no case must they be less than 6 feet from deck to bottom of cowl. For vessels with one deck, the ventilator is as shown in fig. 6 (A). In vessels with two decks, the 'tween decks being clear of coal, the form of ventilator is given in Fig. 6 (B); and when the 'tween decks are filled with coal, it is as shown in Fig. 6 (C).

The hatchways or large openings leading from the upper deck to the bottom of the ship are principally used for lading and unlading; but in the case of cargoes liable to heat, &c., these are left open and serve as ventilators. In iron ships the bitts are often hollow and fitted with screw cap, for ventilating the 'tween decks, &c. Hollow iron masts are used for the purpose. The windsail is an apparatus more frequently met with on men-of-war than in trading vessels. It is practically a sort of canvas cowl, consisting of a tube or shaft, the upper end of which is fitted with wings or valves to direct the wind down the tube. Some windsails require to be trimmed, like ordinary cowls, to every change of wind. Others are self-acting. Illustrations of each kind are given in Macdonald's 'Naval Hygiene.' De Méricourt, referring to the substitution of iron cowls for windsails, appears to prefer the latter. The ventilation of a fine barque, examined by the writer, may be quoted as an example of excellence. A central shaft of iron, 11 feet $2\frac{1}{2}$ inches by $9\frac{1}{2}$ inches inside measurement, was carried from the top of the deck-house to the main hold, and was provided with cover to ship and unship, as required. A similar, but smaller shaft forward, and a third ventilator aft of the above, with a fourth on the poop, completed the system, giving means of ample circulation of air below deck. These ventilators are kept open at sea, and their outlets, being high above water, take in no wet even in rough weather. After a thirteen years' experience of the ship, the captain says he has never known any damage to cargo from overheating, &c.

There can be no doubt that the accommodation for petty officers, apprentices, and men on board merchant vessels is more efficiently ventilated when provided amidships in the form of deck-houses, &c. (*see* 'Accommodation,' p. 542).

The Board of Trade surveyor is the authority who decides as to whether a crew-space is properly ventilated. In the 'Instructions as to Survey,'¹ the following is the text on this point:—'The simplest method is to have an iron pipe with a revolving cowl, which in lower forecastles must be as high as the bulwarks, fitted at each end or side of the crew-space, so that while impure air escapes at one, pure and fresh air will enter at the other, and a constant circulation be kept up. Where such means for ventilation is adopted, one of the ventilators should pass through the deck to at least the lower side of the beams.' Mushroom ventilators are discouraged for forecastles. They are not to be fitted to these places unless they are at least 80 inches high for top-gallant-forecastles and as high as the bulwarks for lower-forecastles. There must always be two ventilators. Among other pro-

visions for top-gallant-forecastles, is one for openings in the top and bottom of the bulkheads, covered with perforated zinc, and fitted with doors for closure.

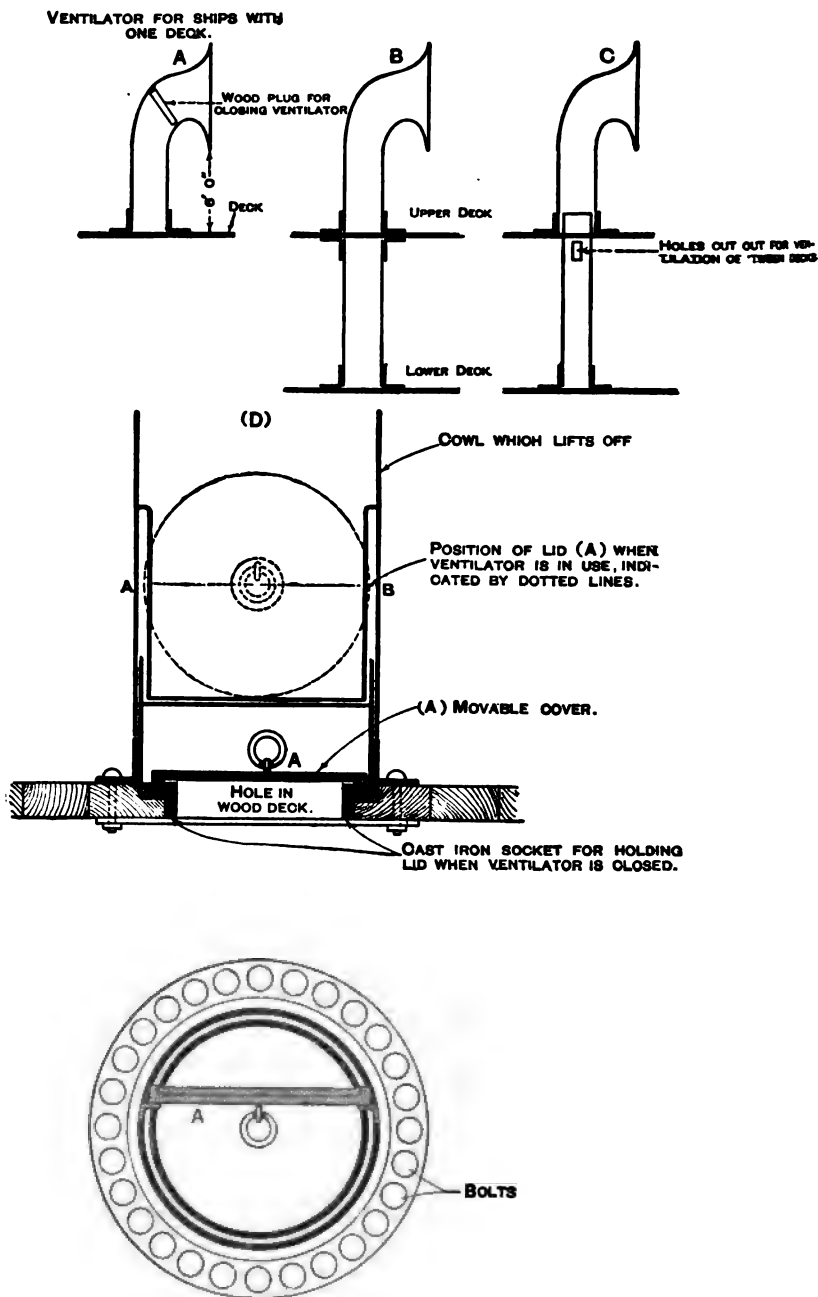


FIG. 6.

Under the Passengers' Act,¹ it is enacted that 'no passenger ship shall

¹ Notice to Passengers, Board of Trade, 1887, Section xxvi.

clear out or proceed to sea without such provision for affording light and air to the passenger decks as the circumstances of the case may, in the judgment of the emigration officer at the port of clearance require; nor, if there are as many as one hundred passengers on board, without having an adequate and proper ventilating apparatus, to be approved by such emigration officer, and fitted to his satisfaction; the passengers shall, moreover, have the free and unimpeded use of such hatchway, situated over the space appropriated to their use, &c., &c.'

The means of ventilation hitherto mentioned include those available through the ordinary openings of the different compartments of ships, and the simpler appliances in general use. These, though useful in their way, are limited in the extent to which they are available. Some are applicable only to the upper compartments, others are liable from their height to be swept off from the deck in rough weather, and to leave openings for the inrush of water, which may sink the ship. Many of what may be termed the natural means of ventilation, such as hatches, companions, skylights, doorways, &c., are of no service for this purpose during a storm, having then to be closed. They are also generally closed at night, and at other times when the apartments to which they belong are occupied, and when their action as sanitary appliances is most wanted. It is therefore necessary to provide more elaborate apparatus, such as can be worked at all times, in all climates, independently of weather, on vessels for cargo, passenger, or war, and on different parts of these vessels.

All artificial systems of ventilation act either by (a) extraction, (b) propulsion, or (c) extraction and propulsion combined.

Many forms of apparatus for artificial ventilation have been proposed, of which the following is a summary:—

Apparatus acting by Extraction.—(1) Pipes from the hold, &c., opening beneath fires, were recommended long ago by Sutton and Duhamel du Monceau.¹ The draught induced by the heat removed the foul air from the lower parts of the ship to the fire where it was consumed.

(2) An up-current may be caused by gas or steam jets discharging into the exhaust pipe; or

(3) By causing the exhaust pipe to pass inside the funnel casing of steam vessels, thus heating and rarefying the outcoming air; or

(4) By fitting its outlet with a mechanical appliance of the nature of an air-pump.

(5) Fan blasts, by being made to revolve the reverse way, may be made to act as exhausts in outlets.

Apparatus acting by Propulsion.—These consist of bellows, pumps, punkahs, rotatory fans, turbines, and other appliances for inducing air currents by means of sprays of water, &c.

The following are illustrations of some of the methods in use for the ventilation of ships.

*Webb's Plan.*²—This is chiefly recommended for steamers, but on other ships the necessary heat may be supplied by a small fire. An ordinary round iron pipe, from two to four inches in diameter, perforated by numerous openings, is led fore and aft under the main deck beams, round each cargo hold and through the coal bunkers, to discharge into the funnel casing. The perforations are made only in those parts of the tube which are in the places

¹ Fonssagrives.

² *Ventilation of Merchant Ships*, by James Webb, Surveyor to Lloyd's. Trans. Inst. Naval Architects, 1884.

to be ventilated. Fresh air is admitted by an inlet on the upper deck, of the same area as the outlet pipe, and is conveyed to the bottom of each compartment. The inlet is bell-mouthed, and is guarded by a ball-valve, acting automatically.

Boyle's System.—This consists of the up-cast and down-cast ventilators acting in combination. Fresh air is introduced by down-cast pipes into the lower parts of the vessel, and the foul air is withdrawn by exhausts (air-pumps), which are fixed and do not get out of order. The objection exists that the shafts may be carried away by heavy seas, &c. The fresh air may be warmed or cooled as required before being supplied into the different compartments.

Fan Ventilators.—Various rotatory fans are in use for purposes of ship ventilation. Some of these have several blades, others have only two. One desideratum of these appliances is that they shall work without noise. They may be made to act either as air-propellers or exhausts. In the larger passenger ships provided with them, the air is tempered before its supply to the saloons, &c. Among the different forms of air-propeller are the Crossley (two blades), and those by Blackman, the Union Engineering Co., &c.

Hall's Ejector is worked by steam, air, or water, causing an exhaust of foul air from the part of the ship on which it acts.

The foregoing systems are undoubtedly an improvement on the crude methods of introducing currents of cold fresh air from the outside, to the discomfort of the persons whose apartments they entered, leaving the foul air to find its way out as best it could. The action of any aspirator will be feeble if the inlet air enters with difficulty. In large saloons the passage of air from inlet to outlet necessitates considerable force. To cause a steady and gradual removal of the products of respiration and the interchange for them of fresh respirable air from the outside, extractive and propulsive power in combination would seem to be necessary. The latter must be so provided that though ample it is under ready control, and the air it sets in motion must be diffused gently and evenly without creating perceptible draught.

Probably the most effectual ventilation is that in which the supply of air at an equable temperature is regarded as the primary object of consideration, and the extraction of vitiated air (though equally important, and perhaps more difficult to effect) a secondary question. The outlets and their exhausts should be adapted to the inlets and supply of air and its density, temperature, &c., and both should be proportioned to the number of persons or amount of combustion consuming the air of the apartment to be ventilated. Among other *desiderata* in all mechanical appliances for this object, they should be automatic; they should also work without noise, and without causing perceptible draught. On shipboard, they should necessitate no long shafts or chimneys, which, if carried away by a wave or the wind, would leave openings for the sea to rush in by. None of the methods above mentioned appear quite to meet these different requirements.

D. C. Green's Method.—Compressed air (8 lb. to 4 lb. per square inch) is employed by means of steam power to induce a larger air-current. It is claimed by the patentees that every cubic foot of compressed air will induce a current of 25 cubic feet of ordinary air; according to this the amount of fresh air to be delivered is capable of accurate regulation. The compressed air is conveyed by small iron pipes to various parts of the ship, and is delivered in the centres of the different ventilating tubes by means of patent automatic air-nozzles, the force of the air issuing through which induces the required current. This process also is adaptable both for exhaustion and propulsion. As in other methods, the air may be tempered before delivery.

The movements of the ship at sea have been taken advantage of by ingenious inventors for the purposes of ventilation. Fonssagrives¹ describes two such methods of ventilation: one acting by the *rolling*, and the other by the *pitching*, of the vessel. The first of these is that of Thiers, of New Orleans, and the second that of Roddy, of New York. The *Thiers* plan has been applied to vessels in the navy, and was described in *The Times* of Nov. 9, 1872, as a 'Ship Ventilating Bilge-pump.' It consists of two vertical cylinders, one partly filled with water and the other with quicksilver, on each side of the ship, each pair being connected by horizontal tubes. Bilge-water pipes from the limbers lead upward and over the side, others for ventilation pass from the bilge to the upper deck. The roll of the ship causes the water cylinders alternately to fill with water or with air, the latter being drawn from below by the vacuum caused by the water leaving the cylinder on one side for that on the other. In like manner, the mercurial cylinders alternately fill with mercury or bilge-water. As the vessel rights herself, the air or water pumped from below discharges from the respective outlet pipes. The pipes are guarded by check-valves.

The *Roddy* plan is similar in principle to the foregoing, but has the cylinders placed fore and aft so as to be actuated by the pitching movement.

Macdonald² quotes Staff-Surgeon Magill, M.D., of H.M.S. 'Thetis,' to show that the Thiers system is not satisfactory in its working, and that 'when ventilation is most required it is altogether inactive.' It is in principle the same as that of *Perkins's Automatic Ventilator*, invented at the beginning of the century. This plan utilised both the pitch and roll of the ship. The illustration (fig. 7) is taken from Macdonald's 'Naval Hygiene,' where the reader will find quoted a description by Dr. Finlayson, R.N.

Notwithstanding the eulogium passed on this apparatus by the describer, it will be obvious that any system of ventilation based on the roll or pitch

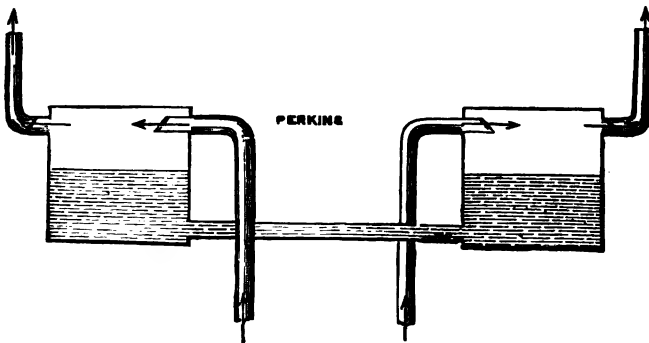


FIG. 7.

of a ship must fail in its operation when the vessel is at anchor, becalmed, or moving steadily. For several other varieties of special systems of ventilation, and remedial measures for badly-ventilated ships as applied in the Royal Navy, the reader is referred to Dr. Macdonald's 'Naval Hygiene.'

¹ *Traité d'Hygiène Navale*, 1877.

² *Naval Hygiene*.

ACCOMMODATION AND SPACE

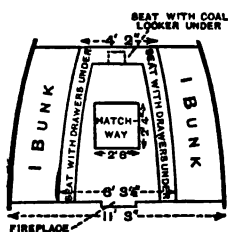
1. *For Crew.*—The sailors' remuneration includes housing, such as it is; and he does not look for much in this respect.

In British merchant ships the crew are generally housed in the top-gallant or lower-forecastles respectively of the larger or smaller vessels. In many foreign traders, even schooners, and in some English steamers and sailing ships of good class, the men are quartered amidships. Occasionally in cattle ships, &c., built within the last few years, the lower-forecastle is in the after part of the ship beneath the poop-deck, and the officers' rooms are amidships. The firemen of steamships are usually placed at one side of a top-gallant-forecastle, and the crew at the other, with a division between. Sometimes the former have their accommodation near their work, opening into the engine-room, &c. From a hygienic standpoint, deck-houses or other accommodation amidships is the best, as in this position light, aëration, dryness, and general comfort are best secured. In lower-forecastles, in addition to many insanitary drawbacks, one great objection is the difficulty of access. In some of the worst cases the hatch is perhaps about two-and-a-half feet square, and the only steps are narrow pieces of wood nailed against the bulkhead, affording an insecure footing, and such as a sick man cannot ascend by. The removal of a case of enteric fever in the horizontal position from such a place is, of course, impossible; and hauling the patient up by his arms is very dangerous. There can be no justification for continuance of lower-forecastles, as the masters of small vessels with deck-houses say that these cause no difficulty in the management of the ship.

The following illustrations (figs. 8 to 18) show the accommodation pro-

CABIN OF KETCH "PHOENIX"
30 TONS REGISTER.—2 HANDS

AFTER END



FORE END

NO VENTILATION EXCEPT
BY HATCHWAY HEIGHT 4' 6"

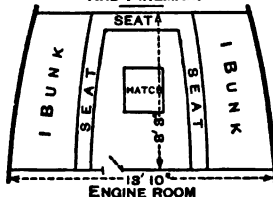
Fig. 8.

S/S LINE FISHING BOAT
"LIZZIE" OF LEITH.

HEIGHT 6' 2"

8 HANDS ALL TOLD

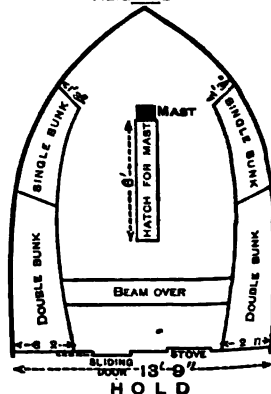
AFTER CABIN FOR ENGINEMAN
AND FIREMAN.



WHEN THE HATCH IS CLOSED, THE
ONLY VENTILATION IS BY THE EN-
GINE ROOM SKYLIGHT.

Fig. 9.

NET FISHING BOAT
"PERSEVERANCE"
OF BUCKHAVEN.
10 TONS REGISTER.—7 HANDS.
HEIGHT 5' 4"



FORE CABIN OR "DECK"

Fig. 10.

vided on different kinds of vessel. Figs. 8 to 11 sufficiently explain themselves. Fig. 12 represents part of the forecabin of a barge, below deck, the approach to which is by a companion, covered by a scuttle. The 'bunks' or sleeping-places are boxes, each 6 ft. 11 in. in length, and having an opening in the side near the middle 2 ft. x 1 foot 11 in. The remainder of the interior of the bunks (length 4 ft. 11 in.) is therefore in deep shadow,

and the head of the sleeper is in a deep recess. The ventilation of such a bunk—if it be ventilation—is necessarily very bad indeed. Fortunately, examples of this kind are not, in the writer's experience, frequent.

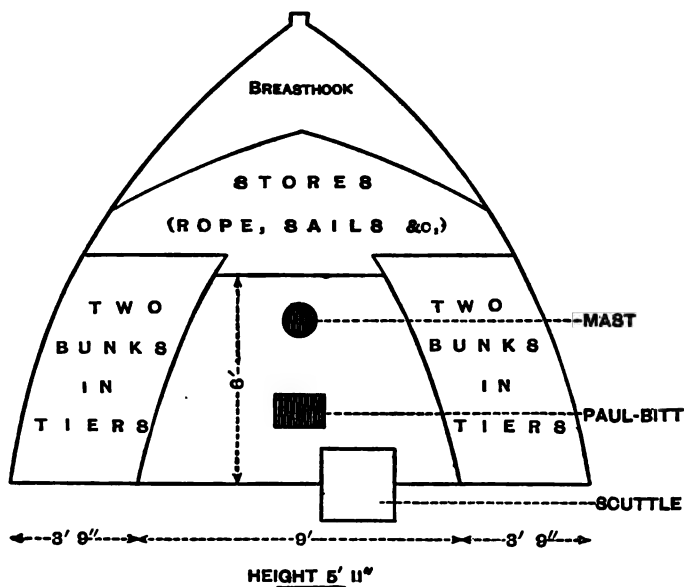


FIG. 11.

Fig. 13 is the ordinary top-gallant-forecastle of a sailing barque. The privies are represented in their usual situation, abutting on the bulkhead

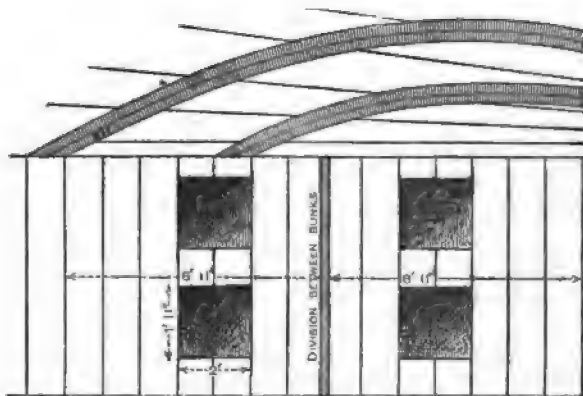


FIG. 12.

of the fore-cabin. The doorways both of privies and crew-spaces are badly placed.

Fig. 14 shows the quarters of the sailors and firemen of a modern flush-deck screw steamer, built by Messrs. John Blumer & Sons, Sunderland. Ten seamen sleep in five double tiers of beds, the cook and boy having a separate berth, all on the same side of the ship. On the other side are the quarters of ten firemen, the carpenter and boatswain, the two latter having a separate berth. The water-closet and offensive stores are on the upper deck, out of the way of the men's quarters. The ventilation is effected by six

ports (three on each side) and two deck ventilators. The officers' quarters on this and the following plan will be referred to later.

Fig. 15 represents the usual accommodation on a well-decked steam-vessel, the men being housed as in the last instance. The water-closets are on the same deck as the crew-space, separated from it by storerooms.

Fig. 16. Here the men's quarters of a well-decked steamer are placed amidships (beneath the bridge), with good arrangement for light and ventilation. The closets are well forward.

Fig. 17 is a plan of the best seamen's quarters on any vessel known to the writer, viz. that of the iron sailing barque 'Inchgreen,' built in Greenock in 1876 by Messrs. Caird & Co., and having a complement of twenty-one hands all told. This vessel was specially constructed for the merchant service. The sailors are quartered in an iron house amidships. Each man has a separate berth to himself, also of iron, measuring

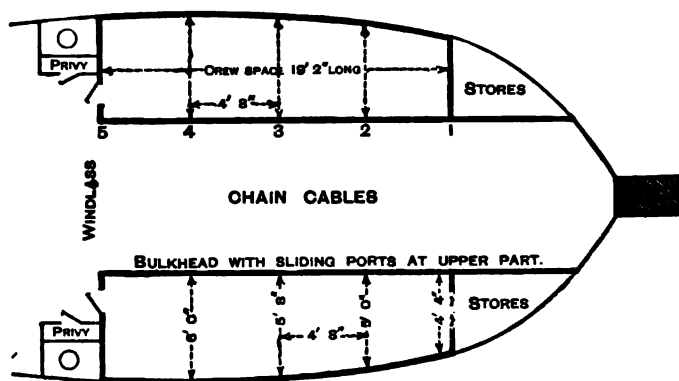


FIG. 13.¹

6 ft. 1½ in. × 5 ft. 1 in. × 6 ft. 2 in. in height (more than double the space required by law). The berth is lined with cork, and painted white, and has a port and a louvred ventilator, 15 in. × 16 in. in measurement, in the door. Beneath the bunk is a chest of three drawers. There is also a washing-basin with can, a seat, and a shelf in each berth. The berths open into a cheerful saloon, containing a stove and a sideboard, and having hinged skylights with roof. These looked very comfortable, and were quite clean.

This is believed to be the only sailing vessel of the kind. The captain finds the crew like their accommodation, and remain well with him.

Another, among other vessels affording excellent accommodation, may be mentioned the four-masted barque 'Loch Torridon,' 2,000 tons register, with thirty-three hands all told, including four apprentices. The crew of twenty-one men live in a deck-house amidships, 7 ft. 11 in. high, with skylight and eight ports. The bunks are several inches clear of the side of the house (a great advantage).

The deck-houses of some foreign timber ships coming to the Tyne offer good examples of accommodation, having sliding doors on each side, ample ventilation, and good skylights, &c. The Danish law requires deck-houses to be at least 6 ft. in height.

The average accommodation for the crews of ships is poor enough. In the older vessels it is in the majority of cases bad, and often very bad. In

¹ From Board of Trade *Instructions*, 1892, Appendix C.

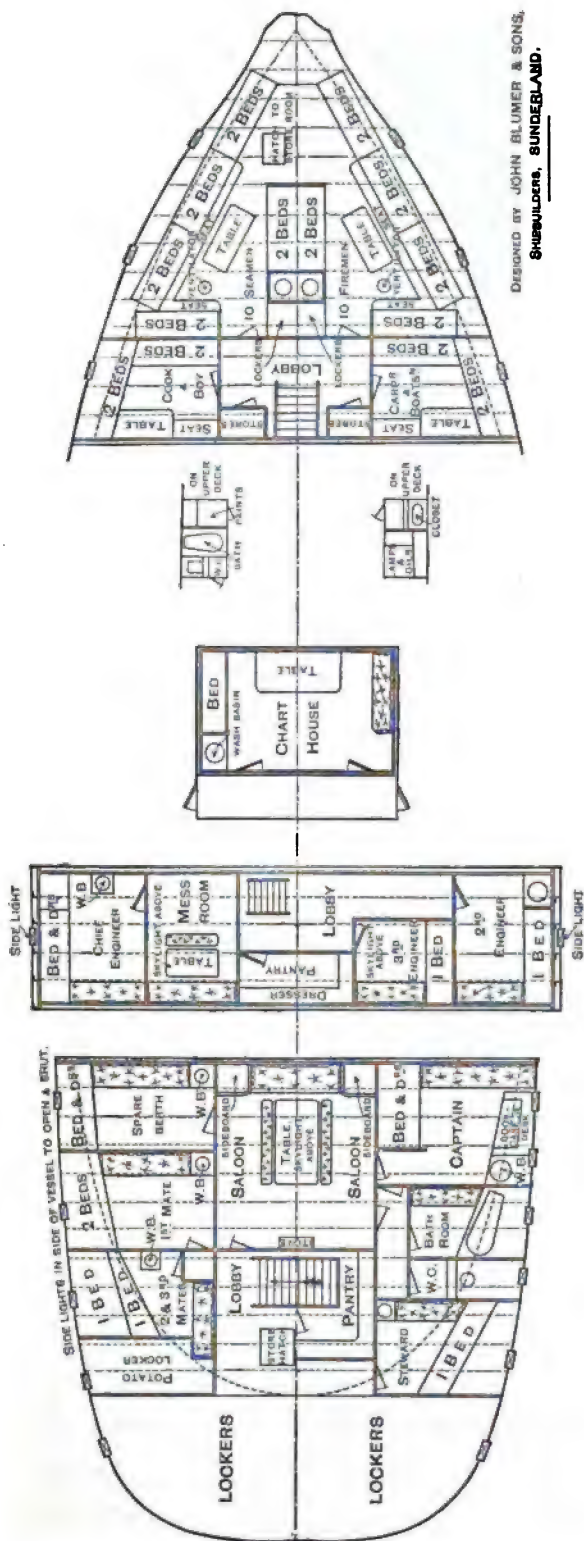
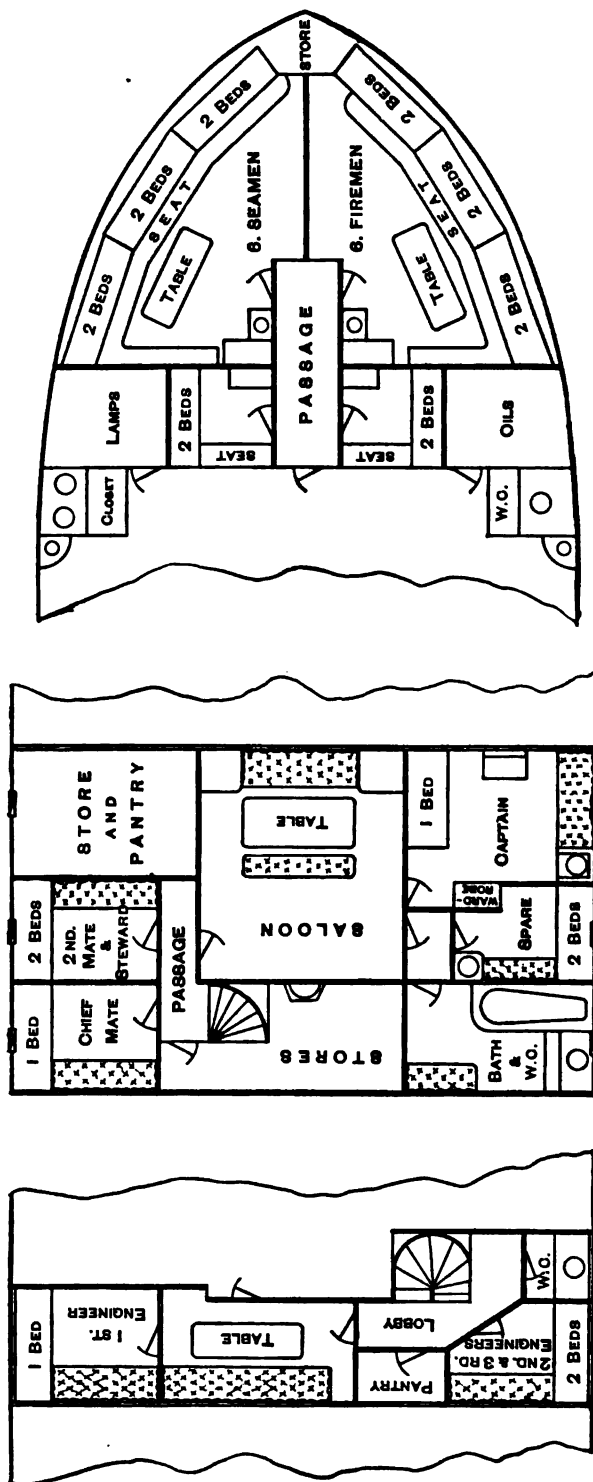


FIG. 14.



DESIGNED BY JOHN BLUMER & SONS,
SHIPBUILDERS, SUNDERLAND.

WELL DECK STEAMER HAVING NO POOP, & OFFICERS ACCOMMODATION
UNDER BRIDGE AMIDSHIPS 260' x 35' x 19'0" DEPTH.

FIG. 15.

the Scotch herring boats the forecaskle (or 'deck' as it is called), is separated from the hold in which the fish are stored by a sliding door only. This apartment, which is occupied by the entire crew of seven hands, is also the galley. It is often very dirty.

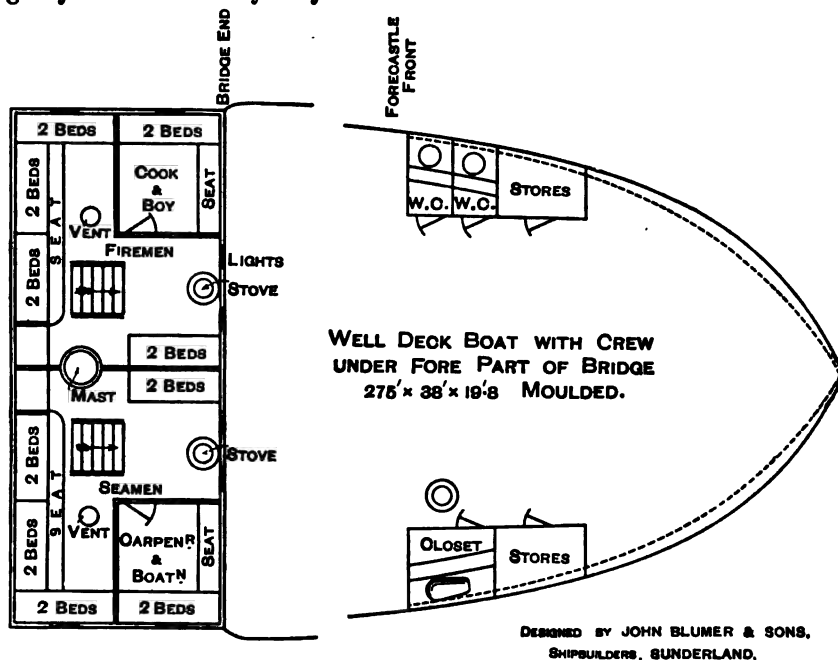


FIG. 16.

A redeeming feature in boats of this kind is the mast-hole, an opening about 6 ft. long by 1 foot in breadth, for the mast to be in when lowered. During fishing, when the mast is raised, this space is left open to the interior of the 'deck' in which the crew stay.

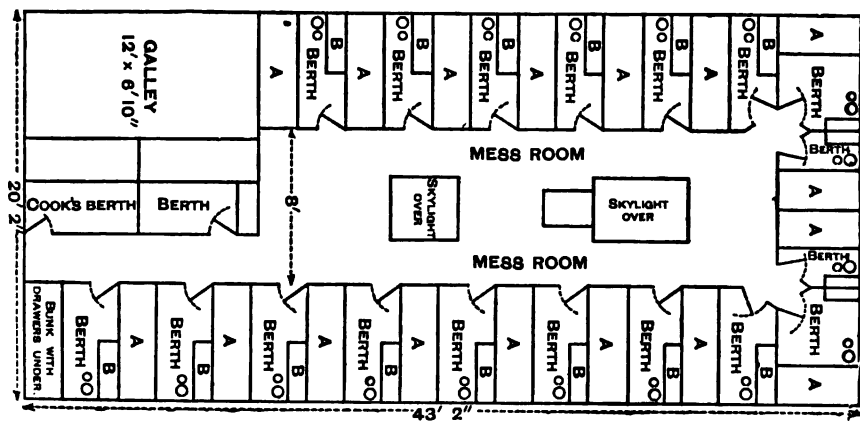


FIG. 17.

A favourable contrast from these boats is afforded by some of the Shields screw-steamer 'line' boats, the cabins of which are sometimes very clean, tidy, airy, and comfortable.

In some of the river craft on the Tyne (towing wherries, &c.) the crew-

space is a lower cabin known locally as the 'huddock.' It is used for meals and shelter, but not for sleeping in.

The contents of crew-spaces are (1) proper and (2) improper. The first includes commonly the hammocks or bunks and bedding, lockers, chests, table, stove and lamp. The articles which should not be, but often are found in forecastles, may include all the different materials and implements on board which it is possible to stow away there, such as sails, cordage, spare blocks, buckets, cans, brushes, tar, paints, oil, paraffin, wet clothes, mouldy boots, &c., and (in the 'weekly boats' previously referred to) provisions. All of these occupy space illegally, and to the privation of the crew, and many of them pollute the air to a serious degree.

The principal defects of sailors' quarters are insufficient height, light, ventilation, means of warming; protection from effluvia of cargo or bilgewater, improper storage, and overcrowding. The latter may naturally be considered in relation to the subject of accommodation.

The amount of space required by law¹ for each seaman is 72 cubic ft., including 12 square ft. of floor space. The Local Government Board and Port Sanitary Authorities have not the power to regulate matters of overcrowding afloat, which are relegated to the Board of Trade and their inspectors. The foregoing regulation is much in need of revision. The accommodation is too little, even in the estimation of the owners, who very rarely crowd the seamen up to its limits. The law requires that the number of seamen to be accommodated shall be cut on the beam in the inside of the crew-space. Frequently the crew-space is certified to hold double the number of men housed in it. In an instance known to the writer, that of a brig with seven hands 'all told,' the crew-space, certified to accommodate twelve seamen, contained four hammocks only.

Enlarging the crew-space means reducing the dues (harbour, light, &c.), as these are charged on registered tonnage, which is determined by space other than crew-space, the latter being deducted. Hence it is to the interest of owners to give liberal accommodation for their crews. In factories, though it is not absolutely laid down by law, yet it is well understood that the amount of space to each workman should not be less than 250 cubic ft.² In forecastles, &c., where seamen have to sleep, it is necessary that the cubic space should be at least as ample as for workshops.

In the larger ships the tendency at present is rather to under-manning than to over-crowding, as defined by law.

In canal boats and small craft there is often more deficiency of cubic space for the men than in larger vessels, though probably this also is not now so prevalent as formerly. Blyth³ quotes Sir Charles Cameron, who mentions the cabin of one canal boat as containing 189½ cubic feet for three occupants, or 61 cubic feet each. Leach also gives evidence of the overcrowding of barges, &c. Some of the small foreign vessels coming to this country with fruit, &c., are frequently overcrowded with men engaged to sell the cargo. Thus during 1888 a small vessel of five hands came to the Tyne with onions carrying nineteen 'passengers' (to hawk the cargo in the streets), who appear to have slept in the hold, there being no other place for them on board.

In the Board of Trade Instructions to Surveyors as to crew-spaces, &c., it is distinctly laid down that the Merchant Shipping Act of 1867 is not intended to regulate or interfere with the manning of vessels or amount of

¹ Merchant Shipping Act, 1867.

² Redgrave's *Factory Acts*.

³ *Dict. of Hygiene*.

accommodation, which are left to the owners and their crews to settle. The tonnage of space deducted has not to settle the number of hands to be carried. Notwithstanding these provisions, it is obvious that any attempt to reduce overcrowding on shipboard which does not exceed the 'accommodation' certified to by the Board of Trade must be very difficult.

It appears to be generally understood on shipboard that the Rules of the Board of Trade, as to 'accommodation,' are to be the standard regulating the amount of cubic space which is to be considered overcrowding or otherwise, and this standard is held as applying alike to officers and men.

Under the 'Passengers Act'¹ all passenger ships are to be surveyed. The amount of deck space required is, for each adult on the upper passenger deck, &c., not less than fifteen, and on the lower passenger deck eighteen square feet. If the height between the latter deck and that above it be less than seven feet, or if the means of lighting and ventilation are below a specified standard, the minimum amount of floor space per adult is twenty-five square feet. No ship is allowed to carry 'a greater number of passengers on the whole than in the proportion of one statute adult to every five superficial feet, clear for exercise, on the upper deck or poop, &c.'

The height between any deck on which passengers are carried and that above it shall not be less than six feet, under a penalty not exceeding 50%.

There shall not be more than two tiers of berths on any one deck, and the interval between each tier, and between the uppermost tier and the deck above it, shall not be less than two feet six inches.

Hospital accommodation in the proportion of not less than eighteen clear superficial feet for every fifty passengers must be provided. The lighting and ventilation must be such 'as the circumstances of the case may, in the judgment of the Emigration Officer at the port of clearance, require.'

The *sleeping accommodation* of the seaman is either a hammock or a bunk. The latter is a square tray with deep sides, sometimes (as in fig. 12) very much enclosed. Hammocks are much to be preferred to bunks as regards the space they occupy, cleanliness, comfort, vermin, and ventilation.

DRAINAGE, CONVENIENCES, CLEANSING, &c.

Iron upper decks are drained by means of scuppers and wash ports at the side, discharging through the bulwarks. The surface of the deck is 'cropped,' i.e. curved with the convexity upwards, to the extent of a quarter of an inch per foot of breadth of the vessel, to cause flow toward the sides. Iron ships with wooden decks have waterways at the sides from twelve to eighteen inches wide. Lower decks are also cropped. They are drained by scuppers leading down to the bilges. Leakage through the seams of the outer skin of the ship flows also to the bilges. From the bilges the drainage flows to the limbers and thence to the well or sink, where it is pumped overboard from time to time. Flatness of the bilges causes lodgment of filth and the evolution of foul gases. This defect, as pointed out by Macdonald, is common in modern ships, and leads to accumulation of stagnant foul liquid (bilge-water). Authorities agree as to the great danger to health from this defect. The pumps should be capable of clearing out *all* of the bilge-water. In the limbers deposit is prevented by chains (limber chains) arranged so as to work in the length of the limbers, and stir up any

¹ See *Board of Trade Notice to Passengers*.

dirt lodged there, which should never be allowed to remain for any length of time. It is well known that neglect of this sanitary precaution has led to serious outbreaks of disease.

Macdonald recommends that the bilge floors on either side of the keelson should be made perfectly smooth, and cemented, lacquered, or tiled, with sufficient incline, from both ends of the vessel, to the well. It is desirable to cleanse and *thoroughly dry* the bilges from time to time. The drainage arrangements of ships are similar to those of old-fashioned houses with cess-pools in the basement, a sanitary defect on shore now absolutely condemned. The modern principles of house drainage, viz. disconnection from the sewer and free ventilation from end to end of private pipes, are, however, scarcely in their entirety applicable to ships.

The *conveniences* met with on shipboard are water-closets of different kinds and pail closets. In the better class of vessels, passenger ships, &c., the water-closets are generally of good form, and placed in good situations. They are also provided with cisterns and suitable arrangements for flushing. In the majority of ships, however, the water-closets, especially those for the crews, are in objectionable positions, frequently being placed close to cabins or crew-spaces (see fig. 18). Fig. 18 is an example of a faulty arrangement of the convenience on a small screw steamer. The water-closet is shown at the bottom of the companion and exactly opposite to the door of the master's cabin. It abuts on the pantry.

The sailors' water-closets are generally flushed by hand-pump and hose-pipe, or by means of a bucketful of water thrown in by hand. In the latter case the closets are often very dirty.

One of the most curious and ingenious forms of water-closet afloat known to the writer is one noted on board the new s.s. line fishing boat 'George Baird,' of North Shields. This closet is about eight or nine feet below deck, and below water-level. The flush-water is drawn directly from the sea by the user of the closet pulling the handle of a pump at his right side. The contents of the basin are discharged by a valve actuated by a handle near his left hand. The basin may be flushed with any quantity of sea water. Owing to the difficulty of the approach (a vertical plank with holes cut in it for the feet and hands), this closet is not so freely used as its sanitary qualifications entitle it to be.

The soil-pipes of ship water-closets sometimes discharge through the bulwarks, or near the top of the covering board, which closes in the upper border of the two 'skins' of the ship. Either method is objectionable when the soil-pipe is not carried down so as to discharge at a proper distance from the deck and out of sight. In the latter case there is risk of leakage into the space between the 'skins.'

The best position for a closet is in the after part of the ship, so that the soil-pipe may shoot over or through the counter near the rudder-trunk. The lower end of the soil-pipe should be protected by a storm-valve closing against pressure from the outside, so as to prevent flooding.

As a rule, the ventilation of water-closets and privies on shipboard is defective.

On some schooners, &c., the dry pail is the only form of convenience in use. Small French ships (and English ones also) have often no proper closet on board, the men using buckets placed behind canvas screens, or defecating from the bow of the ship straight into the sea.

The bulkheads between closets and the sailors' quarters are not always sufficient to prevent nuisance in the latter.

The proportion of privies on passenger ships required by the Board of

Trade is at the rate of two for twenty of crew.¹ No passenger ship is allowed to clear out unless fitted with at least two privies, and with two additional privies for every hundred passengers. Special convenience for female passengers is also required.² Fore-cabin and deck passengers in home-trade ships are to have closets in the ratio of three for every 200 such passengers, 'and a fair proportion must be allotted to the sole use of women and children.'³

It is the business of the surveyor of the Board of Trade to see to the various details necessary for the proper drainage and cleansing of merchant ships, and specific instructions and information are given for him to act on. Some of these are not such as would receive the approval of medical officers of health.

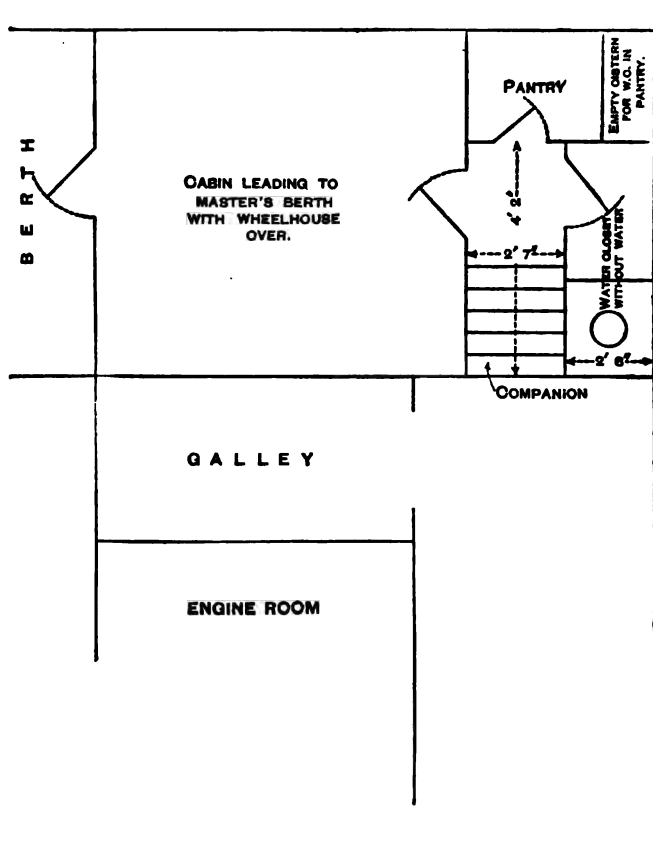


FIG. 18.

The unclean state of ships, especially those of small size, is very common, not only in parts out of sight, but on deck, holds, forecastles, cabins, &c. The habits and customs of the merchant seaman, and his appliances for removing dirt, are not such as to promote general cleanliness either in himself or his surroundings.⁴ Turner says the forecastle in merchant vessels

¹ *Board of Trade Instructions as to Survey*, Sec. 55.

² *Notice to Passengers*, Sec. 25.

³ *Board of Trade Instructions as to Survey*, Sec. 18.

⁴ *Buck's Hygiene*. Art. 'Hygiene of the Naval and Merchant Marine.'

'beggars description for overcrowding, want of ventilation, and filth. The bunks are harbours for vermin ; unaired bedding, wet clothes, and all manner of dirt find a resting-place in the fore-castle. . . . Every writer upon marine hygiene bears testimony to the loathsomeness of this pen.' A most vivid description of this part of the ship is given by Mr. W. S. Lindsay, M.P.,¹ who testifies from personal experience as a cabin boy to its wretched and foul condition in years gone by. Matters have not improved much in these respects during the past twenty or thirty years. There are certainly many fore-castles in a creditable or very clean state, but these are the exception and not the rule. Water-closets, holds, and decks are also apt to be dirty—the first in trading ships of any sort—the two latter in case of special cargoes, &c. Cattle ships afford examples of the filthiest vessels to be found. Imagine the condition of such a ship coming from New York to London, with from six to eight hundred head of cattle on the hurricane, upper and main decks, the dung from which remains on board throughout the passage of about a fortnight's duration, and until after the vessel discharges the live stock part of her cargo and steams out of the Thames ! The decks are generally covered over with planks to afford foothold to the quadrupeds, and beneath these planks much of the droppings collects. The cattle are bedded with hay or straw, and stand with their tails to the ship's side. There should be several large port-holes for discharging the litter by, as well as other structural arrangements to allow of this being done daily whilst the live stock are on board.

Next to cattle boats for general filthiness come fishing boats. It is customary to wash out the holds of these vessels after each 'take,' but the 'decks' or cabins are not cleansed after many 'takes.' Filth collects under the bunks, the cost of paint is a serious consideration, and the use of limewash, so far as the writer is aware, is not practised. The stink of the bilges of fishing boats, when neglected, is something horrible.

Some barges are kept very clean, and their cabins are often very tidy and comfortable, even when carrying dirty cargoes. Norwegian and German merchant ships are, as a rule, much cleaner than the French, Spanish, or Russian. Excellent examples of clean, well-kept ships are afforded by the Dutch East Indiamen, some of which visit the Tyne. Merchant ships, as may be supposed, are not kept so clean as those of the Royal Navy, which it is the duty of the captains—as laid down in Article 499—to see strictly to, and to cause the holds to be whitewashed every six months or oftener if necessary.

In the Royal Navy the decks are cleaned by holystoning (wet or dry), and in the berth-deck by scraping and scrubbing with hot water, wetting only a small portion at a time and drying thoroughly. The two former methods are open to objection in the inhabited parts of the ship, one from filling the air with dust, the other from loading it with vapour. The last-mentioned method is, however, spoken of with favour by Dr. Wilson, of the United States Navy.²

There appears to be a general consensus of opinion against flushing out the bilges with sea-water. Pumping these places well out, and drying them as thoroughly as possible, is always preferable. The use of antiseptics and deodorants, notwithstanding their efficacy in preventing foul smells and the evolution of noxious gases, should never be relied on to the exclusion of the frequent and complete removal overboard of the bilge water itself.

¹ *History of Merchant Shipping and Ancient Commerce*, vol. ii. p. 497. London, 1874.

² *Naval Hygiene*.

Humidity on shipboard is an acknowledged serious evil. It may come from leaking sides or decks, or from the shipping of seas and accumulation of water in the hold, &c., or may be due to the washing of the decks. To keep the vessel dry, decks and planks must be thoroughly caulked, the planking above water must be well coated with paint, and the means of drainage already described must be efficient. With the view of rendering wooden decks waterproof they are sometimes oiled or varnished.

Iron ships are subject to humidity from a cause not in operation on wooden vessels, viz. the condensation of vapour against the metallic plates. This acts to a degree which would scarcely be credited. The walls and ceilings of forecastles and officers' quarters, where constructed of iron merely painted, drip with wet, and are often quite uninhabitable. The remedy is to line the metal plates with wood or cork. The latter is frequently applied in the form of rough granules to the surface of wet paint laid on the iron. An objection to the former plan is that vermin breed behind the woodwork.

TEMPERATURE AND LIGHTING

The temperature of the ship, apart from considerations of climate, depends principally on (1) whether or not the vessel is propelled by steam; (2) whether or not she is constructed of metal; and (3) her cargo.

Steam vessels, from the large amount of coal consumed, attain a much higher temperature than wooden ships. This extra heat is, of course, greatest in the neighbourhood of the furnaces; it may also, of course, be conveyed by steam pipes to various compartments and serve as a means of artificial warming. The parts of steam vessels where the heat is most excessive are the stokeholes and engine rooms. Mahé¹ quotes Admiral Paris to show that in the former, between two sets of furnaces, the temperature sometimes reaches 60° C., and in the Red Sea even 65° C. De Méricourt states that with a temperature of 95° F. in the shade, temperatures of 158° F. and 167° F. have been registered in the stokehole.²

Where temperatures such as these are recorded they are probably due to the fact that the ventilating cowls are wanting or insufficient. The writer is assured by a superintending engineer of merchant shipping of great experience, that in hot climates the stokehole is sometimes, from its depth below water and from the currents of air which are brought down to it by the air-shafts, the coolest part of the ship. If suitable provision for the admission of fresh air to the stokehole is not made, the fires will not burn properly. It is, therefore, to the interest of the owner to make such structural arrangements as are necessary to facilitate the removal of warm foul air and the influx of cool fresh air from above.

The engine-room, especially when the skylights are down, becomes very hot. The quarters of the firemen sometimes open into this room, and are unhealthy from high temperature. The evil effects on the hearts of stokers from working under continued heat from the fires is referred to by several writers. They will be referred to under the head of 'Health and Disease,' (p. 574, &c.).

The hold of sailing ships is frequently warmer than the other parts of the vessel. This is partly accounted for by its depth and consequent distance from the influence of the external air; but more commonly this increase of temperature depends on the cargo. Coal, sugar, grain, lime, and many other

¹ *Manuel Pratique d'Hygiène Navale.*

² Turner (Buck's *Hygiene*) gives instances of still higher temperatures (181° and 198° F.).

cargoes, are apt to undergo various chemical changes attended with the evolution of much heat. Cotton waste, if greasy and under pressure, in the absence of free aëration, may be subject to spontaneous combustion.

Occasionally cargoes are shipped whilst sensibly *hot*. For example, this has happened to the knowledge of the writer in a small vessel laden with briquettes of compressed fuel, which gave off both heat and the vapour of tar.

The temperature of the holds of steamships is raised by blowing out the hot-water of the boilers into the bilges.

The obvious remedy for the over-heating of stokeholes is the copious admission of air from above deck by ventilating shafts, with cowls trimmed to the wind. Where these are not admissible, as on torpedo boats and other war vessels, the furnaces and, with them the stokeholes, are supplied with air by fan blasts.

In tropical climates iron decks and all parts exposed to the direct action of the sun's rays become exceedingly hot, and this heat, by conduction, &c., is imparted to the interior of the ship. Much of the discomfort arising therefrom may be reduced by the use of light-coloured, and especially white paint, which less readily absorbs the sun's rays. Iron ships are liable to extreme variations of temperature (Bourel-Roncière). In high latitudes they become much colder than wooden vessels. Wilson records the coating of the walls, &c., of crew-spaces with ice from the freezing of the condensed vapour, which he considers to be as a serious cause of ill-health of seamen.

The different compartments of ships are warmed artificially by fire-places, stoves, steam-heaters, &c. In the forecables of merchant vessels there is generally a 'bogey,' or small stove. This is rarely absent in British vessels, but is said to be less common in the crew-spaces of the French mercantile marine. Under certain circumstances, the warming of crew-spaces by means of steam-pipes is resorted to. So far as is known to the writer, there is no provision under the English law for the warming of crew-spaces, &c.

The *lighting* of the quarters for officers by day is generally fairly good. That for passengers on emigrant ships, and for crews, is often deficient, and in lower-forecables is usually bad. In top-gallant-forecables the lighting is effected by ports and side- or deck-lights. The latter consist of prisms of glass, which do not allow much light to pass through. In some small vessels the only light in the lower-forecastle is that admitted by the hatchway, consequently the greater part of the crew-space is buried in obscurity so dense that, on first entering it, the visitor is unable to distinguish one object from another. Even in steamships the crew-spaces are sometimes so dark as to necessitate the use of a lamp in the daytime. The reading of 'ordinary print,' in any part of the crew-space is recommended to surveyors under the Board of Trade as the criterion of sufficiency of light admitted. By night, the illumination is effected by means of oil, candles, or electricity.

(C) CREWS, PASSENGERS, &c.

*Royal Navy (England)*¹

The total force in the service afloat is 55,670 officers and men, of whom about 57 per cent. are between fifteen and twenty-five years of age; about 81 per cent. aged from twenty-five to thirty-five; about 10 per cent. from thirty-five to forty-five; and about 1.6 per cent. above forty-five years of age.

¹ *Statistical Report of the Health of the Navy for 1891.*

Merchant Service

The number of persons employed in the Mercantile Marine of the United Kingdom in 1887¹ was as follows :—

—	Total	Foreigners	Lascars
Sail	81,189	12,436	170
Steam	159,291	17,831	23,867
Total	240,480	89,267	24,037

The total includes 18,291 persons on fishing boats registered under the Merchant Shipping Acts, but does not include the crews of boats registered under the Sea Fisheries Acts, which are given below. The crews of yachts and vessels employed on rivers and in inland navigation are not included.

*Persons employed in Boats registered under the Sea Fisheries Act, 1868.**

—	Approximate		Total
	Men and boys constantly employed	Persons other than regular fishermen occasionally employed in fishing	
England	88,044	9,011	42,055
Scotland	82,861	19,872	52,783
Ireland	10,600	14,754	25,354
Total	76,505	43,637	120,142

Apprentices²

Number of apprentices' indentures enrolled in each of the understated years :

Year	No. enrolled
1845	15,704
1855	7,461
1865	5,688
1875	4,397
1885	2,504
1891	2,186

Apprentices are not now required by law, as was formerly the case.

Manning of Ships⁴

The manning of ships has declined of late. In 1891 the proportion was 2.95 (3.00 for steam, and 2.85 for sailing vessels).

In the purely home trade there has been but a slight reduction of manning, and that only in steamships.

¹ *Annual Statement of the Navigation and Shipping of the United Kingdom for 1891* (Board of Trade), pp. 262-268.

² *Ibid.*, pp. 279-288.

³ *Board of Trade Returns of British Merchant Shipping, May 25, 1892.*

⁴ *Ibid.*

An analysis of the crews of twenty-nine British sailing vessels, belonging to well-known firms in different ports of the United Kingdom, having an aggregate tonnage of about 28,000 tons, ranging from 200 to 2,000 tons, and trading to various parts of the world, shows that in ten years there was a decrease in total crews of 106 (629 in 1890 against 735 in 1880). This was principally under the head of 'able' seamen and apprentices.

A similar comparison of the crews of thirty-two British steamships of an aggregate of about 58,000 tons employed in the foreign trade shows at the close of the same decade an increase of total crews of 12 (2,461 in 1890 against 2,449 in 1880), though there was a diminution of 'able' seamen (460-406), boys (41-82), and firemen (460-452), the number of *Lascars* and other Asiatics employed having slightly increased (332-347).

The following table¹ shows the population of men to tonnage in ships of different sizes and kinds:—

Classification of tonnage				1891 Average number of men to a ship	
				Sailing	Steam
Under 50	.	Tons	.	3.6	6
Of 50 and under	100	"	.	4.4	9
" 100	200	"	.	5.8	13.2
" 200	300	"	.	8.2	18
" 300	400	"	.	10.1	19.5
" 400	500	"	.	12.6	19.3
" 500	600	"	.	13.7	21.0
" 600	700	"	.	15.5	20.9
" 700	800	"	.	16.6	20.2
" 800	1,000	"	.	18.7	23.6
" 1,000	1,200	"	.	21.7	25
" 1,200	1,500	"	.	24.8	29.7
" 1,500	2,000	"	.	29.1	38.2
" 2,000	2,500	"	.	32.0	69.1
" 2,500	3,000	"	.	35.2	89.5
" 3,000 tons and above	.	.	.	38.8	143.1

THE SAILOR, &c.

The seafaring population of the United Kingdom (Royal and Merchant Navies, fishermen, and passengers) numbers therefore nearly three-quarters of a million, about half of which are mariners by profession.

The personal hygiene of this body of men, not only on account of their numbers and the peculiar character of their avocations, but also by reason of their importance to the nation, demands special study. The attention claimed by the subject generally has not been given to it. With the exception of the Royal Navy, which forms, as appears by the statistical returns already given, only about 15 per cent. of the entire population engaged in the marine service of this country, our sailors and fishermen as a body cannot be said to meet with that amount of sanitary care and consideration to which, from the privations they suffer, the risks they run, and the duties they perform, they are fairly entitled. To render the mercantile and fishing service efficient, it is necessary that the men should be carefully chosen from a good class; that their status and condition, social and hygienic, be made as attractive as possible; and that they be well trained and disciplined. How these matters are attended to will be understood by comparing the

¹ Prepared from the *Annual Statement of the Navigation and Shipping of the United Kingdom* for 1891, by the Board of Trade, pp. 262-63.

information available with respect to the different navies for the purposes of war, commerce, and fishing.

'The scarcity of competent sailors, and the consequent rise in wages, threaten to injure seriously the vast commercial interests of this country,' as was said by Leach¹ nearly a quarter of a century ago, may be repeated at the present day. The sea is still as unpopular as a service as it was when the above words were written. Lindsay,² referring to the shipping of crews, says the shipping masters were generally paid per head engaged and put on board before sailing, and the men were sometimes carted to the ship drunk. Everyone knows of poor Jack as the victim of the crimp, and of impressment in the old times. Upwards of a century ago, Lind,³ writing of the equipment of a fleet, mentions as part of it men 'picked from the street or prisons.' The careful attention now paid to hygiene in the Royal Navy renders this observation quite inapplicable to that service at the present day. The seamen in the merchant service are, as regards the source from which they are drawn, a very variable body. The sea is too often the last resort of the idle, the careless, and the ne'er-do-well. Small matter for wonder, then, if the crews often present a poor appearance. And few among them really merit the title of 'able-bodied seamen.'

After his entering on sea life, the status of the merchant sailor (boy or man) is not likely to be improved. He is cut off from the society of all but his shipmates—perhaps six or eight in number—during the most of his life, and subjected to no improving agencies other than that of maritime discipline, often of a coarse and brutal kind. He wears no uniform, but provides himself with such clothing as his means and tastes prompt. He undergoes no hygienic exercises other than those of his regular work. These and other circumstances combine to render merchant seamen a heterogeneous body, without organisation and with little *esprit de corps*. In the service of the Honourable East India Company, the life of the sailor was very different from what it is in the merchant service now. Their crews were nearly four times the present number; they wore uniform; they were well looked after; and their life was indeed that of the ideal 'Jolly Jack Tar.'

Lord Brassey⁴ states that the consuls answer the question 'Has the British seaman deteriorated?' in the affirmative; whereas official witnesses and owners of steamships speak in favour of the present status of our mariners. Their wages are higher than what are given in other countries, but the value got in return is *less*. Among the faults complained of are intemperance, insubordination (especially in large ships), ignorance of duties (steam injures seamanship), thieving, desertion, and venereal disease. Ten per cent. of the A.B.s shipped in the Port of London are foreigners, because English are not obtainable. English mercantile seamen are inferior to Swedes, Norwegians, and Germans. The Swedes have a system of apprenticeship by which, according to the testimony of one witness (Captain Murray), they 'cut out' the English system in every direction.

On the whole, the author considers the charge of deterioration not proven.

The *physique* of the seaman should be good. With regard to *height*, Wilson properly observes that tallness, apart from good development, is no

¹ *Report on the Hygienic Condition of the Mercantile Marine*, by Harry Leach. (*British Medical Journal*, 1867.)

² *History of Merchant Shipping*.

³ Lind, *On Seamen*, A.D. 1774.

⁴ *The British Navy*, by Sir T. Brassey, K.C.B. London, 1888.

advantage, and often leads to awkward weakness. On the other hand, this characteristic, with general good development, is a special advantage in the performance of duties aloft. Wilson, in an aggregate of 253 men (U.S. Navy), found the mean height 67.05 in., the tallest being 74, and the shortest 58 in.

In the Royal Navy¹ of this country, artificers over 18 years of age when first entered are not to be less than 5 ft. 4 in. in height, with a chest measurement of at least 32 in. For stokers, the same standard of height is required, with the following chest measurements:—

Between 18 and 19	not less than 32 in.
" 19 " 20	not less than 33 in.
Over 20	not less than 34 in.

The following defects disqualify for admission into the Navy.² Persons of whatever class or age, who are found to be labouring under any of the under-mentioned physical defects or infirmities, are to be considered unfit for entry into Her Majesty's service:—

(a) A weak constitution, imperfect development, or important malformation, or physical weakness, either hereditary or acquired.

(b) Skin disease, unless temporary or trivial, extensive marks of cupping, leeching, blistering, or of issues. . . .

(c) Malformation of the head, deformity from fracture or depression of the bones of the skull, impaired intellect, epilepsy, paralysis, or impediment of speech.

(d) Blindness or defective vision, imperfect perception of colours, fistula lachrymalis, or any chronic disease of the eyes or eyelids (full normal vision is required as determined by Snellen's tests, each eye being separately examined).

(e) Impaired hearing, discharge from or disease of one or both ears.

(f) Disease of the bones or cartilages of the nose, and nasal polypus.

(g) Disease of the throat, palate, or tonsils, unhealthy gums, scrofulous disease of the glands of the throat or neck, external cicatrices from scrofula or suicidal wounds.

Seven teeth deficient or defective (unless special authority is obtained from the Admiralty), or if the biting or grinding capacity be seriously impaired owing to a smaller number of unsound teeth; for instance, three or four incisors or four molars in the same jaw.

Beyond the above, no exact rule with respect to defective teeth can be laid down, but the examining officer should take into account the condition of the teeth generally and the probability of their lasting.

(h) Functional or organic disease of the heart or blood-vessels, deformity or contraction of the chest, phthisis, hæmoptysis, bronchitis, asthma, dyspnoea, aphonia, chronic cough, or any symptom of lung disease or tendency thereto.

(i) Undue swelling or distension of the abdomen, disease of the liver, spleen, or kidneys, hernia or tendency thereto from weakness of the abdominal rings, stricture of the urethra, incontinence of urine, syphilis, or gonorrhoea.

(j) The non-descent of one or both testicles, hydrocele, varicocele, or any other serious defect or malformation of the genital organs.

(k) Fistula or fissure of anus, hæmorrhoids, or any disease of the stomach or bowels.

(l) Paralysis, weakness, impaired motion, or deformity of the upper or lower extremities from whatever cause—a varicose state of the veins, especially of the leg—distortion or malformation of the hands or feet, fingers or toes.

(m) Distortion of the spine, of the bones, of the chest or pelvis from injury, disease, or constitutional defect.

The instructions for the government of medical officers in the physical examination of applicants for engagement in the United States Navy is given by Dr. Turner.³

¹ *Admiralty Instructions*, 1893, Article No. 846.

² *Ibid.*, article No. 1,154.

³ Art. 'Hygiene of the Naval and Mercantile Marine,' Buck's *Hygiene*.

Age.—No man above fifty years of age is admissible into the service of the Royal Navy, except under express authority. Lind, writing in 1774, says: 'When there is choice of men the captains generally prefer the most able-bodied raw young fellows,' but indicates the superiority of hardened veterans under the trials of a hot climate, &c. Wilson correctly considers youthfulness an advantage in a new recruit on account of his greater facility for accommodating himself to the circumstances of sea life than is possible for an adult.

The physical qualifications of the man-of-war's man, summarised as follows by Macdonald,¹ may be accepted as equally applicable to the seaman of the navy of commerce:—'The duties of the man-of-war's man are such as call for a light, active, and muscular frame. His physical condition should approximate as nearly as possible to that of the athlete, and the nature of his training should tend to bring him into that condition. His hands should be strong and powerful, yet so thoroughly taught that they may be capable of executing the very finest work. His arms should have their muscular development without being redundant, and the same may be said of his legs. His eyes should be clear, and the sight good. The nervous system should be so well balanced that no situation of peril should easily affect it.'

The physique of the *fisherman* affords a favourable contrast with that of the merchant seaman. Those of Cullercoats are in general burly, fine-looking fellows, of ruddy complexion, and remarkably clear-skinned, as artists well know. Theirs is a very old-established community. Inter-marriage is very common among them; indeed, these fishermen seldom go out of the village or trade to seek wives. This, together with exposure at nights in their open cobbles, alternating with overcrowding at home (many of them with their families occupying a single room), has its effect, for phthisis is one of the diseases from which they suffer. They are, as a rule, steady and industrious, and have plenty of good food.

The physique of the Scotch herring-fishermen is also good. The men are well-built, ruddy, clear-complexioned, occasionally fine-looking, and with bushy beards. They say their wives and children are very healthy. The men generally marry among their own kind, 'as it doesn't suit a fisherman to have a wife who cannot help him' (in bait-gathering, baiting, &c.). Many of the men on the Scotch herring-boats are known as 'Dalesmen.' These are farm labourers (? from the dales) who take to fishing in the season.

Tests of Fitness for Service.—The physical examination to be undergone by everyone before admission into the Royal Navy is indicated by the foregoing list of requirements for certain men, and the disqualifying defects for all. There is no corresponding examination for admission into the merchant service. The Board of Trade have instituted an examination as to ability to distinguish colours,² 'open to any person serving or about to serve in the mercantile marine,' for admission to which a fee of one shilling is charged. But this is evidently intended for masters and mates only, and is not compulsory even on them, for under Regulation 48 a candidate 'unable to pass the colour test will, notwithstanding, be permitted to proceed with the examination in navigation, &c., for the certificate of the higher grade.' Under Regulation 48, 'If he fails it will be open to him to be examined again in colours as often as he pleases, on payment of the fee of one shilling at each fresh attempt.' This would appear to be a very faulty regulation, for if a man is once colour-blind he is always so, and

¹ *Naval Hygiene.*

² *Regulations relating to the Examinations of Masters and Mates, A.D. 1888.*

having failed at one examination cannot be expected to recover his position at any subsequent one.

It is much to be desired that all intending merchant seamen were tested in colour perception before being allowed to enter on duty. There can be no doubt that such a course would lead to the prevention of many collisions, which too often ensue from failure to recognise accurately the different signals used in navigation.

The *hearing* of seamen as a qualification for duty is second only to their sense of sight.

The *health* of seamen in various capacities, and the diseases to which they are liable, will be considered elsewhere (pp. 574, &c.).

Sanitary Regulation and Supervision.—These, in the case of the sailor of the Royal Navies of England, France, and other countries, are carefully seen to. As much can scarcely be said for merchant seamen. Thus, in the former case, re-vaccination is strictly carried out. In the case of unsuccessful re-vaccination of men or boys entering the service of the British Royal Navy, it is the duty of the medical officer invariably to perform the operation a second time. On merchant vessels there is no such regulation.

The discovery of cases of small-pox on such vessels on their arrival in British ports leads to attempts to induce the crews to consent to this means of protection. The men may, and frequently do, refuse their consent, and after setting out from port sicken with the disease. Space does not allow to follow out in detail the numerous ways in which the general hygienic care of seamen in the merchant service compares most unfavourably with that of those in the Royal Navy.

Food

The subject of food at sea is of importance to six different kinds of seafaring persons—viz. the *crews* of the mercantile, fishing, and fighting navies, and the *passengers*, including adults, children, and the sick.

Importance of Good Food to the Sailor, and Results of Defective Feeding.—The true economy and importance of providing the sailor with plenty of good food should be self-evident and its advocacy unnecessary. Yet the want of sufficient attention to this matter has been commented on by medical officers of ships from the days of Lind and Blane until the present time. Among the modern writers on this matter the name of Rattray may be specially mentioned¹ as one who has clearly pointed the errors to be avoided, the demands to be supplied, and modifications desirable, under varying circumstances of ship-life in port and at sea, in temperate and in tropical climates. The occasional and even frequent recurrence of scurvy among crews proves the neglect of proper and well-recognised preventives. This prevalence in the mercantile navy, its history in the 'Dreadnought' hospital, and its effect on our merchant seamen, are well shown by Barnes.² The too great use of salt meat as a cause of digestive ailments has been pointed out by many authors. Liebig states that the brine changes the composition of the flesh, separating the albumen. Long-kept salt meat (especially beef)

¹ *The Disting of Seamen.* From the Statistical Reports of the Health of the Navy (Royal), 1867. 'The Influence of Diet and Climate on Health and Disease as indicated by Weight.' *Ibid.* for 1866.

² *On the Occurrence of Scurvy in the Mercantile Navy*, by Robert Barnes, M.D. (In Report of Medical Officer to Privy Council, 1863.)

becomes hard, indigestible, and innutritious, as is well known. Continued diet of salt provisions is disrelished by sailors. De Méricourt states that the issue of salt beef has been discontinued in the French Navy. Sometimes the imperfect preservation of food on shipboard renders it as loathsome and harmful, or more so, than its over-pickling. The writer has noticed junks of putrid beef and pork in the pickle-tubs of ships, where the brine had been either too weak originally or too long in use. The so-called preserved meats in use on shipboard occasionally undergo fermentative or other changes which render them highly poisonous. Instances of this are quoted by Fonssagrives.¹

The need of a diet adapted to the requirements of climate is felt more by sailors than by any other persons; for they travel from tropical to temperate or frigid zones continually. The importance of this was understood by Lind, who laid down specific instructions for different voyages. Notwithstanding all that has been said as to the necessity of suitable dietaries for the various climates, 'but little has been practically effected' (Macdonald). Rattray, in the article on 'Dieting of Seamen' already referred to, attributes the improved health of the Navy more to changes in other sanitary respects than that of food. He says: 'Ulcer and dysentery, at least, and other ailments that often directly or indirectly result from peculiarities in provisioning, and have a distinct scorbutic alliance, are still frequent at sea.'

Quality, Examination, and Testing of Food at Sea.—The quality of food of all kinds supplied to ships should be subjected to close supervision. Beef, pork, &c., should be well selected (so as to conform to the characters described on p. 496 *et seq.*, Vol. I.), and should be properly pickled. Frequently the pickle is defective, sometimes being too salty and sometimes the reverse, so as to let the meat become putrefied. The Merchant Shipping Act of 1892 provides that in the case of ships trading or going from any port of the United Kingdom through the Suez Canal, or round the Cape of Good Hope or Cape Horn, an officer of the Board of Trade shall, before shipment whenever practicable, inspect the barrels of beef and pork, preserved meat and vegetables in tins, and the casks of flour or biscuits intended for the use of the crews of such ships, and, if satisfied they are fit for such use, certify the same accordingly. He may also proceed on board a vessel to ascertain whether the stores and water provided have been duly inspected, or, if not, whether they are of a quality fit for the use of the crews of such ships. If he finds the same not to have been inspected, and deficient in quality, he is required to detain the ship until such defects are remedied to his satisfaction.

Rules for the inspection of provisions and water under this Act, by the Board of Trade, and dated March 11, 1893, define the conditions of inspection, and provide for the inspection of surplus stores left over after a previous voyage, and for turning out the contents of all casks of wet provisions among such surplus stores. Other provisions of these rules relate to notice being given to the inspector for the inspection of stores, and for supplying to him a list of the stores. The requisite condition of beef, pork, preserved meats and vegetables, vegetables in tins, flour and biscuits, is defined. Briefly stated, animal food is to be sweet, and properly packed, and pickled in pickle of full strength; vegetables are to be fresh and sound, properly preserved, and in strong and suitable tins. Flour is to be of fine grade, milled from fully matured, good sound wheat, containing a proper proportion of nutritious matter, and packed in suitable casks or tanks. Biscuits are to be thoroughly baked and dried, and made of fully matured

¹ *Hygiène Navale*. ('Aliments altérés.')

wheat flour, containing a proper proportion of nutritious matter. When stored in tanks, these are to be thoroughly cleansed, lined with fresh lime, and dried before being refilled. The water left in the ship's tanks from the former voyage must all be completely emptied, and the tanks must be thoroughly cleansed and refilled with good fresh water. The inspector is empowered to require that all stores deficient in quality shall be landed, and he is required to grant a certificate if he is satisfied that he has seen all the stores intended for the particular voyage, and that they are of a quality fit for the use of the crew. One of the rules provides for the exemption from inspection of the stores of a ship trading from one port to another in the United Kingdom, and which have not been opened.

The Merchant Seamen (Provisions) Bill, 1898, is intended further to amend the law in this matter.

De Méricourt¹ testifies to the excellence of the bread supplied to the seamen in the French Navy. The best wheat only is selected; it is freshly ground, must be of good taste and smell, and contain 8 per cent. of dry gluten. It must also be free from beyond 20 per cent. of husk, bran, or flour of inferior quality; must have not more than 18, or less than 10, per cent. of water. The flour is analysed by whatever means the authorities think fit to adopt, and the ovens in which the bread is baked are subject to inspection and supervision.

The biscuit in the British Royal Navy is manufactured from a meal consisting of flour and middlings made from raw wheat (Macdonald). 'The baking should be so conducted as to give the biscuit a uniform yellowish-white. The gluten, if too large in amount or unequally distributed, makes a long-baked biscuit flinty and difficult to masticate. Biscuit may be examined microscopically and chemically for adulteration, &c. Alum or sulphate of copper are sometimes used to whiten dark-coloured flours. Old kept biscuit is liable to be affected by worms and insects, of which the weevil (*Calandra granaria*) and the larva of the chocolate moth (*Ephestia elutella*) are the most common. Sourness of soft bread indicates usually either over-'steeping' of the dough, defects in the flour, or fermentation of the bread, or bad yeast.

For the examination of flour, see Vol. I. p. 461.

Ordinary beans, according to Dr. Turner,² are unfit for food, if after immersion for some hours in water they do not sprout. The same remark applies to peas. The former is an item in the ration of the American, and the latter of the British, War Navy.

Preservation of Foods at Sea

The best means of providing fresh food, animal or vegetable, is keeping it alive or rearing it to use as it is wanted. This is, of course, practicable only to a very limited extent on shipboard. Cattle, sheep, pigs, fowls, eggs, milk, and certain anti-scorbutic and other vegetables may be so kept or produced, but the expense is prohibitory of their general use. Wilson³ recommends small tanks and gardens on board ship for turnips, cresses, radish, &c.

Among the processes for preserving food of different kinds, the following are mentioned by Wilson:—Salting, desiccation, smoking, immersion in oil (for fish, &c.), immersion in vinegar (for animal and vegetable foods), immersion in molasses (for potatoes), heating and seclusion from the atmosphere (as in tinned foods, soups, fruits, &c.), refrigeration.

Salting diminishes the nutritive value of meat as previously stated.

¹ *Rapport sur le Progrès de l'Hygiène Navale.*

² *Buck's Hygiène.*

³ *Naval Hygiène.*

Drying is not in general use as a means of preserving animal foods in this country. It is the process by which the pemmican of the American hunter is prepared. Desiccation by pressure has been successfully applied by Chollet and Masson for the preservation of fresh vegetables, which are said to regain their original bulk, and freshness after immersion for thirty or forty minutes in tepid, or from six to eight hours in cold, water.¹

De Méricourt considers that under this process the vegetables lose their anti-scorbutic qualities. He is in favour of Fastier's process for preserving meat as adopted for the French Navy, which consists in plunging tins of the meat into water at a temperature of 262° F. (effected by dissolving certain substances in the water so as to raise the boiling-point). The air is driven out of the tins by the steam created by this high temperature, and the tins are then closed and soldered. This method resembles the ordinary process for preserving the tinned meats so largely consumed in this country.

Storage of Food on Shipboard.—One defect on board many merchant vessels is the want of proper places in which to store provisions. Hence these are often exposed to unwholesome exhalations. In timber ships occasionally the vapour from the cargo affects the food with an unpleasant flavour. In the 'weekly boats' the food of the crew is kept in the forecabin exposed to their exhalations, &c. In such cases lockers are not always provided. In steamers making longer voyages one occasionally finds the bread-store opening into the seamen's quarters, or the crew's provisions kept there. Bread and other provisions, whilst unbroached, are, in well-arranged vessels, lodged in the 'lazarette,' the opened casks or tins, &c., of food being kept in the store-room. An example of a pantry in a faulty position is given in fig. 18.

Cooking and Apparatus

These on shipboard are not generally of first-class character, and on small vessels are very poor indeed. On fishing boats the 'provider' and cook is generally a boy, who learns his vocation as best he can, and usually with very scanty apparatus. In Norwegian schooners the galley is perhaps inside the deck-house. Oftener it adjoins the deck-house in small ships, or stands separately; and in these cases may be covered in, but is often not so, causing the exercise of the culinary art in rough weather to be a matter of no small difficulty. In one instance, which may serve as a specimen, noted by the writer, the galley of a brigantine was merely a stove having an oven behind and near the deck, and a small boiler above and behind, and a flat plate over the fire for boiling potatoes, &c., the whole being enclosed in a metal case 3 feet 9 inches high \times 3 feet 3 inches square superficially; so that the cook had no shelter. The pans and general arrangement were very defective. Comfortable covered galleys are met with on some of the smaller Swedish and Norwegian vessels trading to the Tyne. A good galley for a merchant ship of twenty hands or so should be about 12 feet in length by 7 feet in breadth, well ventilated, entirely clear of crew-space, and provided with a separate room for the cook, but not opening into it.

Rations

On next page is given the scale of victualling in the British Royal Navy.² (When women and children are carried, they are to be victualled in accordance with the scale for victualling Her Majesty's troopships.)

¹ Fonssagrives.

² *Queen's Regulations and Admiralty Instructions*, 1893, Appendix xxi. pp. 987, &c.

—	When to be issued	Articles	—	Seamen	
				Officers, crew, and others at a seaman's full allowance	Super-numeraries at two-thirds of a seaman's allowance
1	Daily . . .	Biscuit . . .	lb.	1½	1
2		or			
3		Soft bread . . .	"	1½	1
4		Spirit . . .	pint	1½	1½
5		Sugar . . .	oz.	2	1½
6	Weekly . . .	Chocolate—Ordinary . . .	"	1	1
7		—Soluble . . .	"	1-2	1
8		Tea . . .	"	1	1
9		Oatmeal . . .	"	3	2
10		Mustard . . .	"	1	1
11	Daily when procurable . . .	Pepper . . .	"	1	1
12		Vinegar . . .	pint	1	1
13		Fresh meat . . .	lb.	1	1
14		Vegetables . . .	"	1	1
<i>When fresh provisions cannot be secured :—</i>					
13	Every other day . . .	Salt pork . . .	"	1	1
14		Split peas . . .	"	1	1
15		Celery seed . . .	½ oz. to every 8 lbs. of split peas put into the coppers		
16	On one alternate day . . .	Salt beef . . .	lb.	1	1
17		Flour . . .	oz.	9	6
18		Suet . . .	"	1	1
19		Raisins . . .	"	1½	1
20		Preserved meat . . .	lb.	1	1
21	On the other alternate day . . .	with either			
22		(1) Preserved potato . . .	oz.	4	2½
23		or			
24		(2) Rice . . .	"	4	2½
25		or			
26		(3) Preserved potato . . .	"	2	1½
27		and			
28		(4) Rice . . .	"	2	1½
29		or			
30		Flour . . .	"	9	6
31		Suet . . .	"	1	1
32		Raisins . . .	"	1½	1

Scale of Substitutes¹

In case it should be necessary to issue substitutes for any of the articles in this scale of victualling, the following proportion is to be adopted, viz. :—

Biscuit . . .	1 pound	are to be considered equal to each other
Flour . . .	1 "	
Rice . . .	1 "	
Wine . . .	½ pint	Do. do.
Spirit . . .	1 gill	
Porter . . .	1 pint	
Coffee . . .	1 oz.	Do. do.
Cocoa . . .	1 "	
Chocolate, Ordinary, 1 "	1 "	
Soluble, 1-2 "	1 "	
Tea " . . .	1 "	

The following, when issued with Meat Rations, are to be considered equal to each other :—

1	Split peas . . .	1 pound
	Peas whole . . .	1 pint
	Flour . . .	1 pound
	Calavances . . .	1 pint
	Dholl . . .	1 pint
2	Rice . . .	2 pound
	Vegetables . . .	1 pound
	Compressed mixed vegs. . .	1 oz.
3	Preserved potato . . .	2 ozs.
	Oatmeal . . .	1 pint or 2 ozs.
	Split peas . . .	1 pound

When the men desire it, ½ lb. of flour may be issued in lieu of ½ lb. of biscuit, and ships proceeding to sea are to fill up on this basis.

Articles 1,729 and 1,780 of the Admiralty Instructions provide that no person is to receive a spirit ration in kind until he is twenty years of age. The spirit is to be mixed and issued every day upon deck with three parts of water to one of spirit, and is to be served out at dinner time only.

¹ *Queen's Regulations and Admiralty Instructions, 1893, Appendix xxi. p. 993.*

Scale of Rations per Man																						
Daily																						
Days of the Week	Salt Beef	Flour	Buck	Bacon	Salt Pork	Split Peas	Preserved Meat	Compressed Mixed Veggies	With Fresh Meat an additional 4 oz. of Bread or 3 oz. of Butter is to be issued to each Man		Biscuits	Fresh Bread	Rice	Porter	Preserved Potatoes (uncooked)	Sugar (unrefined)	Tea	Vinegar	Mustard	Pickles (of various descriptions)	Pepper (ground)	Salt
	oz.	lb.	oz.	lb.	oz.	lb.	oz.	lb.	oz.	lb.	oz.	lb.	oz.	pt.	oz.	oz.	oz.	oz.	pt.	oz.	oz.	oz.
	oz.	lb.	oz.	lb.	oz.	lb.	oz.	lb.	oz.	lb.	oz.	lb.	oz.	pt.	oz.	oz.	oz.	oz.	pt.	oz.	oz.	oz.
Sunday	12	6	1	2	12	6	12	1	12	1	12	1	—	—	2	2	2	—	—	—	—	—
Monday	12	6	1	2	12	6	12	1	12	1	12	1	—	—	2	2	2	—	—	—	—	—
Tuesday	12	6	1	2	12	6	12	1	12	1	12	1	—	—	2	2	2	—	—	—	—	—
Wednesday	12	6	1	2	12	6	12	1	12	1	12	1	—	—	2	2	2	—	—	—	—	—
Thursday	12	6	1	2	12	6	12	1	12	1	12	1	—	—	2	2	2	—	—	—	—	—
Friday	12	6	1	2	12	6	12	1	12	1	12	1	—	—	2	2	2	—	—	—	—	—
Saturday	12	6	1	2	12	6	12	1	12	1	12	1	—	—	2	2	2	—	—	—	—	—

Scale of Rations per Woman																						
Days of the Week	Salt Beef	Flour	Buck	Bacon	Salt Pork	Split Peas	Preserved Meat	Compressed Mixed Veggies	With Fresh Meat an additional 4 oz. of Bread or 3 oz. of Butter is to be issued to each Woman		Biscuits	Fresh Bread	Rice	Porter	Preserved Potatoes (uncooked)	Sugar (unrefined)	Tea	Vinegar	Mustard	Pickles (of various descriptions)	Pepper (ground)	Salt
	oz.	lb.	oz.	lb.	oz.	lb.	oz.	lb.	oz.	lb.	oz.	lb.	oz.	pt.	oz.	oz.	oz.	oz.	pt.	oz.	oz.	oz.
	oz.	lb.	oz.	lb.	oz.	lb.	oz.	lb.	oz.	lb.	oz.	lb.	oz.	pt.	oz.	oz.	oz.	oz.	pt.	oz.	oz.	oz.
Sunday	8	6	1	2	8	6	8	1	8	1	8	1	—	—	2	2	2	—	—	—	—	—
Monday	8	6	1	2	8	6	8	1	8	1	8	1	—	—	2	2	2	—	—	—	—	—
Tuesday	8	6	1	2	8	6	8	1	8	1	8	1	—	—	2	2	2	—	—	—	—	—
Wednesday	8	6	1	2	8	6	8	1	8	1	8	1	—	—	2	2	2	—	—	—	—	—
Thursday	8	6	1	2	8	6	8	1	8	1	8	1	—	—	2	2	2	—	—	—	—	—
Friday	8	6	1	2	8	6	8	1	8	1	8	1	—	—	2	2	2	—	—	—	—	—
Saturday	8	6	1	2	8	6	8	1	8	1	8	1	—	—	2	2	2	—	—	—	—	—

¹ Queen's Regulations and Admiralty Instructions, 1898, Appendix xxi. p. 990.

² Omitted from Regulation of 1893.

Note.—The rations for young persons of different ages are fixed so as not to include stimulants. Fresh meat and fresh vegetables are to be issued in port whenever practicable. Invalids are to have fresh bread every day, and live stock (not pigs) is to be provided liberally for them. Water is to be liberally allowed, the minimum for each individual embarked being six pints when out of the tropics and one gallon within the tropics. (For further particulars, Substitutes, Medical Comforts, &c., see 'Queen's Regulations, 1893,' pp. 992–95.)

For an interesting account of the rations of the British Royal Navy from 1720 onwards, and of the British, Dutch, and French Navies in 1871, the reader is referred to Blyth's 'Dictionary of Hygiene' (Article 'Hygiene'). Those of the French,¹ Portuguese, and Belgian War Navies are also described by Fonssagrives; Turner (Buck's 'Hygiene') gives a comparison of the different rations issued to the United States men-of-war's-men, at sea and in port; together with a statement of the tissue-forming and heat-producing equivalents of each article. Wilson treats of the same dietary. Rattray, in the article before quoted, gives some valuable information as to the dietary of the British Royal Navy in 1873 (in port and out of port).

In British merchant ships (foreign-going) the average food allowance is 1 lb. of bread every day, $1\frac{1}{2}$ lb. of beef, and $1\frac{1}{4}$ lb. of pork on alternate days, and $\frac{1}{2}$ lb. flour and $\frac{1}{4}$ lb. peas on alternate days. In coasting vessels, where the work is hard and constant, the allowance is usually unlimited. Lindsay² comments on the large amount of suffering from paucity of provisions and water, the latter being commonly issued at the rate of three quarts per person per week. In passenger ships the scales of dietary for passengers are of two classes, according to the length of the voyage, as follows:

PROVISIONS³

Weekly, per Statute Adult

	Scale A. For voyages not exceeding 84 days for sailing vessels, or 50 days for steamers	Scale B. For voyages exceed- ing 84 days for sailing vessels, or 50 days for steamers
	Lbs. oz.	Lbs. oz.
Bread or bisouit, not inferior in quality to		
Navy bisouit	3 8	3 8
Wheaten flour	1 0	2 0
Oatmeal	1 8	1 0
Rice	1 8	0 8
Peas	1 8	1 8
Potatoes	2 0	2 0
Beef	1 4	1 4
Pork	1 0	1 0
Tea	0 2	0 2
Sugar	1 0	1 0
Salt	0 2	0 2
Mustard	0 0 $\frac{1}{2}$	0 0 $\frac{1}{2}$
Black or white pepper, ground	0 0 $\frac{1}{2}$	0 0 $\frac{1}{2}$
Vinegar	One gill	One gill
Lime juice	—	0 6
Preserved meat	—	1 0
Suet	—	0 6
Raisins	—	0 8
Butter	—	0 4

N.B.—Full issue of lime juice imperative only in the tropics, 26 & 27 Vic. c. 51, s. 9; and at other times at the discretion of the medical officer.

¹ Levy, *Traité d'Hygiène*, tome ii., gives the rations of the French Navy as fixed in 1874.

² *Merchant Shipping and Ancient Commerce*.

³ *Board of Trade Notice to Passengers*, 1887, p. 38.

Substitutions.

1 lb. of preserved meat	for	1 lb. of salt pork or beef.
1 lb. of flour or of bread or biscuit, or } $\frac{1}{2}$ lb. of beef or pork	"	1 $\frac{1}{2}$ lb. of oatmeal or 1 lb. of rice, or 1 lb. of peas.
1 lb. of rice	"	1 $\frac{1}{2}$ lb. of oatmeal, or <i>vice versa</i> .
$\frac{1}{2}$ lb. of preserved potatoes	"	1 lb. of potatoes.
10 ozs. of currants	"	8 ozs. of raisins.
8 $\frac{1}{2}$ ozs. of cocoa or coffee, roasted or } ground	"	2 ozs. of tea.
$\frac{1}{2}$ lb. treacle	"	$\frac{1}{2}$ lb. of sugar.
1 gill of mixed pickles	"	1 gill vinegar.

In case of failure to supply issues of good and wholesome provisions in accordance with the above scales, the master is liable to a penalty not exceeding fifty pounds.¹ The provisions for the crews of passenger ships are not to be inferior to those of the passengers.²

The American dietary scale for passengers is given by Turner (Buck's 'Hygiene and Public Health'), together with the rations of the crews of passenger ships.

As respects the general defects of diet scales for seamen, Rattray gives as his opinion that

1. They are too unvarying in all latitudes, tropical and temperate.
2. The highly-salted beef and pork, and scanty supply of vegetable matter are objectionable, especially in the tropics.
3. The same diet is given in warm as in cold regions, instead of one less nitrogenous and stimulating, and more vegetable.
4. The spirit ration, especially in the tropics, is unnecessary and injurious.

Rattray advocates the use of preserved meat, &c. He proposes a naval dietary for (1) temperate and (2) tropical climates, (3) for use at sea, and (4) in harbour. These dietaries will well repay perusal.

There is much necessity for having the rations of coasting as well as ocean-going ships fixed by law.

Quality of Provisions, &c., supplied to Merchant Seamen

'Any three or more of the crew of a British ship may complain to any officer in command of any of Her Majesty's ships, or any British Consular Officer, &c., that the provisions or water for the use of the crew are bad.' The person complained to may, after examining the food and finding it defective, signify the same to the master of the ship, who shall be liable to a penalty of twenty pounds in case of failure to provide proper provisions, &c., in lieu thereof.³

The principle of giving pay in place of food on board of coasters and 'weekly boats' is bad, inasmuch as the men are not likely to provide themselves with proper or sufficient food, and they have not proper storage for their provisions.

WATER AND BEVERAGES

Sources and Quality.—In port, ships are usually furnished with water by 'water boats' fitted for the purpose with tanks, from which the supply is pumped to the receptacles of the vessel requiring it. Under proper arrangements no objection can be raised to this. But in cases of enteric fever, &c., it is sometimes difficult to trace the disease to its origin owing to the impossibility of ascertaining the source from which the water-boat derived its

¹ *Notice to Passengers*, p. 63.

² *Ibid.* p. 30.

³ *Boyd's Merchant Shipping Laws*, 1876, p. 196.

supply. The boats themselves are occasionally found to be dirty, or have leaking decks.

We have known the drinking-water for a ship to have been pumped directly from the river in a foreign port. On long voyages vessels have often to send ashore for fresh water from springs, streams, &c. This is done by filling casks, or even the ship's boats, which should first be lined with canvas as described by Macdonald. In canal boats the water is frequently drawn directly from the canals, the boatmen being too lazy to lay in a stock before starting.¹

Fishing boats and other small craft are not unfrequently supplied with water from objectionable sources, perhaps owing to unwillingness to pay for a good supply. Instances have come under our observation where the crews of such boats have complained of the water causing sickness and diarrhoea. In some ships visiting the Port of Tyne the water has been found to yield a plentiful deposit of clay and other matter, as though drawn from a dirty place, or from a stream in flood. Large vessels provide most of their water by distillation. This process, now so familiar and simple, was, as practised prior to the time of Lind, mysterious and complex. Different methods, introduced by different persons, in which the sea-water to be distilled was mixed with such ingredients as *Lapis infernalis*, calcined bones, soap lees, chalk, lime, ashes, &c., had in turn been tried and found unsatisfactory. The fact had long been known that the vapour expelled from sea-water by the aid of heat contained no saline matters; but the water when distilled had at first a disagreeable taste, believed to be due to bitumen, which the ingredients above named were intended to remove.² Lind showed this idea to be 'a mere imagination,' and in a paper read before the Royal Society in 1762 demonstrated that pure water might be distilled from sea-water alone. Ten years later Dr. Irving received a grant of 5,000*l.* from Parliament for 'the discovery of an easy and practicable method of making sea-water fresh and wholesome,' which appears to have been merely a modification of the method of Lind. Four years after Lind's discovery, Dr. Poissonnière, a French physician, had also a pension settled on him on account of the same.³ The history of this remarkable discovery, and the steps which led to it, are fully described by Fonssagrives,⁴ who claims the credit of the invention for his compatriot Gauthier, a physician of Nantes, who in 1717 made trial of a distilling apparatus at sea. His process caused great heat and risk of fire, imparted a disagreeable flavour to the distillate, and, having other disadvantages, did not give satisfaction. It was soon abandoned. Fonssagrives fails to show in what respects the method of Gauthier was a forestalment of the principle of which we maintain Lind to have been the discoverer—viz. that of the distillation of sea-water alone, and the condensation of its vapour into a pure and palatable fluid.

For an account and illustrations of various apparatus in use for distillation from the time of Gauthier onward, the reader is referred to the work of Fonssagrives.

Passenger ships may proceed on their voyage with only half the quantity

¹ *Annual Report of the Local Government Board*, 1887-88.

² Saint Basil relates in his Fourth Homily how, being shipwrecked on an island where water was not procurable, he showed his companions in misfortune the means of collecting a sufficient quantity to quench their thirst; sea-water was heated in an iron basin and the vapour became condensed on sponges, which yielded sweet water when pressed (Fonssagrives).

³ Lind on Seamen, 1774.

⁴ *Traité d'Hygiène Navale*.



of pure water required by the Act, provided that there be on board an efficient and competent distilling apparatus under proper management.¹

The Board of Trade regulations as to the survey of steamships carrying passengers² state that the distilling apparatus of emigrant ships should, with certain exceptions, be taken to pieces every voyage and tested. 'The water should be cold, pure, and fit to drink immediately after it is drawn off from the filter. No distilling apparatus should be passed unless fitted with a suitable-sized filter, charged with animal charcoal.' A list of patent distilling apparatus, approved by the Board of Trade, including fifteen kinds of which Normandy's stands at the head, is given in the regulations above-mentioned.

A condensing apparatus of the first class will distil as much as 800 gallons in ten hours.

Examination and Testing of Water.—For details on these matters the reader is referred to Vol. I., p. 274 *et seq.*

Preservation and Storage.—In large ships water is stored in galvanised iron tanks, holding often 600 gallons or more each. In small ships casks are still in common use. These are generally fixed on the decks of schooners, &c., and are apt to be washed overboard in stormy weather. Tanks are stored in the holds of large ships, and in various parts of small vessels; thus one frequently finds them beneath the cabin floors, or in any convenient corner. In passenger ships, when casks are used they are required by statute to be 'sweet and tight, of sufficient strength, and, if of wood, properly charred inside, and . . . not capable of containing more than three hundred gallons each. The staves of water casks shall not be made of fir, pine, or soft wood.' Breach of these requirements renders the offender liable to a penalty of fifty pounds.

It is stated by Medlock, on the authority of Turner,³ that a coil of iron wire in contact with water for twelve hours will remove or convert from a soluble to an insoluble form all traces of organic matter, rendering it capable of removal by filtration. Dr. Wilson states that in iron tanks the contained vegetable matter of water soon decomposes, after which the water quickly becomes good and remains so. Iron tanks also decompose the salts of brackish water, and thus improve it.

Under the Passengers Acts it is provided that the supply of fresh water to passenger ships shall be sufficient to secure throughout the voyage three quarts daily to each statute adult, exclusive of the amount necessary for cooking, and that the latter shall be shipped 'after the rate of at least ten gallons for every day of the prescribed length of the voyage for every one hundred statute adults on board,'⁴ and also for the use of the crew and all other persons on board an ample supply, &c.' On passenger ships water is not allowed to be carried on the upper or 'passenger decks,' unless with the approval of the emigration officer.

Supply allowed.—The 'Queen's Regulations and Admiralty Instructions' state that water for 'troops or third-class passengers' 'is to be issued on the most liberal scale possible; and the minimum daily allowance of water is to be for each individual embarked, six pints when out of the tropics, and one gallon when within the tropics, which quantities are to suffice for all purposes.' There is not (so far as we are aware) any other regulation in the

¹ *Board of Trade Notice to Passengers*, 1887, Appendix I.

² 1892, s. 145.

³ Buck's *Hygiene*.

⁴ Passenger ships provided with proper distilling apparatus are required to store only half of this amount.

above instructions for the supply of water to persons on board ships of war. The supply to passenger ships is indicated in the preceding paragraph. On merchant ships the allowance is usually three quarts per man. Sometimes we are told that the rule is 'sufficient without waste,' or that the supply is 'unlimited.'

Purification of Water.—For information as to the methods for purifying water, the reader is referred to Vol. I., p. 253.

Beverages other than Water.—The spirit ration on board ships of war is $\frac{1}{8}$ of a pint daily. Grog is not now an allowance on merchant ships. Coffee is a favourite drink of merchant seamen and fishermen, but does not form part of the ration on board a ship of war. Tea and chocolate are, however, issued daily. Firemen use considerable quantities of oatmeal and water. According to Bourel Boncière, the stokers in the French Navy consume large quantities of acidulated water (three or four quarts in a watch), the result of which is seriously detrimental to health.

In the French Navy, Bordeaux and other light wines are used on ship-board.¹ We agree with Fonssagrives as to the desirability of introducing beer into the services afloat as a hygienic and anti-scorbutic beverage. If it were possible either to ship it in sufficient quantity, or to brew it on board, the substitution of beer for the spirit ration would be a great benefit to the seamen.

CLOTHING

Owing to the peculiarly hygrometric condition of the marine atmosphere, and the rapidity of change from one climate to another, clothing at sea is even more important in relation to health than on shore. The exposure of the sailor or fisherman to bad weather whilst on duty makes it more urgent that his garments should be a protection against wetting than is necessary in the case of most other occupations. The material of the sailor's jacket, vest, and trousers is usually woollen in temperate regions, whilst firemen wear linen or cotton outer garments. Cotton conducts heat more freely than wool. Flannel clothing is more porous than cotton or linen, but is much more hygroscopic (Pettenkofer). Waterproof tarpaulins, &c., for seamen and fishermen are among the necessities of life. The sailor is not always provided in this respect, and he is proverbially careless of himself. One great disadvantage under which he labours is that he is without proper means for drying his wet clothes. To do this in a close fore-castle where he has to sleep is to expose him to serious risk of contracting respiratory disease, rheumatism, and other ailments. Sailors are also apt to put away their recently washed underclothing in a damp state, and to wear it without having it dried—another cause of danger to health.

Fabric for Clothing.—Turner² gives the physical characters required in the material of the clothing worn in the United States War Navy.

He states that the United States Navy regulations require that the colour of all fabrics shall be blue, and that the dye shall be pure indigo, which may be detected by maceration in fuming sulphuric acid. The dense blue liquid (sulphindigotic acid) can be tested by nitric or chromic acid for indigo.

Bleaching on immersion of the cloth in solution of chlorinated lime, and permanency of colour in solution of caustic soda, are further tests of indigo; whereas with Prussian blue dyes the reverse is the case with each of these tests.

Kits and Outfits.—In the merchant navy of this country there are no

¹ Fonssagrives.

² Buck's *Hygiene*.

regulations as to the outfit of seamen. Each man provides for himself as he thinks fit, and the result is want of uniformity. Turner states that in the United States Merchant Marine it is directed by statute that every vessel is to be provided with at least one suit of woollen clothing for each seaman. Fishermen, on the other hand, are distinguished by their characteristic dress, and present a good appearance. The fishermen of Cullercoats, for example, on going out to fish at night, generally wear a jersey and an oilskin, with or without a waistcoat underneath the latter, according to season, &c. The head is protected by an oilskin sou'wester in rough weather; at other times by a cap of cloth or sealskin. The feet and legs to above the knees are encased in sea-boots, and the trousers are often made of oilskin. The underclothing is of thick wool.

The *regulation kit* for seamen in the British Royal Navy¹ is compared with that of other countries in the table on the following page.

In the list we notice with pleasure the presence of two sanitary requisites in the kit of the French man-of-war's-man—viz. toilet and tooth brushes, and would be glad if they were provided for the British sailor. The hygienic advantage of warm underclothing for seamen need not be enlarged on. An item recommended by the Surgeon-General of the United States Navy, and spoken of with favour by both Wilson and Turner, is the cholera belt, a girdle of flannel about forty inches long by four wide, to wear in warm climates, for the prevention and cure of dysentery. It is said to afford support to the abdominal walls, and to be very useful as a protection against chills, &c. It is the same as the Indian cummerbund. Fonssagrives also recommends it.

Waterproof capes would be an excellent addition to the clothing of the sailor.

The headgear consists of hats and caps. The most suitable covering for duty and comfort at present in use is undoubtedly the cap, which in the British Navy is made of blue cloth. Woven Glengarry caps and Tam o' Shanter bonnets are specially adapted for the use of sailors from the difficulty with which they are blown off in a gale of wind. Being porous, they are well ventilated and cool. They look well and are very comfortable.

The sailor of the British Navy has one pair of boots allowed him; shoes are optional. It is a matter of much importance that the covering for the sailor's feet should have soles adapted to the special life of the wearer—i.e. for use on deck and aloft. The sole should be thin and rather pliant. For warm climates the use of sandals or slippers is desirable.

Among the most important items of body clothing are the woollen vest and the jersey. The former is an almost universal article of clothing among Scandinavian sailors. From personal experience the writer can testify to the unwillingness of Norwegian and Swedish seamen admitted to hospital with fever to allow themselves to be stripped of their thick, knitted, warm, and generally dirty, woollen underclothing, to which, from long wear, they have, in almost more senses than one, become attached! The jersey is close-fitting, warm, and elastic. As an external garment of fishermen, it repels wet well, and in appearance is most picturesque.

¹ *Uniform Regulations for Men and Boys of the Fleet*, 1890.

REGULATION KIT

	British Royal Navy	United States War Navy ¹	French War Navy ²
Monkey jacket No.	1	—	—
Round jacket, cloth "	1	1	Cloak 1
Jersey "	—	1	Vareuses 2
Blue cloth pea jacket "	—	1	Pantalons de fatigue 2
Trousers, blue cloth Pairs	2	1	—
" blue satinnet "	—	1	—
Serge trousers "	2	—	—
Blue serge frock No.	2	—	—
" jumper "	2	—	Gaiters (pairs) . . 1
Drill frocks "	2 or more according to climate	—	—
Duck jumpers, with collars "	2	—	—
" trousers "	4	—	—
Working jumpers "	2	—	—
Black silk neckerchief "	2	1	—
Hat, white "	1	—	—
" felt "	—	—	1
" straw "	—	—	1
Hat cover "	1	—	—
Cap ribbons "	2	—	—
Cap, blue cloth "	2	—	—
" " " "	—	1	—
" white cover "	2	—	—
" working "	—	—	2
Flannels "	2	—	—
" over shirts "	—	2	—
" under shirts "	—	2	—
" drawers "	—	2	—
Woollen drawers "	2	—	—
Shirts, blue check "	2	—	—
" linen "	—	—	4
" swansdown "	—	—	2
Cholera belts "	2	—	—
Linen frocks "	—	2	—
" trousers "	—	2	—
White " "	—	—	2
Socks or stockings Pairs	2	—	2
" yarn "	—	2	—
Towels No.	2	—	—
Type "	1	—	—
Knife with two lanyards "	1	—	—
Shoes Pairs	—	—	1
" calfskin "	—	1	—
Boots "	1	—	—
Bed covers No.	2	—	—
Bed "	1	—	—
Blanket "	1	1	—
Mattress "	—	1	—
Tricots, cotton (or knitted cotton shirts) "	—	—	4
Duck bag "	1	—	—
Grand sac (linen) "	—	—	1
Petit sac "	—	—	1
Collars "	2	—	—
Cravats, wool "	—	—	1
" lasting "	—	—	1
Muffler "	1	—	1
Brushes (clothes, shoe) "	1	—	1
" (toilet and tooth) "	—	—	1
Scrubbing brush "	1	—	—
Comb "	1	—	1
Hood "	—	—	1
Ditty box "	1	—	—
Soap bag "	1	—	—
Optional—Waterproof coat, sou-wester, leggings, pair of shoes "	1	—	—

Among other articles of clothing to be condemned are the Norwegian soft leather jackets, or sleeved vests, lined with flannel, much worn by sea-

¹ Wilson's *Naval Hygiene*, 1879. In addition to the articles above named in the American kit, are numerous small stores (thread, ribbon, tape, pocket-handkerchiefs, brushes, &c.) which cannot well be compared with items in the British kit.

² Fonssagrives, *Hygiène Navale*, 1877.

men who can afford to pay for them. These jackets cost 25*s.* and upwards each. They are warm, wear well, and look well, even when old, but have no ventilation, and are therefore very unhealthy.

The seaman's clothing is usually kept in his bag or chest. The former is more easily carried, but is less tidy, and has no lock. The chest is often used as a table, or the sailor may sit on it, straddlewise and secure, in a tossing ship. In schooners and other small vessels the wet clothing hangs in the fore-castle, filling the air of the place with moisture.

HEALTH, DISEASE, ACCIDENT, AND DEATH AT SEA

Sickness and Mortality of Navies, &c.—The benefits of hygienic reform are nowhere shown to greater advantage than in the British Royal Navy. In former times the life of the man-of-war's-man was worth but little, for, apart from the risk to which he was exposed from any national enemy, his health was continually in great danger from disease of one kind or another. In proof of this, we may cite Dr. Guy,¹ who states that out of 961 men in Anson's fleet (A.D. 1742) there were in nine months no less than 626 deaths, 'or very nearly two out of three!' 'Even so late as 1780, Sir Gilbert Blane found that a fleet manned with between 7,000, and 8,000 men had in one year lost one in every seven' from scurvy alone. The same authority gives the strength and mortality of the English Royal Navy in 1779, from which we calculate that its rate of sickness was no less than 408 per 1,000, and its mortality 28·6 per 1,000 from all causes. Sir Gilbert Blane's tables, as quoted by Dr. Guy, give corresponding statistics for subsequent years, from which we gather that the rate of sickness in the British fleet was as follows:—

Rate of Sickness per 1,000 Strength of the British Fleet in the Under-mentioned Years.

A.D.	No. of cases of sickness per 1,000 men	A.D.	No. of cases of sickness per 1,000 men
1782	229	1799	122
1795	205	1804	76
1797	171	1805	67
1798	131	1806	64

The steady improvements in these years marks the progress of the practice of sanitation, of which the use of lime juice and the improved ventilation of ships were important features. How these returns are surpassed by the excellent results of recent times will presently appear. Levy² states that in 1598, in the Southern Seas, the fleet of De Wert had a mean annual mortality of 49·1 per cent.; that of the fleet of Admiral Lancaster (1610) was 38 per cent. (due to scurvy, &c.). It is not to be understood that these experiences were universal. Thus Cook, in 1772, lost only 1·2, and in 1778, 1·3 per cent. The rate with Parry in the expedition of 1819 was ·7, and that of 1821 was 2·1, and in 1824 was still further reduced to ·5.

¹ *Public Health.*

² *Traité d'Hygiène.*

The following are the returns of the health of the British Royal Navy at the present time :—

BRITISH ROYAL NAVY

(From the Statistical Report of the Health of the Navy for 1891.)

Average daily sick-rate	41·23 per 1,000.
Finally invalided out of the service	910, or 16·34 „
Deaths	344, or 6·17 ¹ „

Death-rate from disease alone, 4·68, and from injury alone, 1·49 per 1,000.²

The Sick List includes, *inter alia* :—

	Cases	Deaths
Small-pox	17	0
Measles	98	0
Scarlet fever	58	1
Influenza	3,527	6
Diphtheria	2	0
Simple continued fever	1,894	1
Enteric fever	206	46
Cholera (East Indies and China Stations)	50	29
Dysentery	51	1
Malarial diseases	1,559	14
Septic „	42	5
Venereal „ Syphilis, primary 3,031 }	4,525	4
„ „ secondary 1,494 }		
„ „ gonorrhœa, &c.	4,015	1
Rheumatism	2,563	4
Tubercular diseases	42	15
Diseases of nervous system	1,390	10
„ „ circulatory system	262	23
„ „ respiratory system	745	72
„ „ digestive system	6,455	15
„ „ urinary and generative system	491	5
„ „ connective tissue and skin.	8,193	1
Poisoning	31	1
Wounds and injuries, general, 172 }	9,912	79
„ „ „ local, 9,740 }		
Suicide		4

*Merchant Service of the United Kingdom.*³—The number of masters and seamen reported to the Registrar-General of Shipping and Seamen as having died in merchant vessels registered in the United Kingdom during the year 1886 was 2,249 (521 in the first, and 1,728 in the second half of the year). The information is reported principally by the masters of the vessels. The numbers and causes of deaths in steam and sailing vessels respectively are shown in the table on the next page :—

¹ N.B. *Invaliding*. To what extent does this affect published death-rate?

² For details of the above statistics as regards the different naval stations, see *Health of the Navy*, 1891.

³ Merchant Service Board of Trade (No. 325) to the House of Commons, August 4, 1888 (extracts from and calculations based thereon).

*Number and Causes of Death in Merchant Vessels registered in the United Kingdom and the Isle of Man, of Masters and Seamen, reported to the Registrar-General of Shipping and Seamen in 1886-7, and appropriated to the Year 1886.*¹

Causes of death	Vessels		
	Steam deaths	Sailing deaths	Total deaths
Yellow fever	4	38	42
Ague	6	1	7
Continued fevers	70	50	120
Small-pox	5	2	7
Measles	—	—	—
Diseases of the brain and nervous system	63	30	93
" " heart and great blood-vessels	44	43	87
" " respiratory organs	44	17	61
Consumption	26	36	62
Diseases of the digestive organs :			
(a) Cholera	33	21	54
(b) Dysentery	13	24	37
(c) Diarrhoea, inflammation, &c.	36	27	63
Diseases of urinary and genital organs (including venereal diseases)	9	13	22
Diseases of the skin, &c. (abscess, ulcers, gangrene, tumours, and erysipelas)	6	2	8
Rheumatic fever, &c.	2	—	2
Scurvy	—	1	1
Other diseases	6	5	11
Natural causes	2	4	6
Accidental deaths: (a) Drowned by wreck	315	560	875
(b) " casualty	32	34	66
(c) Killed at time of wreck	3	5	8
(d) " " casualty	3	8	11
(e) Drowned by accident other than wreck or casualty	141	209	350
(f) Other accidental deaths	63	66	129
Murder and homicide	3	3	6
Suicide	26	19	45
Unknown causes	69	7	76
Total	1,024	1,225	2,249

The rate of mortality of the British Mercantile Marine (merchant vessels) calculated on the deaths reported during 1886, and the total crews in 1887 (see p. 577) is 10·5 per 1,000. The rate for the crews of steam vessels is 9·6, and for sailing vessels 21 per 1,000.

Returns of the deaths of *fishermen* under all circumstances are not available. The Registrar-General of Shipping states the number of those who died in fishing vessels during the year 1886 at 129. This relates almost entirely to accidental deaths, eleven only being assigned to other causes. The rate of death calculated on the above number (129), and the total number of persons constantly engaged in fishing under the Fisheries Act during 1887, is 1·8 per 1,000.

Dr. Ogle, in his Supplement to the 45th Annual Report of the Registrar-General of England, gives the mean annual death-rates per 1,000 living fishermen in the three years 1880-2 as 8·32, at ages from twenty-five to forty-five years, and 19·74 at ages from forty-five to sixty-five years; but states that these figures 'must be accepted with some degree of hesitation.'

¹ Prepared from Board of Trade Return No. 325.

owing to the uncertainty attaching to the numbers of living and of deceased fishermen.

Comparison of the foregoing statistics with those of a previous modern period may be usefully made by means of the following tables obtained from a paper on the 'Health of Merchant Seamen,'¹ by J. O. M'William, M.D., Medical Inspector of Her Majesty's Customs:—

Table showing the Deaths from all Causes on board Merchant Ships of the United Kingdom and Channel Islands, employed in the Home and Foreign Voyages during the following Years, viz. :—

Royal Navy, Foreign and Home Stations.

Years	Rate of deaths per 1,000	Rate of deaths per 1,000
1852	13·82	From 1830 to 1837 (a period of peace) the mortality in the Navy averaged from all causes 13·8 per 1,000 15·5 19·4 25·8 — —
1853	19·05	
1854	17·06	
1855	19·68	
1856	20·40	
1857	19·56	
1858	19·60	
1859	19·68	
1860	21·91	
Average . . .	18·98	20·25

Table showing the Main Causes of Death on board of the Merchant Ships of the United Kingdom employed in the Home and Foreign Voyages during the following Years, viz. :—

Years	Rates per 1,000		Total strength from each of the under-mentioned causes		
	Fevers	Dysentery	Scurvy	Drowning and other accidents	Causes not ascertained
1852	4·86	1·00	·13	4·46	3·35
1853	7·55	1·96	·08	5·23	4·15
1854	4·02	2·40	·22	5·71	4·71
1855	4·18	3·65	·32	6·44	5·09
1856	4·67	·99	·42	7·99	6·53
1857	4·61	·93	·17	8·67	5·14
1858	3·40	1·11	—	9·95	4·93
1859	2·76	1·22	·21	9·14	5·09
1860	2·69	1·34	·14	11·95	5·02
Average .	4·80	1·62	·22	7·72	4·88

N.B. the steady fall of deaths from fevers, and as steady rise of deaths from drowning, in the successive years of this Table.

The rate of mortality in the merchant service as a whole has, therefore, fallen from an average of about 19 to 10·5 per 1,000 in the past quarter of a century. The improvement is mainly due to reduction of deaths on steam vessels—that on board sailing vessels in the year 1836 being, as already shown, 21 per 1,000.

No returns are issued by the Board of Trade relative to the sickness (not fatal) of seamen.

Judging by the experience obtained in the River Tyne Port as to the

¹ *Trans. Nat. Assoc. for Promotion of Social Science, 1862.*

zymotic diseases, and ailments somewhat resembling those diseases, admitted into the Floating Hospital of the sanitary authority during the nine years 1881-9 inclusive, the most frequently occurring febrile disorder among merchant seamen is *enteric fever*. Following this, in order of numbers, are small-pox, continued fever and febricula, scarlet fever, erysipelas, measles and German measles, diphtheria, African Coast fever. It is unnecessary to state that implicit reliance should not be placed on any mere statement of admissions to a hospital as a criterion of the relative liability of seamen to the diseases for which they were admitted.

Diseases of Sailors and Sea-goers in general.—The extent of the relative prevalence of certain ailments among sailors may be gathered from some of the foregoing statistics. Sea-life, either with mariners or other persons, is *per se*, apt to give rise to morbid conditions, some of which are more or less characteristic, and one at least is peculiarly of nautical origin. Obstinate constipation, lymphangitis, boils, and erysipelas are of frequent occurrence among sea-goers. So formerly was scurvy among Europeans, &c., and so at present is beriberi, its congener, among Indian and Indo-Chinese races. Ship fever is one of the old names for typhus. Sea-sickness is an ailment of marine life only.

But sailors in particular have special diseases, the result of their surroundings and the life they lead. Patissier,¹ in a chapter on the 'Maladies des Marins,' says seamen are subject to contagious fevers, scurvy, colic, diarrhoea, itch, ennui, hypochondriasis, melancholia, nostalgia, sea-sickness, the effects of vicissitude of climate, catarrhal affections, intermittent, remittent, bilious, putrid, ardent and exanthematic fevers, rheumatism, dysentery; in the tropics to cephalalgia, 'des douleurs rebelles, des boutons, le dragonneau.' He points out that on the open sea there is generally less sickness than along the coasts.

Lind specifies among the diseases most prevalent among crews of ships in the northern seas, catarrhs, rheumatism, pleurisy, and pneumonia.

Turner² mentions typhus and yellow fevers as peculiarly diseases of ships; and rheumatism, phthisis, cutaneous and venereal affections as also being prevalent among crews.

Scurvy is perhaps the disease above all others associated in the popular mind with the life of the seaman. Full particulars as to the history, causes, pathology, and prevention of this scourge of ships may be found in the writings of Lind, Alexander Armstrong, Macdonald, Wilson, Blyth ('Dictionary of Hygiene'). The Report for 1863 of the Medical Officer to the Privy Council contains an excellent monograph on scurvy in the Mercantile Marine by Dr. R. Barnes, with some important observations by the Medical Officer (Mr. Simon); and the half-yearly reports of the present Medical Officer of Health of the Port of London³ give valuable information as regards the prevalence of the disease in recent years, and its explanation. Among French writers on this subject, Fonssagrives may be specially named.

Cholera is closely connected with ships, and the contagion of it is invariably brought by them to this country. An account of this disease and its prevalence in Europe during 1884-5 will be found in the Report of Dr. Thorne to the Local Government Board in 1886. Other Blue Books and numerous medical works describe it from different points of view.

Yellow fever also belongs, in a sense, to ships and seamen. Commencing usually in the 'sailor quarter' of towns, it is carried aboard either by fomites

¹ *Traité des Maladies des Artisans*. Paris, 1822.

² Buck's *Hygiene*. Art. 'Hygiene of the Naval and Merchant Marine.'

³ Dr. William Collingridge.

or clothing, &c., or in the person of one of the ship's company, in the incubatory stage of the disease, and is there spread among the crew, and conveyed to other countries, occasionally to England.

The special duties of certain men engaged on shipboard subject them to disorders, the direct result of those duties. Attempts have been made by Fonssagrives and others to classify the diseases of seamen according to their particular occupations. Thus it appears that—

Men engaged aloft are very subject to cardiac hypertrophy, palmar psoriasis, hernia, excoriations on the anterior aspect of the thighs and legs, traumatic lesions of the hands and feet, &c. Arnould¹ mentions 'laryngites des gabiers' as a special ailment of this set of sailors.

Boatmen suffer from the effects of long exposures and frequent wettings, and in addition, according to Fonssagrives, to disease due to excessive drinking.

Steersmen are liable to accidents from the wheel, &c. Prolonged effort to keep on the alert for whistles and signals during fogs, &c., coincident with exposure to damp and cold, tends to cause various diseases of the organ of hearing. Dr. Cane² has recently shown that 'sailors are particularly liable to acute and sub-acute ailments of the ear.'

Look-out-men are said by Fonssagrives to suffer from weak sight, with amblyopia, photophobia, circumorbital pains and loss of accommodation.

Those whose duties confine them much to the interior of the ship, *cooks, bakers, storekeepers, men working in the hold, &c.*, are anæmic and debilitated. *Painters* on shipboard, as on shore, are apt to suffer from lead-poisoning (colic, &c.)

Work under conditions of high temperature, as in the performance of the duties of engineer, stoker, &c., is insalubrious. Thus Fonssagrives states that among this class there are many sick, their ailments being anæmia, acute inflammations, and other ailments arising from suppression of perspiration on sudden changes from heat to cold; in extreme cases, vertigo, debility, stupor, convulsions, or coma. The glare and heat of the fires, especially when followed by sudden changes to opposite conditions, seriously injure the eyes, causing conjunctivitis, corneitis, iritis, choroiditis. Hemeralopia or night-blindness is mentioned by various authors as a common ailment of firemen. 'Fireman's heart,' a special disease characterised by atony of the organ, was described by Levick, of Philadelphia, and is referred to by Turner³ and other authors. Phthisis is also prevalent among men of this class.

The disease, once so prevalent among the crews of French men-of-war, and described by many writers as *colique végétale* or *colique sèche*, which has long been known to have been plumbism, was one affecting firemen and engineers more than other persons on shipboard. At the present day it is difficult to understand how the real nature of this affection, which is stated by Boudin⁴ to be 'one of the most grave maladies of hot climates,' and which was met with principally on French steamships, could so long remain misunderstood. Boudin appears to be surprised at the absence of reference to it in English medical literature. He makes, at considerable length, a differential diagnosis between *colique sèche* and lead colic, and quotes Rochard, who states positively that the former is *not* lead poisoning. Numerous theories of the most opposite character were promulgated with regard to it. It was described by Segond as neuralgia of the great sym-

¹ *Nouveaux Eléments d'Hygiène.*

² *Lancet*, April 18, 1889.

³ *Buck's Hygiène.*

⁴ *Traité de Géographie et de Statistique Médicales.* Paris, 1857.

pathetic, by Langevinas as rheumatic gastro-enteralgia, by Fonsagrives (who afterwards admitted his error) as nervous endemic cholera of hot countries; and as stoker's colic, and under other titles by other observers. *Colique végétale*, in the words of De Méricourt, 'gave rise to innumerable treatises, and moreover put the physicians of the French marine into the highest passion for thirty years.'¹

M. Le Fevre at last demonstrated beyond dispute that *colique végétale* was nothing more or less than lead poisoning, due chiefly to impregnation of the drinking-water with that metal from the tubes of a distilling apparatus then in the use of the French Navy, the solder of joints in connection with the feeders of water-tanks, &c. The large amount of water, &c., consumed by stokers at once accounted for the great prevalence of the disease among this class.

Boiler-cleaners are liable to asphyxia. Admiralty Instruction, No. 1,229, enjoins precautions against this casualty.

The sailor's life is threatened at one time by congelation, at another by insolation or fulguration. Numerous traumatic accidents may befall him including, on steamships, explosions. Few catastrophes are more appalling than that of a ship on fire. Finally, in storms he may be washed overboard, or his vessel may be lost with all hands.

Fishermen.—'The lot of the fisherman,' says Patissier, writing sixty years ago, 'is worthy of compassion. . . . Their garments, continually wet, subject them to all disorders depending on suppressed transpiration, acute fevers, rheumatism, pleurisy, pneumonia, catarrhs, and other pectoral ailments.' The same writer describes this class as cachectic, and dropsical from fish diet; liable to obstinate ulcer of the leg, constipation; they are in danger of loss of life at sea. Let us compare the foregoing with the results of the more precise and wider observation of Dr. Ogle,² remembering always his caution as to implicit reliance on the completeness of his records. Dr. Ogle says: 'Taking them (the statistics) as they stand in the table . . . fishermen are a remarkably healthy set of men, rivalling in this respect the agriculturists. . . . The mortality figures for nervous diseases, for phthisis, and for diseases of the respiratory organs are remarkably low. The fisherman . . . has a much higher mortality than the farmer from diseases of the heart and other organs of circulation. . . . The mortality of fishermen from accidents is exceedingly high. . . . Out of 105 deaths of fishermen, 79 were thus occasioned.'

Health Preservation and Hygienic Rules at Sea.—By an Order in Council, dated January 1864, 'rules are in force on board passenger ships for preserving order, promoting health, and securing cleanliness and ventilation.'³

The sections of the Merchant Shipping Acts with respect to provisions and water for seamen have been already referred to. Under 80 & 81 Vict. c. 124, s. 10, a local marine board may appoint an inspector, whose duty it shall be, on application, to examine seamen applying for employment.

S. 280 of the Merchant Shipping Acts, 1854–1876 (consolidated), provides that every foreign-going ship, having one hundred persons or upwards on board, shall carry a duly qualified medical practitioner.

S. 41 of the Passengers' Acts, 1855–1870 (consolidated) provides that every passenger ship shall carry a duly qualified medical practitioner—

(1) When duration of the intended voyage is computed to exceed

¹ *Rapport sur le Progrès de l'Hygiène Navale*. Paris, 1857, p. 49.

² *Supplement to Forty-fifth Report of the Registrar-General*, 1885.

³ See *Notice to Passengers*, Appendix H.

eighty-five days in the case of sailing-ships, and forty-five days in the case of steamships, and the number of passengers exceeds fifty.

(2) Whenever the number of persons on board exceeds three hundred.

Re-vaccination in the Royal Navy.—Under Admiralty Instruction No. 1,157, all men and boys entering the service are to be re-vaccinated, and if no results follow the operation is to be repeated. There is no similar regulation in the merchant service.

Much valuable information as to the protection of health of crews and passengers will be found in various reports of the Medical Officer to the Local Government Board.

(D) CARGOES AND OTHER CONTENTS OF SHIPS IN RELATION TO HYGIENE

The hygienic condition of a merchant ship depends very largely on the nature of the cargo. Cargoes may be dangerous to life and health from the following causes—viz. (1) Infection; (2) unwholesomeness or decomposition and evolution of noxious vapours or gases; (3) combustion or 'heating'; (4) 'shifting' and interference with the equilibrium of the vessel.

Infectious Cargoes.—One of the most dangerous cargoes on account of infection is that of rags, of which a large quantity are transmitted by sea from one country to another. Thus, the State of Massachusetts in one year consumed 62,922 tons of rags in the paper industry,¹ about 40 per cent. of which were imported from foreign ports. In 1884 rags were brought to the United States from sixty-five different countries. Although rags are shipped in bales and tightly compressed, the amount of dust they give off in the hold of a ship during unloading is sometimes very great, and speaks volumes as to their unwholesomeness and filthiness. The attempt to secure disinfection of rags at the port of export before loading was made by the authorities of New York, Newhaven, and Boston in 1884, but without success.

The diseases most likely to be communicated by means of shipments of rags are small-pox and cholera. Infection of the former of these has undoubtedly been disseminated in this way at paper mills both in England and America. As stated by Dr. Buchanan in his memorandum (August 1888) as to rags from Egypt, 'it is not known that, as a matter of fact, cholera has ever been introduced into England by rags.' There is, however, great reason to fear danger from this, and accordingly the Local Government Board have from time to time since 1882 issued orders prohibiting the landing at British ports of rags from cholera-infected countries, unless under precautionary conditions as regards disinfection. Occasionally other fabricated material infected, or suspected to be so, forms part of the cargo, and has to be dealt with accordingly. Thus, in 1884, the Medical Officer of the Port of London (Dr. Collingridge) was called on to deal with a consignment of cast-off regimentals from Egypt, worn by the army of occupation in the previous year, and afterwards sold. In the absence of information they were treated as suspected and disinfected by superheated steam.² The clothing of persons dying abroad of infectious disease is occasionally sent home to England without satisfactory evidence of proper disinfection, and has to be dealt with on reaching its destination. Thus, the chests of a ship's officer who had died

¹ *Eighteenth Annual Report of the State Board of Health of Massachusetts, 1887.*

² *Report of the Medical Officer of Health to the Port of London for Half-year ended June 1884.*

of cholera in China were forwarded to Newcastle-upon-Tyne in 1889, and were afterwards disinfected by the Health Department of the Sanitary Authority.

Cargoes may be foul by nature, apart from any question of specific infection. Thus, consignments of live animals, fish, fruit, potatoes, bones, rotten grain, onions, manure, guano, &c., are apt to be a nuisance and injurious to health. Large quantities of frozen meat are now brought by ships to this country, whole cargoes of which are sometimes, from accidental causes, more or less decomposed on arrival, and have to be destroyed. Such material on board in such quantities as are actually met with necessarily renders the vessels very unhealthy.

Some cargoes are liable to undergo spontaneous combustion to a greater or less extent, and endanger the ships and those on board. Lime, soda, or potash, to which the sea-water has found access, will set a ship on fire. Among other articles liable to undergo spontaneous combustion the following may be mentioned as specially dangerous—viz. sugar, grain, tarpaulin, coal, greasy wool, cotton, hemp, and rags. Dr. Collingridge, in the report above quoted, mentions a noteworthy instance of a sailing barge with a foul cargo of spent lime from a gasworks. The lime had heated, and 'the fumes given off were so intense that it was impossible to navigate the vessel. The master and crew had quite lost their eyesight (temporarily), and a woman and two children had been taken ill.'

The heating of rice, seeds, and similar cargoes in iron ships is sometimes due to condensation of the vapour from the grain against the ironwork of the hold, the liquid flowing along the deck and beams to the lee side of the vessel, trickling down the waterways and stringer-plates, and, in the absence of scuppers, overflowing on the cargo.¹

On timber ships the steam from staves and other cargo may sometimes be seen issuing from the hatchways.

The Board of Trade Returns show that from 1871 to 1881, 231 vessels were lost or damaged, and seventy-five persons lost their lives, owing to explosions of coal gas or spontaneous combustion of coal cargoes.²

Certain materials as cargo, such as grain in bulk, &c., are dangerous for another reason, viz. tendency to 'shift' under the movements of the ship, and thus lead to foundering. Sec. 22 of 39 & 40 Vict. c. 80 provides that 'no cargo, of which more than one-third consists of any kind of grain, corn, rice, paddy, pulse, seeds, nuts, or nut kernels . . . shall be carried on any British ship unless . . . contained in bags, sacks, or barrels, or secured from shifting by boards, bulkheads, or otherwise.'

Cargoes and Dangerous Goods on Passenger Ships.—The coasting passenger boats between the Thames, Tyne, and other ports usually carry general cargo also. Under the Passengers' Acts no 'passenger ship' shall clear out or proceed to sea if there shall be on board as cargo, horses, cattle, gunpowder, vitriol, lucifer matches, guano, or green hides, or other articles 'likely to endanger the health or lives of the passengers or the safety of the ship.' 36 & 37 Vict. c. 85, s. 23, places restrictions on the carriage by sea of dangerous goods (explosives, &c.) without distinctly marking the nature of the article on the outside of the package. Under 39 & 40 Vict. c. 80, s. 24, any ship, whether British or foreign, arriving in a port of the United Kingdom, and carrying timber as deck cargo, renders the master liable to penalty.

¹ *Shipping Gazette*, May 24, 1889.

² *Trans. Inst. Naval Architects*, 1884. Art. 'Ventilation of Merchant Ships,' by James Webb, Surveyor to Lloyd's.

(E) PORT SANITATION AND SPECIAL MEASURES FOR THE PREVENTION OF DISEASE ON SHIPBOARD

The object of the sanitary supervision of ships is twofold. It aims at (1) the protection of the health of those on board, and (2) the prevention of the introduction of disease into ports by ships. For this purpose three sets of machinery, so to speak, are provided. The first set operates prior to the departure of the ship on her voyage, and includes the various details for securing, as far as possible before putting out to sea, the housing of crews and passengers under conditions necessary for, and favourable to, health. The second refers to such measures as are required to maintain the hygiene of ships at sea, and to prevent the occurrence of disease or spread of infection among persons whilst travelling by sea. The third relates to the means of ascertaining the health of ships' companies immediately before arrival, and subsequently during their stay, in port; as also the mode of dealing with any cases of infectious disease on board ships at these times, and of abating any nuisances or conditions injurious to health in connection with vessels whilst at their moorings. The first and second of these groups of appliances have, with certain exceptions, been treated of in the preceding pages. The third group comes under a special department, and includes, *inter alia*, the important measures of isolation and disinfection, which, though they may also have to be practised on shipboard during the voyage, are generally more effectually done in harbour, and therefore will be described under the head of Port Sanitation.

Constitution of Port Sanitary Authorities.—Originally formed with the sole object of preventing the introduction of cholera and other infectious diseases into this country, some of the oldest of the present port sanitary authorities of Great Britain were merely joint committees of local sanitary boards. These joint committees were in 1872 constituted port sanitary authorities by the Local Government Board, and the ports on which their districts abut were constituted port sanitary districts. For some years various port sanitary authorities were at first temporarily constituted, and became permanent afterwards. In the year 1886 there were in England and Wales thirteen permanent and thirty-four temporary port sanitary authorities,¹ whose duty it was, and is, as above stated, to secure the good sanitary condition of vessels and the prevention of the importation of infectious disease.

The special powers under which English port sanitary authorities act are as follow :—

The Public Health Act, 1875, ss. 287–291, provides for the constitution, jurisdiction, and finances of such authorities under *provisional orders of the Local Government Board*. S. 110 provides that, as regards nuisances, 'any ship or vessel, except those of British or foreign Governments, lying in any river, harbour, or other water, within the district of a local authority shall be subject to the jurisdiction of that authority in the same manner as if it were a house . . . ;' and under s. 130 the Local Government Board may make, alter, or revoke regulations for the treatment and prevention of cholera and other epidemic diseases.

The Public Health (Ships) Act, 1885, provides that s. 110 of the above

¹ *Report on the Sanitary Survey of Port and Principal Riparian Sanitary Districts, 1885–86*, by F. W. Blaxall, M.D., Medical Inspector to the Local Government Board. A return to the House of Commons shows that by 1887 the total number of port sanitary authorities was fifty-five.

Act shall have effect, not only as regards nuisances, 'but also for the purpose of such provisions of that Act relating to infectious diseases and hospitals' as are referred to in sections 120, 121, 124-6, 128, 131-3 of that Act.

The General Orders of the Local Government Board of 1883 define the duties of officers of port authorities, and lay down regulations in case of the appearance of cholera. The adoption of the *Infectious Diseases Notification Act of 1889* by port as by other sanitary authorities is optional. Prior to the passing of this Act the Local Government Board had power, under s. 125 of the Public Health Act, 1875, to authorise regulations made by a port sanitary authority for the compulsory notification and removal to, and detention in, hospital of any case of infectious disease on shipboard. Such regulations were in operation in the London, Bristol, and the River Tyne Port Sanitary Districts some years prior to the passing of the Notification Act of 1889. During and since 1888 several *Orders with respect to regulations from cholera-infected countries* have been issued to port sanitary authorities by the Local Government Board, and withdrawn when no longer required.

The Merchant Shipping and Passengers' Acts relate partly to matters of hygiene (accommodation, space, conveniences, ventilation, lighting, provisions, water, medicines, antiscorbutics, health rules, dangerous cargoes, &c.); and, having been in operation before the era of port sanitation, appear to some extent to take precedence in these matters over the special statutes and legal provisions above referred to, with which they are not always in accord. Thus, for example, as regards accommodation for crews, the amount prescribed, presumably in connection with purposes of lading only, dominates the action of sanitary officers, who might hope in vain to secure more space per person than the regulation seventy-two cubic feet laid down by Her Majesty's Customs. Again, on passenger ships, hospitals, privies, light and ventilation, &c., are under the approval of officials not subject to the direction of the sanitary authority.

The Quarantine Act of George IV. is still in operation, and, like the Merchant Shipping Acts, is administered by a body working principally for reasons of commerce, and constituted long before the present system of sanitary authorities was in existence. Hence arises some overlapping of duties of the respective departments (see 'Quarantine,' p. 587).

The *mode of procedure* on the arrival of a vessel from a foreign or colonial port is thus described by Dr. Blaxall: ¹—

'At each Customs port a station or stations have been appointed by the Commissioners of Customs for the boarding of vessels arriving from foreign or colonial ports, and captains are required on arrival to take their vessels with despatch to these stations without touching at any intermediate place, and not to remove therefrom except with the knowledge of the Customs officer.

'The primary duty of the Customs officer is to guard the revenue; but he is further required to exercise certain functions in respect of the health of ships. Besides such duties, hardly more than nominal, imposed on him by the quarantine laws, he has more important duties in respect of indigenous infections and of cholera. He has to make inquiry as to the health of the port whence the vessel has come, and as to the health of the crew and passengers during the voyage home and at the time of arrival. In the event of infectious sickness being reported to him, he is, in compliance with instructions from the Commissioners of Customs (General Order, April 20, 1861), to acquaint the local authority (port or riparian, as the case may be) of the

¹ *Report to the Local Government Board, 1885-86 (Sanitary Survey).*

circumstance. Upon this authority will then devolve the responsibility of taking steps to prevent the introduction and spread of the disease. For this purpose the medical officer of health is to board the vessel, and, should he find the sickness to be of an infectious character, his duty is to take steps to secure the isolation of the sick, together with efficient disinfection of the vessel, bedding, clothes, &c., and to satisfy himself as to the health of the rest of the persons on board.'

The Board of Trade, in their General Order ¹²⁹
1888, point out that 'unless

plague, yellow fever, or cholera has existed on board a vessel there is no legal authority for detaining her on sanitary grounds;' and direct their officers, in cases of small-pox, measles, &c., to give notice to the sanitary authority 'if possible before any sick person has landed; but the vessel should not be placed under detention.' So far as the experience of the writer enables him to speak, however, the practice is to give immediate notice to the medical officer of health, and to detain the vessel for a short time (not exceeding a very few hours), until he is able to board her and cause the necessary action to be taken.

In case of *cholera* on board a vessel, the *Cholera Regulations Order* of August 28, 1890, of the Local Government Board is brought into operation, of which the following extracts include the principal administrative points:—

'For the purposes of these regulations every ship is to be deemed infected with cholera in which there has been a case of the disease during the voyage or stay of such ship in a port in the course of the voyage.

'The Customs officers are to detain any such cholera-infected or suspected ship, cause it to be moored as they direct, and inform the sanitary authority, who are to cause their medical officer of health to visit the ship. No one is to leave the ship whilst she is under detention.

'If the medical officer of health finds the ship free from infection, detention ceases. If she is infected with cholera she is to be moored at a place previously fixed by the sanitary authority for the purpose. If not visited by the medical officer of health within twelve hours after notice is given by the Customs, the ship is released.

'The sanitary authority are required to fix a place for the mooring of infected or suspected ships, with the approval of the chief officer of Customs, and to provide for the reception of cholera patients and persons suffering from illness suspected to be that disease.

'The medical officer of health may visit any ship if he believes her to be cholera-infected, or if she have come from a cholera-infected place, whether she have previously been examined by Customs officers or not. If he is of opinion that the ship is infected with cholera he gives the master a certificate to that effect, and makes a duplicate. The master must then moor the ship in the mooring place of the sanitary authority above mentioned, and no one is to leave until the medical officer of health has examined everyone on board. He certifies those affected with cholera. Every person so certified by him is, if fit to bear removal, to be removed to hospital, and must remain there till certified by him to be free from infection. If not fit to bear removal, the patient is to stay on board until the medical officer of health authorises his removal, and the ship is to remain under the control of the medical officer of health.

'Any suspicious case of illness may either be detained on board for two days or be taken to hospital and there detained for two days, till the case is cleared up, when, if it is cholera, the patient is dealt with as already described; or if otherwise he is discharged.

'After the examination of the ship as above mentioned, all who are not certified as having cholera, or suspected of having it, may land on giving their names, places of destination, and addresses.

'The medical officer of health is to give directions and take steps necessary to prevent the spread of infection, and the master must carry out such directions.

'Corpses of persons dying of cholera on shipboard are to be buried in the sea or by the sanitary authority.

'The master is (or in his default the sanitary authority are) required to destroy articles soiled with choleraic discharges, and to disinfect, or, if necessary, destroy, any other infected bedding or clothing.

'The master is also to cause the ship to be disinfected, and all infected articles therein, in addition to the foregoing, to be disinfected or destroyed.

'The master of every cholera-infected ship, whilst within three miles of the coast of England or Wales, must keep a yellow flag hoisted from sunrise to sunset.'

This Order was amended in 1892, when cholera was present on the west coast of Europe, by the issue of other Orders of August 29 and 31 of that year, and these last two Orders were further amended and consolidated by the Order of September 6, 1892, which gave increased powers to the medical officer of health. Article I. of this Order prohibits the landing of any person on board an infected ship unless he can satisfy the medical officer of health as to his name, place of destination, and address at such place. Article II. provides that when a ship is not infected with cholera, but has passengers on board who are in a filthy or otherwise unwholesome condition, or has come from a place infected with cholera, the medical officer of health may, if in his opinion it is desirable, with a view to checking the introduction or spread of cholera, give a certificate in duplicate (one to the master of the vessel, the other to the sanitary authority) to the effect that persons should not be allowed to land unless they satisfied him as to their names, places of destination, and addresses at such places. These amendments aimed at the detention of vessels bringing Jewish immigrants, many of whom were at the time arriving from Hamburg, where a severe epidemic of cholera then prevailed. Persons of this class arriving in the Thames were detained, and many were subsequently removed to the hospital of the port sanitary authority at Gravesend by Dr. Collingridge, who was able to keep them under observation until a sufficient time had elapsed to ensure that they would not subsequently develop cholera from any infection received on the Continent or during the voyage. Article IV. provides that when a ship is infected with cholera, or comes from a port infected with cholera, the medical officer of health may direct the bilge-water¹ to be pumped out before the ship enters any dock or basin, and, on the sanitary authority providing a proper supply of water for drinking and cooking purposes for persons on board the ship, he may direct all casks and tanks on board the ship containing water for the use of such persons to be emptied and cleansed, and the master is required to cause these directions to be carried into effect.

Numerous rag orders, providing from time to time for special temporary requirements, have also been made.

The entire question of cholera-prevention, from the point of view of port sanitary authorities, was fully discussed at a largely attended conference of the port sanitary authorities of England and Wales held at the Guildhall,

¹ No legal provision is made for dealing, in like manner, with the much more serious matter of *ballast* (water or sand), which is often taken from infected rivers or their shores—e.g. from the Elbe during the cholera epidemic of 1892.

London, on February 17, 1898,¹ under the presidency of the Lord Mayor, when the following subjects were considered, namely:—1. The desirability of cholera precautions in ports being carried out at the national expense. 2. The medical inspection of ships arriving from cholera-infected countries. 3. The periodical issue of a recognised list of ports infected with cholera. 4. The detention of port medical officers on board of ships not having a clean bill of health. 5. The supervision of suspected vessels whilst in port. 6. Passengers' addresses of destination. 7. The detention of crews on board of ships whilst in infected ports. 8. Infected ballast. 9. Report of a committee of port medical officers of health appointed to consider the appliances necessary for the thorough disinfection of ships and their contents throughout. 10. The disposal of infected corpses. 11. Hospital accommodation required, &c.

The above-mentioned report on the appliances for disinfection of ships and their contents recommended the fitting up of a hulk, propelled by steam, with apparatus for disinfection by means of the mercuric drench, sulphurous fumes, and steam. The fumigation of infected spaces, &c., by burning sulphur is now superseded by the employment of sulphurous acid liquefied under pressure, which is supplied by the manufacturers in cylinders available for convenient use.

Quarantine.—In case of *yellow fever* or *plague* the Customs are required, under the Quarantine Acts, &c., to take precautions—an apparent anomaly in sanitary administration, since, as pointed out by Dr. Blaxall in the report already quoted from, 'the Imperial Government possesses neither quarantine establishments nor isolation hospitals for the purpose.'

In one instance in the experience of the writer the quarantine laws above referred to appeared not to work in harmony with other sanitary regulations. A certain vessel on arrival in the Tyne was reported to the port sanitary authority by the Customs as having no 'bill of health.' She was then boarded by the assistant medical officer of health, who, finding her free from infection, left her before she was formally admitted to 'pratique' by the Customs. This leaving of an uninfected ship by the port sanitary medical officer, although quite in accordance with his duties, was objected to by the Commissioners of Customs as a breach of the Quarantine Act.

Space is not at present available to enter into the details of quarantine. For opinions as to the efficacy of this means of dealing with foreign disease the reader is referred to the Report of the Medical Officer of the Privy Council for 1865.² Valuable information as to the comparative merits of quarantine *v.* systematic inspection will be found in the Supplementary Report of the Medical Officer to the Local Government Board for 1877.³ The medical officer's Supplement to the Report of the Local Government Board for 1879-80 contains Mr. Netten Radcliffe's lucid statement of 'Facts relating to Quarantine in the Red Sea,' with inferences and deductions therefrom, together with a *résumé* of current scientific opinion in Europe as regards quarantine in cholera and plague, and its administration in relation to the pilgrim traffic.

A summary of the enactments, past and present, British and foreign, on the subject will be found in 'The Laws of Quarantine,' by Sir Shenstone Baker. A full description of the 'Holt System of Quarantine' is given in 'Public Health' for April 1889,⁴ and subsequently by Dr. Holt himself.⁵

¹ A full report of this conference is given in the *Shipping Gazette* of February 18, 1898, which contains also the text of the Merchant Seamen (Provisions) Bill, 1898.

² By Mr. (now Sir) John Simon.

³ By the late Dr. E. C. Seaton.

⁴ By A. Campbell Munro, M.D.

⁵ *An Epitomised Review of the Principles and Practice of Maritime Sanitation.* New Orleans, 1892.

PORT SANITARY STAFF AND APPLIANCES

For the proper administration of their powers and fulfilment of their duties, port sanitary authorities require the aid of officers, such as a medical officer of health, an inspector of nuisances, &c., and also certain appliances for dealing with infectious disease and ascertaining nuisances and sanitary defects on board of ships. These comprise—

1. Hospital accommodation for infectious diseases.
2. Means of transporting infectious cases to hospital, and officers from ship to ship.

3. Apparatus for disinfection.

Hospital Accommodation.—In considering the question of the provision of a hospital for the isolation of cases of infectious disease occurring on ship-board, over which cases, under section 124 of the Public Health Act, the sanitary authority have power of compulsory removal, several points claim attention, and among others the following :—

1. The amount of the accommodation to be provided.
2. The character of the accommodation to be provided.
3. The site of the hospital.

The number of beds for the reception of infectious cases in an urban district on shore is usually calculated at the rate of one bed for every thousand persons in such district. This rule, which, in towns where the residents may be regarded as practically stationary, works out fairly well in practice, is obviously inapplicable in the case of ports where the population is, in more senses than one, *floating*. The people in ports come and go continually, and the total number varies greatly from day to day, so that it cannot be estimated beforehand even approximately. The number of persons who arrive in a given time—say a year—affords little guide as to probable requirement in the way of beds for infectious diseases. In the Tyne Port the total arrivals of crews and passengers in a year is about 250,000. To provide on this basis, and at the same ratio, as for a town would be absurd. Actual experience on the Tyne shows that the number of cases requiring isolation does not ordinarily exceed a dozen in the year. But ordinary experience does not involve the dangers and possibilities of an epidemic of cholera or a large outbreak of typhus, small-pox, or scarlet fever (for example), in an emigrant ship—contingencies which may at any time occur, and should, as far as practicable, be provided for. Besides, the ordinary admissions may or may not be chiefly cases of the same disease. Having these different points in view, the Tyne Port Sanitary Authority, with the approval of the Local Government Board, have provided accommodation for *thirty* patients in their hospital, presently to be described. The hospital of the Port of London was formerly a ship, but is now on land, as is the case in some other ports—e.g. that of Sunderland. In many instances—perhaps the majority—port sanitary authorities have preferred to fit up floating hospitals within the limits of their own districts rather than send their patients ashore. There are at least two advantages in such a course—viz. (1) obviation of the double carriage of the sick by water and by land, and (2) facility of drainage and refuse disposal. Floating hospitals for port districts are also desirable for the reason of confining the infection to the sanitary district in which it is found. Whether on water or on land, it is advisable that the hospital should be in as isolated a position as possible.

The character of the accommodation to be provided will involve the following considerations :—

1. Diseases to be isolated.
2. Sex of the patients.

The cases most likely to be sent to hospital, under ordinary circumstances, are the more common zymotic diseases (enteric and other 'fevers,' scarlet fever, small-pox, measles, and diphtheria), together with ailments suspected of being or mistaken for these. Generally speaking, all of the patients will be males; hence it will be unnecessary to provide separate wards for each sex and each disease, as in hospitals for infectious disease on land. In arriving at a conclusion as to the number of different wards to be built, it should be borne in mind that a single case of infectious disease is sufficient to occupy an entire ward to the exclusion of other diseases. For this reason it is desirable to provide as many wards and as few beds in each as is consistent with proper nursing and economic administration in other respects.

The *Tyne Floating Hospital*, which is somewhat exceptional in construction, as shown in the accompanying illustration (fig. 19) and plan, is described by the architect¹ as follows:—

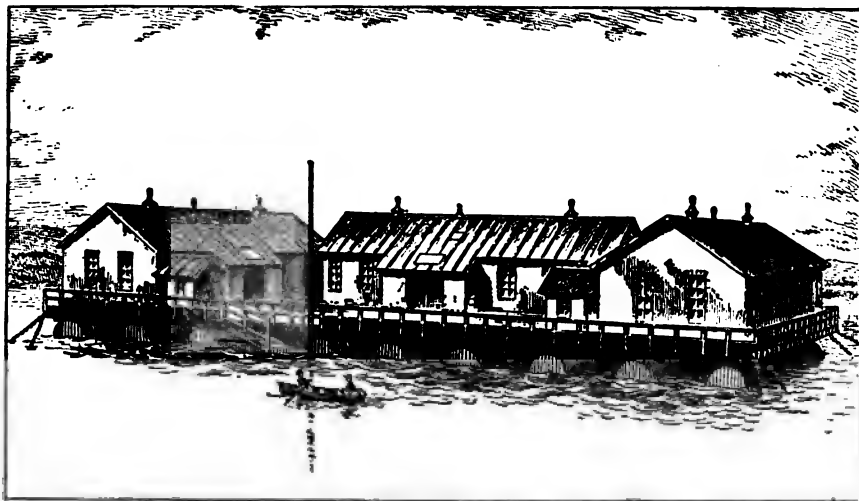


FIG. 19.—Floating Hospital of the River Tyne Port Sanitary Authority.

The float consists of a series of ten wrought-iron cylindrical pontoons, each 72 feet long and 6 feet in diameter, with hemispherical ends. The displacement of each pontoon is 53½ tons, or a total for the ten of 535 tons. Seven rolled wrought-iron girders, each 140 feet long and 12 inches deep, connect the pontoons, being fixed to curved saddles which rest on the cylinders and are riveted thereto. The connection between the saddles and the girders resting on them is by screw-bolts, so that any pontoon can be detached, partially sunk by admitting water, and so removed from its position for repair or painting. The distance between the centres of the pontoons when in position is 14 feet 6 inches. Across the iron girders are laid creosoted joists, 11 inches by 8 inches, 4 feet apart, which are bolted to wrought-iron chubbs riveted to the upper flanges of the girders, and across these joists again is laid a close deck of creosoted planking 3 inches thick, spiked down with 6-inch spikes. The whole forms a platform or deck 140 feet long and 70 feet wide, which serves to carry the ward blocks, &c.

In order to prevent any tendency to racking of the float a system of diagonal bracing of 4-inch by 8-inch T-irons connects the longitudinal

¹ Wm. Geo. Laws, M.I.C.E., City Engineer for Newcastle-upon-Tyne. The description is taken from the *Transactions of the Association of Municipal Engineers* at their meeting at Newcastle in 1887.

girders, being riveted to the under-side of their upper flanges, thus securing perfect stiffness in the plane of the deck. The whole deck, however, being thin relatively to its area, would yield partially to any inequality of support caused by heavy waves, and as an additional precaution against this, and to relieve the strain caused by the tidal current acting on the submerged part of the pontoons, four barks of timber are run the whole length of the float below the pontoons, and are secured to the iron work of the deck by $1\frac{1}{2}$ -inch bolts. These were not intended in the original design, but having been added for the purpose of facilitating the launching and protecting of the pontoons while being floated down the river, it was decided to retain them as likely to be still useful after the vessel was moored. The pontoons have each on their upper sides two manholes with bolted covers, for the purpose of giving access to the interior. These are reached through trap-doors in the deck above.

Access from boats to the deck is obtained by means of a sloping gangway, which at its lower end rests on a fender-beam attached to the ends of three of the pontoons, and serves to protect them from damage by the steam launch when she comes alongside. The height of the deck above water is 4 feet. The platform is protected all round by a strong wooden hand-rail, and as the main buildings do not come nearer than 2 feet to the edge of the deck, there is a narrow passage thus left all round the outside to give access for painting, repairs, &c.

The ward blocks are three in number, built of timber, and with a double skin of cleading. They are each 65 feet long, $23\frac{1}{2}$ feet wide, and about 12 feet 6 inches high to eaves, and 21 feet 6 inches to ridge. The roof is of zinc, on Braby's system. It is carried on half-coupled timbers with purlins and rafters. Each ward block is divided into two wards for patients, containing six and four beds respectively; and to each ward is attached a small offshoot, 8 feet square, containing w.c. and scullery. In the centre of each ward is a Musgrave's slow-combustion stove, the chimney of which is carried up through the roof, passing through a ventilating shaft 18 inches square, widening out to 3 feet at ceiling level. The stove-pipe thus does not come in contact with the wood in any part, and by its heat establishes an upward current through the ventilating shaft, which is controlled by light iron flaps worked by cords from below.

The wards are lined with pitch-pine boards dressed and varnished, and the ceiling is of the same material carried along the couples and below the collar beam. There are eight windows in the larger ward, and six in the smaller. These are each 8 feet high by 2 feet 6 inches, and have sliding sashes for two-thirds of their height, the upper third forming a swinging sash, worked by a rack and screw by cords from below, so that they can be set to any opening, and fixed by the same operation. The entrance of pure air below is effected by Kite's ventilators fixed in the walls under each bed. The ventilation is thus under complete control.

Each ward block has an independent floor laid on joists, and 9 inches above the deck, thus further providing for ventilation. The two wards in each block are separated by a space of 10 feet wide, in which is contained the nurse's room, 18 feet by 10 feet, and the bath-room, 10 feet by 9 feet.

A small window in each side of the nurse's room enables her to command a view of all the beds. In this room is placed a patent stove, formed of wrought iron and welded in one piece. This gives a constant supply of hot water by means of a circulating cistern in the roof, from which it is carried in wrought-iron pipes to the bath-room and scullery. Cisterns for fresh

water, and river water for washing, are also contained in the roof, in the space above the ceilings.

The soil pipes and waste pipes discharge directly into the water outside, and are cut off short 80 inches below deck-level, so as to form an effectual air trap, and prevent any of the excreta or slops hanging about their ends. A force pump in each scullery affords the means of raising river water for washing purposes. The fresh-water cisterns are filled from water boats plying on the river. Rain tubs are so placed as to catch the roof water and preserve it for use. A small porch over the three entrance doors forms a lobby to each ward, and protects it from draughts.

The bath-room contains a lavatory with two tip basins, and a movable bath on wheels, which can in case of necessity be taken into the wards alongside the patient's bed.

The three ward blocks are arranged as three sides of a rectangle, leaving a clear deck space of 80 feet by 85 feet. It is proposed to fit up a light crane over the gangway, so that patients may, when necessary, be lifted out of the launch on a stretcher.

The moorings consist of eight $1\frac{1}{2}$ -inch chains, averaging thirty fathoms, each attached on the river side to screw piles, and towards the Slake to beams sunk in the sand. In-board they are attached to eight double bollards securely bolted to the deck and its iron framing. Thus moored, the float rides quite easily. The berth chosen in Jarrow Slake is peculiarly suitable, being in the centre of a wide expanse covered by water at high tide, and inaccessible at any time except by boat.

The total cost has been as follows:—

	£
Pontoon float	3,060
Three ward blocks	2,110
Dredging and moorings	530
Total	5,700, or £190 per bed.

The existing administrative block of the hospital (figs. 21 and 22) is

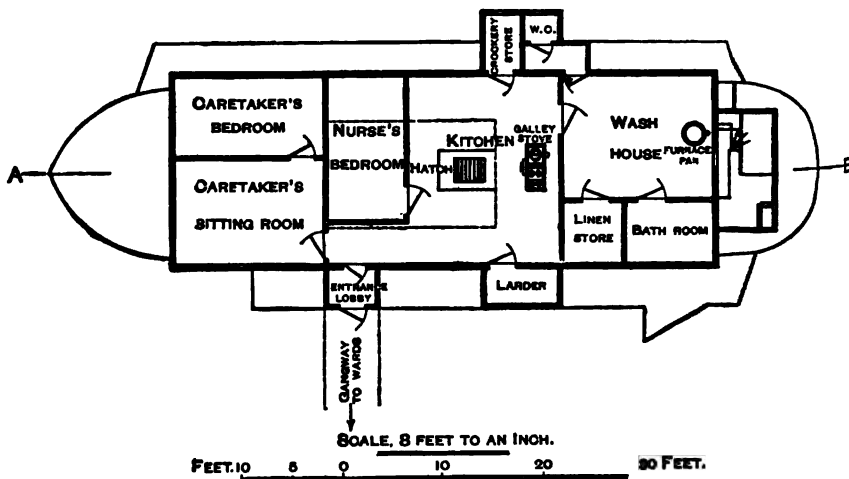


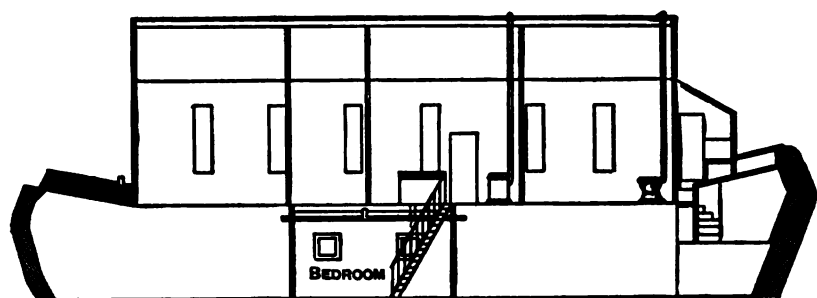
FIG. 21.

placed on a separate float moored to the south of the pontoons, and connected immediately with the pontoon deck by a gangway.

Among other advantages of this pontoon hospital are the following :—

- (1) Its stability, even in very rough weather.
- (2) The proximity of the deck to the water, and consequent facility of access for patients and others.

Means of Transport of Patients, &c.—In many ports the rule is to cause all vessels having cases of infectious disease on board to moor at a certain place, suitable for the removal of the patient to hospital. Thus, when the hospital is on a float, certain buoys are generally fixed near it, to which infected ships only are to be moored. These are distinguished by colour (white). In such cases, and when the hospital is on land, the patient may have to be lowered from his ship and conveyed to hospital or shore in an open boat. The foregoing process involves some detention of the ship and loss of time before the patient can be removed from her. The quickest and in every way most satisfactory method is to have a steam-launch fitted up at one end for the transport of the sick. The launch of the Tyne Port Sanitary Authority is provided with a fore cabin for this purpose, sufficient to



SECTION ON LINE A.B.

FIG. 22.—Floating Hospital. Administrative Block moored to south of the Pontoon Hospital.

accommodate two patients. The skylight of this cabin is double-hinged in the centre, and removable entire, so as to allow of the lowering of the ambulance couch and patient directly into the proper place in the cabin, as shown in fig. 28.

The maintenance of the horizontal position in the removal of the sick from place to place is a matter of much importance, especially in cases of fever; and in enteric fever this is imperative, as neglect of such a precaution may very readily lead to rupture of the intestine. To obviate danger of this kind, the writer has had a special cot or couch made (see figs. 28 and 24). The cot consists of a chain and spiral spring mattress (Chorlton's) 4 feet 2 inches by 1 foot 8½ inches in a light iron frame, attached to a curved ash frame, made a little shorter in length, so as to allow the mattress to 'dish' or curve downwards a little, for the patient to rest on. The iron frame is loosely attached at the sides to stout ash poles with hinged handles, so as to allow of turning in small space. The poles are separated by cross-pieces, to which the ends of the mattress are firmly fixed. To the upper of these cross-pieces a head-board is hinged, which may be set at any angle required. The cross-piece of the lower end is provided with a foot-board. Over the mattress and head-board are placed an india-rubber air-bed and pillow, each in a calico case. When the patient is in the cot

the bed-clothing is placed over him, and all is made secure by means of straps and buckles.



FIG 28.—Method of lowering patient to his place in the Ambulance-launch.
River Tyne Port.

The method of lowering a patient in the cot is as follows :—The rope, which is fixed by the eyelets and clove-hitches as shown in the illustration,

is made to cross over the middle of the patient's body in equal lengths, and under the crossing the hook of the lowering tackle is put in 'fore and aft' and firmly lashed in its place. The tackle is fixed to the end of the yard-arm or the head of a davit, and the patient lowered with safety to the launch. On arrival at the gangway of the hospital he is hoisted from the cabin and carried on the cot by bearers to his ward.

Disinfection afloat is practically the same as elsewhere, with certain exceptions, treated of in the Report adopted by the Conference of Port Sanitary Authorities (see p. 587),¹ that the apparatus commonly employed for the purpose of purifying bedding and clothing on land are too large and too heavy for use anywhere except on land. Appliances in which the required temperature is obtained by means of gas are, of course, not available on shipboard.

Nuisances on shipboard in port are dealt with in the same manner and by the same powers as on shore.

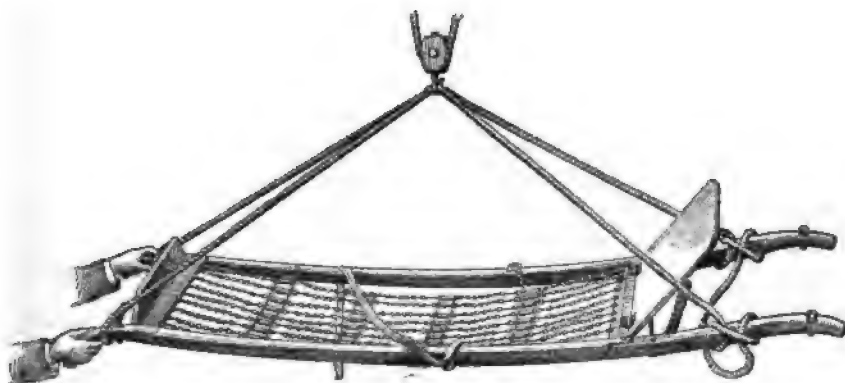


FIG. 24.

Structural sanitary defects, crew-space, passengers' accommodation, &c., are, as previously shown, to a considerable extent controlled by officers appointed under the Merchant Shipping and Passengers' Acts. A definition of the position and duty of the officers of port sanitary authorities in relation to those of surveyors of the Board of Trade, emigration officers, and other officials who at present have to do with sanitary matters on shipboard, would be advantageous to the health of both crews and passengers.

All vessels, whether in the foreign or the home trade, should be compelled to report their cases of sickness on arrival. At present coasting vessels are not required to do this. Such reports should be made to the sanitary authority and not to the Customs.

The writer of the foregoing article is much indebted to Dr. James Hindhaugh for several photographs required for illustration; to Messrs. John Blumer & Sons; to Mr. William Whyte, M.Inst.C.E., for plans and other valuable information respecting various parts of ships; and to many other friends for the loan of works on marine hygiene—to all of whom he desires to express his grateful acknowledgments.

¹ And described in full in the *Shipping Gazette* for February 18, 1898.

(F) BIBLIOGRAPHY OF MARINE HYGIENE

English.

- Armstrong, Alex., M.D., R.N. : Observations on Naval Hygiene and Scurvy, 1858.
 Armstrong, Henry E., M.O.H. : Sanitary Administration on the Tyne : a Seven-Year Retrospect, 1881-87. Public Health, June 1886.
 " " Annual Reports of the M.O.H. to the Tyne Port Sanitary Authority, 1881 onwards.
 Baker, Sir Sherston, Bart. : The Laws Relating to Quarantine, 1893.
 Blaxall, F. H., M.D., B.N. : Report of Medical Officer to Local Government Board, 1881. (Emigration and Immigration).
 Blyth, Wynter A. : Dictionary of Hygiene. Arts. 'Hygiene (Naval),' 'Scurvy,' 'Ventilation,' &c.
 Board of Trade : Annual Statement of the Navigation and Shipping of the United Kingdom for the Year 1891. House of Commons, 1892.
 " " Instructions as to the Survey of Passenger Accommodation, Crew-spaces, Lights, and Fog Signals, 1892.
 " " Merchant Service. Returns Relating to the Deaths of Seamen and Fishermen. House of Commons, August 1888.
 " " Navies of England and other Countries. Return to House of Commons, April 1888.
 " " Notice to Passengers under the Passengers' Acts, 1887.
 " " Regulations as to the Examination of Masters, Mates, and Engineers in the Mercantile Marine, 1888.
 " " Regulations as to the Survey of Steamships carrying Passengers, 1888.
 " " Sea Casualties. (Wreck Statistics.) Return to Parliament, 1892.
 " " Tables Showing the Progress of British Merchant Shipping. Return to House of Commons, May 1892.
 Boyd, A. C. : The Merchant Shipping Laws, 1876.
 Brailly, W. A., M.D. : On Colour Blindness. Heath's Dictionary of Practical Surgery, vol. i. 1886.
 " " On Colour Blindness. Transactions Ophthalmological Society, vol. i. p. 191, and vol. ii. p. 184.
 Brassey, Sir T. C., K.C.B., M.P. : The British Navy. London, 1888.
 British Encyclopædia, 1860. Art. 'Shipbuilding.'
 Buchanan, G., M.D. : Report of Medical Officer to Local Government Board, 1883. (Bag from Egypt).
 Campbell-Munro, H., M.D. : Art. 'The Holt System of Quarantine.' Public Health, April 1889.
 Canal Boats' Acts.
 Cane, F. E., L.R.C.P. : The Hearing of Seamen. Lancet, April 18, 1889.
 Collingridge, W., M.D. : Annual Reports of the M.O.H. to the Port of London.
 Edridge-Green, F. W., M.D. : On the Detection of Colour Blindness. Baillière & Co., 1888.
 Eighteenth Annual Report of the State Board of Health of Massachusetts, 1886. (Bag and Infectious Disease.)
 Guy, W., M.D. : Public Health. (Scurvy, &c.)
 Leach, Harry, M.D. : Report on the Hygienic Condition of the Mercantile Marine. Reprint from British Medical Journal, 1867.
 Lind : Essay on the Health of Seamen. London, 1774.
 " Treatise of the Scurvy. Edinburgh, 1753.
 Lindsay, W. S., M.P. : History of Merchant Shipping and Ancient Commerce. London, 1874.
 Macdonald, J., M.D., R.N. : Naval Hygiene, 1881.
 Merchant Shipping Acts. Passengers' Acts. Public Health Act, 1875.
 Merchant Shipping (Fishing Boats) Act, 1883.
 McWilliams, J. O., M.D., R.N. : Health of Merchant Seamen. Reprint Transactions Social Science Association, 1862.
 Navy Health Reports.
 Paasch, H. : From Keel to Truck. Antwerp, 1885.
 Parkes, E. A. : Practical Hygiene.
 Public Health (Ships, &c.) Act, 1885.
 Queen's Regulations and Admiralty Instructions, 1893.
 Rattray, Alex., M.D., R.N. : On the Dieting of Seamen. Navy Health Report for 1867.

- Report of Local Government Board, 1887-8. Mr. J. Brydone on Canal Boats.
 Report of Medical Officer to Privy Council, 1863. (Simon.)
 " " " " (Barnes.)
 " " " " 1865. (Simon.)
 Sea Fisheries (England and Wales). Sixth Annual Report of the Inspector (for 1891).
 Sea Fisheries of the United Kingdom. Board of Trade Return, February 1888.
 Supplement to Forty-fifth Report of Registrar-General, 1885. (Fishermen.)
 Thorne Thorne, R., M.D.: Report of Medical Officer to Local Government Board, 1886.
 (Cholera in Europe.)
 Trans. Inst. Naval Architects. (Ventilation of Ships.) 1884.

American.

- Holt, Joseph, M.D.: An Epitomized Review of the Principles and Practice of Maritime Sanitation. New Orleans, 1892.
 Maury: Physical Geography of the Sea.
 Turner, T. J., M.D., R.N.: Hygiene in Naval and Mercantile Marines. (Art. in Buck's Hygiene.)
 Wilson, Jos., M.D., R.N.: A Manual of Naval Hygiene. London and Philadelphia, 1879.

French.

- Arnould: Nouveaux Eléments d'Hygiène. (La Marine.) 1881.
 Becquerel, A.: Traité Élémentaire d'Hygiène. (Profession Maritime.) 1877.
 Bouchardat: Traité d'Hygiène. (H. Navale.) 1881.
 Boudin, J. Ch. M.: Traité de Géog. et de Statistique Méd., 1857.
 De Méricourt, Le Roy: Rapport sur le Progrès de l'Hygiène Navale, 1867.
 Fonssagrives: Traité d'Hygiène Navale. Paris, 1877.
 Levy, M.: Traité d'Hygiène. (Profession Navale, &c.) 1879.
 Mahé, J.: Manuel Pratique d'Hygiène Navale, 1874.
 Patissier, Ph.: Maladies des Artisans. (Pêcheurs, Marins, &c.) 1822.

AUTHORS NOT QUOTED.

English.

- Armstrong: De Nautarum Sanitate Tuenda. Edinburgh, 1789.
 Blane, Sir Gilbert, M.D., R.N.: Diseases Incident to Seamen, 1785.
 " " " Dissertations, 1803.
 " " " Health of the Royal Navy at end of Eighteenth and beginning of Nineteenth Century, 1880.
 Cameron, A. C., M.D.: Carbonic Acid in the Air of Canal Boats. Chem. News, vol. xxx. 1874, S. 169.
 Cockburn: Sea Diseases. London, 1693.
 Dublin Journal of Medical Science, 1838. Healthfulness of Iron Ships.
 Finlayson, M.D., R.N.: On the Baneful Influence of so frequently Washing Decks, 1828.
 Fletcher (Works of).
 Gillespie: Observations on Diseases on H.M. Squadron, 1808.
 Hayne: Carbonic Acid in the Air in Wooden Frigates. Med. Chir. Inst., 1874.
 Hunter, Jno., M.D.: Observations on the Dietaries of British and Foreign Seamen.
 Leach, Harry, M.D.: Hygienic Condition of the Mercantile Marine in the Port of London, 1871.
 " " Half-yearly Reports of Medical Officer of Health to the Port of London, 1873 onwards.
 Macdonald, J., M.D., R.N.: The Ventilation of Ships, especially of Low Freeboard. London, 1874.
 Naval Science (April 1872). Art. 'Sanitary Science Afloat.'
 Plimsoll, S., M.P.: Our Seamen, 1873.
 Pringle: Discourse on the Health of Mariners, 1776.
 Benwick: An Inquiry into the Nature and Causes of Sickness in Ships of War, 1792.
 Sanitary Record, Aug. 15, 1883. Art. 'Quarantine.'
 Saunders, Dr.: Hygienic Hints.
 Saunders, W. M., M.D., R.N.: Hygienic Manual and Surgical Hints for Naval Officers, 1856
 Ships Detained as Unsafe under the Merchant Shipping Act, 1876. Parliamentary Return, 1888.
 Smyth, Admiral: The Sailor's Word-Book.
 Trotter: Medicina Nautica, 1793-1803.
 Turnbull: Naval Surgeon, 1876.

American.

- Bulletins of National Board of Health: On Quarantine. Washington.
 Gihon, A. L., M.D., U.S.N.: Practical Suggestions on Naval Hygiene. Washington, 1871.
 Horner: Diseases and Injuries of Seamen. Naval Hygiene. Philadelphia, 1854.
 Reports to Bureau of Medicine and Surgery by Medical Officers of United States Navy.
 Washington, 1872 and onwards.

German.

- Bohlfs: Gemeinfassl. Heilk. u. Gesundheitslehre für Schiffsofficiere, 1873.

Italian.

- Roupe: De Morbis Navigantium, 1764.
 Trompeo: Cenni sull' igiene della gente marina. Torino.

French.

- Barthélemy: Etudes sur la Nature et les Causes des Lésions Traumatiques à bord des Bâtiments de Guerre suivant les Professions. (Archives de Médecine Navale, III, 1865.)
 Beaumanoir: Essai sur la Ventilation des Transports, 1875.
 Bourel-Roncière: Etudes sur les Appareils Distillatoires de la Circ. (Archiv. de Méd. Nav., xiv. 1870.)
 „ Contributions à l'Hygiène de Cuirassés. (Archiv. de Méd. Nav., xiii, 1875.)
 Buez: L'Organisation du Service sanitaire dans le Levant, &c., 1874.
 Chastang: Hygiène du Soldat appliquée aux Troupes de la Marine, 1873.
 Chirac: Obs. générales sur les Maladies des Equipages des Vaisseaux, 1724.
 Daolnai: Précis d'Hygiène Navale, 1827.
 Decante: Plan de Ventilation à l'Usage des Transports de Troupes. (Rev. Maritime & Coloniale, juillet 1872.)
 Delivet: Précis d'Hygiène Navale, 1808.
 De Méricourt: Notes sur l'Influence de la Transformation des Constructions Navales sur la Santé des Equipages. (Bull. de l'Académie Impériale de Médecine, 1866.)
 „ Influences des Constructions Navales sur la Santé des Equipages, 1866-7.
 Dutrolau: Des Modifications dans l'Hygiène Navale par l'application de la Vapeur à la Navigation, 1864.
 Fleury: Obs. pratiques d'Hygiène et de Médecine Navale. Montpellier, 1847.
 Forget: Médecine Navale, 1832.
 Foucat: La Navigation Transatlantique de nos Jours. (Arch. de Méd. Nav., 1868.)
 Holsbeek: Précis d'Hygiène et de Médecine Navales, &c. Bruxelles, 1860.
 Kerandren: Art. 'Navigation' in Dict. des Sc. Méd., 1808.
 „ Maladies des Marins, 1817.
 „ 'Nourriture des Equipages' in Ann. d'Hygiène, 1829.
 Lapparent: Du Déperissement des Coques de Navires. Paris, 1862.
 Lefèvre, A.: Recherches sur les Causes de la Colique Séche observée sur les Navires de Guerre Français. Paris, 1859.
 „ De l'Emploi des Cuisines et Appareils Distillatoires dans la Marine, 1861.
 Maréchal, J.: Considérations Médicales sur les Apprentis Canonniers du Vaisseau d'Ecole 'Louis XIV.' (Archiv. de Méd. Nav., ix. 1868.)
 Maroin: Hist. Méd. de la Flotte Française pendant la Guerre de Crimée, 1861.
 Mathelin: De la Prophylaxie du Scorbut dans la Marine Marchande. (Bull. de la Soc. de Méd. Publ., 1879.)
 Mauran: Avis aux Gens de Mer sur leur Santé. Marseille, 1786.
 Mélier: Rapport sur l'Epidémie de Fièvre Jaune de Saint-Nazaire. (Mém. de l'Académie Impériale de Méd.)
 Pallois: Essai sur les Scorbut (Hygiène Navale, &c.), 1801.
 Péron: Application des Observations Météorologiques à l'Hygiène Navale, 1808.
 Pingrenon: Manuel des Gens de Mer, &c., 1780.
 Poissonnier-Desperrières: Traité des Maladies des Gens de Mer, 1780.
 Quémar, C.: Etude sur les Conditions Hygiéniques des Bâtiments cuirassés. (Arch. de Méd. Nav., t. v. p. 462), &c., &c.
 Quermelenc: Hygiène des Diverses Professions à bord des Navires, 1860.
 Rey: Les Mécaniciens et les Chauffeurs à bord des Navires de l'Etat, 1862.
 Sunard: Statistique Médicale de la Marine Anglaise, 1864.
 Taulier: De l'Alimentation du Marin, 1873.

MILITARY HYGIENE

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MILITARY HYGIENE

THE term Military Hygiene is used to signify the care of troops. A large body of men are employed by the State, and placed by it under its own social and sanitary conditions, while at the same time it removes from them much of the self-control connected with hygienic rules which other men possess. It is, therefore, bound by every principle of fair and honest contract to see that these men are in no way injured by its system (Parkes).

It is important that every officer in a regiment should know something of the rules laid down to prevent the spread of those diseases to which the soldier is liable at home and abroad, and of the sanitary conditions which tend to keep him effective under the varying circumstances in which he may at any moment be placed. As an army depends entirely on the physical character of the men who compose it, it is necessary briefly to refer to this part of the subject first.

THE SELECTION OF SOLDIERS

The British Army is enlisted on the voluntary system. The terms of enlistment are for 'long service,' i.e. twelve years' Army service, or 'short service,' i.e. seven years' Army service and five years in the Reserve, which may be extended to eight years' Army service and four years in the Reserve, if the period of Army service expires while a man is serving abroad. In the Foot Guards, short service consists of three years' Army service and nine years in the Reserve. The limits of age at which recruits are taken are from eighteen to twenty-five years, except for boys who are enlisted as drummers. Recruits must also be of a certain height, which varies for the different arms of the service—that is, for Cavalry, 5 ft. 6 in. to 5 ft. 11 in.; for Engineers, 5 ft. 4 in. to 5 ft. 6 in.; for Artillery, 5 ft. 4 in. to 5 ft. 6 in.; for the Foot Guards, 5 ft. 8 in. and upwards, and for the Infantry of the Line, 5 ft. 4 in. and upwards. A certain minimum girth of chest according to height is required, and also a certain minimum of weight; the chest measurement must not be less than 38 inches, nor the weight less than 115 lbs. For Cavalry and Artillery this weight is too small; experience has shown that the minimum weight for these branches of the service should not be less than 125 lbs., as this is the lowest weight that would give a Cavalry soldier power at once to control his horse and wield his weapon, or a driver strength to manage a pair of horses. In the event of war, the measurements are all reduced according to the demand for men, and even in times of peace the same standard is not always maintained.

Before the enlistment is completed, the recruit is carefully examined by a medical officer of the regular or auxiliary forces according to a scheme laid down in the *Medical Regulations*. The examination is a strict one, and aims at investigating, as far as possible, the mental condition, the senses, the absence of any infirmity or injury likely to interfere with his duties as a soldier; the condition of the heart, lungs, and abdominal organs generally; the general formation of the body; the condition of the joints; the state of his feet; the absence of hernia, varicocele, &c., and his power of vision for long ranges, &c. Of the recruits examined in the United Kingdom from 1885 to 1888, there were 48·42 per cent. rejected, while during the same period in London 47·17 were found unfit. Of the causes for rejection in

London, defective vision gave 10·84 per cent. ; under height, 5·59 ; under weight, 12·62 ; and under chest measurement, 40·40 per cent. ;¹ thus 70 per cent. are included under these four headings, and what is found in London may also be accepted for other parts of the United Kingdom, as these recruits are drawn from every part of the country.

As regards *age*, many competent officers consider that no recruits should be enlisted under twenty to twenty-one years. This opinion is based on the fact that the most effective armies are those in which the youngest soldiers have been over twenty-two years of age. At eighteen the bones are not fully formed, nor do the muscles reach their mature growth much before twenty-five years ; while thus undeveloped and immature, as they must be at eighteen years, it is useless to expect any long-continued exertion or energy from men at that age. If enlisted, the State should recognise this, and suit the work to their strength ; at eighteen, recruits have not only to work, but to grow and develop, and they should have precisely the amount of exercise and kind of work best fitted for them.

As regards *vision*, the experience of the London recruiting officers is that imperfect or defective vision increases with ascent in the social scale ; it is, on the whole, less perfect among the better than the lower class of recruits, and in town than in country.

Sir W. Aitken² pointed out the importance of the correlation of height, weight, and chest measurement, in estimating physique as a whole ; good weight for height being of the first importance. An easy rule is that up to 5 ft. 7 in. thrice the height in inches ought to be about the weight in pounds ; and add 7 lbs. for every inch above 5 ft. 7 in.

It has also been observed that a close correlation exists between the physical and moral development of men ; in fact, lowering the physical means lowering the moral standard of recruits, or, as Dr. Beattie states it, if we dip too low for our recruits, we shall be liable to get men, not only small, but unsteady, wanting in mental ballast as well as in physical height. The nerves and muscles are built up by the same processes of nutrition, and the weighing machine is the best of all means we have for testing the general fitness of the recruit.

The measurement of the 'chest capacity' is of great importance in determining the vigour of the recruit. From a large number of observations made at St. George's Barracks,³ it has been found that the maximum expansion of the chest of a man of average size, between eighteen and twenty-five years of age, is about 2 to 2½, rarely 3 inches. The method adopted for ascertaining these measurements is as follows :—On carefully adjusting the linen tape over the point of the shoulder-blades behind and above the nipples in front, the recruit is directed to take a deep breath and expand himself to the utmost ; this being done two or three times, the maximum expansion is ascertained ; the minimum is found by deducting 2 to 2½ inches according to the height and general physique of the man. The minimum and maximum are then recorded above each other, as $\frac{88}{85}$ or $\frac{84}{86\frac{1}{2}}$ as the case may be.

Dr. Seggel⁴ gives the measurements he had taken of soldiers enlisted at Munich during a period of five years. His results correspond almost entirely with the measurements given by Frölich and Vogl. They are shown in the following table :—

¹ *Journal United Service Institution*, 1889.

² *Growth of Recruit and Young Soldier*. By Sir W. Aitken, M.D., F.R.S.

³ *Journal of the Royal United Service Institution*, 1889.

⁴ *Trans. International Medical Congress*, Berlin, 1890.

—	Seggel	Vogl
Average height	1.6086 m.	1.67 m.
„ weight	64.3 kilos. (129.6 lbs.)	68.2 kilos. (129.4 lbs.)
„ chest measurement	0.848 m.	0.848 m.
„ chest expansion	7.3 c.m.	7 c.m. (Frölich)
„ width of shoulders	41.1 c.m.	—
Antero-posterior lines of chest (sagittal measurements)	$\left\{ \begin{array}{l} a. 23.7 \text{ c.m.} \\ b. 21.4 \text{ c.m.} \\ c. 18.7 \text{ c.m.} \end{array} \right.$	— — 18.7 c.m.

Dr. Seggel arrives at the conclusion that the width of the shoulder is an important measurement to make in examining soldiers. He takes the measurements with the arms hanging at the sides or held straight out in front of the body; the width of the shoulder should not be less in a properly built man than two-ninths of the man's height, the best minimum to take being one-quarter of the height. The antero-posterior diameter of the chest is measured at three points—the superior border and middle of sternum and tip of ensiform cartilage. Dr. Seggel found that the greater this sagittal measurement, the greater was the chest expansion.

In the French ¹ Army the minimum height was fixed in 1887 at 66 inches (1.70 metre) for Cuirassiers and 60 inches (1.54 metre) for Infantry of the Line.

In the United States Army the minimum height is 5 ft. 4 in.; maximum height for Cavalry, 5 ft. 10 in.; the minimum weight being 128 lbs., the maximum 190 lbs.

BARRACKS

The selection of the site is of the first importance. Sites should be so selected as to secure a fall from the building in one direction at least, and if possible in more, for this will facilitate drainage, and natural drainage outlets should always be provided for. A perfectly free circulation of air should prevail around the buildings. Aspect should never be sacrificed to prospect. In England the south-east is the best aspect, for it is least exposed to rain and boisterous winds. The soil should be porous; clay soils and all retentive soils should, if possible, be avoided. The level of the ground water should be noted, and when this is near the surface the site should be drained as far as possible, to lower its level and to prevent changes, either in a rise or fall, taking place. Provision must also be made for the rapid and effectual removal of all water from the buildings, so that there may be no dampness. In order to test the healthiness of a site an inquiry into the rate of sickness and mortality in the district will afford valuable information, and the nature of the prevalent diseases should, if possible, be ascertained.

In the tropics and in sub-tropical countries all these conditions are of even greater importance. The following sites, which are proved by experience to be unhealthy, should be avoided:—

1. Clay soils, especially in India.
2. Ground at the foot of hills or in deep valleys or ravines which receive the drainage from higher levels.
3. Ravines are always dangerous, as are also elevated sites near them. Malaria is carried up through them by air currents, and generally they are receptacles for decaying and rank vegetation.
4. Any ground covered with rank vegetation, as where this exists the

¹ *La Vie du Soldat*. By Dr. E. Ravenez, 1889.

subsoil water is close to the surface and there is usually much decaying matter.

5. Low-lying banks of rivers or any grounds subject to periodical flooding, and especially any marsh lands partly covered with salt and fresh water. The removal of town barracks to country districts is not always possible. Military reasons must determine the position to be occupied by a military force, but whenever barracks can be placed in the open country, such positions should, if possible, be selected in preference to sites in town districts. for although it is not always possible to assign the precise influence which the position of barracks exercises on the health of troops, there is no reason to doubt that barracks located in close unhealthy neighbourhoods are influenced by the same conditions which govern health in such neighbourhoods. More especially is this the case with regard to hospitals, on account of the great susceptibility of sick men to the effects of impure air. It is obvious that when barracks have to be placed in towns, the buildings should be placed over as wide an area as possible.

Construction of Barracks.—The plan on which barracks were formerly built in Great Britain, Ireland, and the Colonies exhibits every possible variety both as regards their design and internal arrangement. In many cases the chief object in their construction appears to have been to place as many men as possible on the ground at the disposal of the engineer who designed them. Since the Royal Commission on the Sanitary Condition of Barracks and Hospitals issued their report and pointed out the errors made in this respect, a great improvement has taken place, and now all barracks are built on a standard plan, with such modifications as are necessarily required according to locality and climate.

In the construction of a barrack, where a number of men must be massed together, it is of the first importance to keep the air in the immediate vicinity of their rooms as pure as possible; therefore, all latrine accommodation, baths, kitchens, &c., should not form part of the buildings but should occupy separate sites away from them. The buildings themselves should have free external ventilation all round, and so placed in temperate and cold climates as to receive as much sunlight as possible. In this country the best arrangement is a single line, lying north and south, so as to allow the sun to shine on both sides during the day.

If the arrangement is in the form of a square, the angles of the square should be left open to allow of the circulation of air. Free access of sunlight should be provided for.

Barracks are best constructed of only two stories. The ground floor may be used for libraries, day rooms, or administration purposes; but basements should never be occupied by men as barrack rooms; they are always liable to be damp, and the air in them is generally stagnant.

Each range of barracks should consist of separate houses, completely independent of one another. Where houses abut, the party walls ought to be carried above the roof.

Each house should be divided up the middle by a large staircase, extending to the top and ventilated through the roof. This will prevent the air of opposite barrack rooms intermingling.

The limit of size for a barrack room is—Length, 60 ft.; breadth, 30 ft.; height, 12 ft. This size of room will give 14,400 cubic feet, and is intended for twenty-four men. Each man has, therefore, 600 cubic feet of space. As some of this, however, is taken up with furniture, &c., an additional 2 ft. has been allowed to the length in all the new barracks. If the length is 62 ft. each man will have 52 superficial feet of space. At one end of the room is the door leading

on to the central staircase and a room for the sergeant of the section, 14 ft. long, 10 ft. wide, and 12 ft. high. At the other end is a narrow passage leading to an ablution room, one basin and a urinal being provided for every four men. In some of the latest barracks the lavatory and urinals are placed

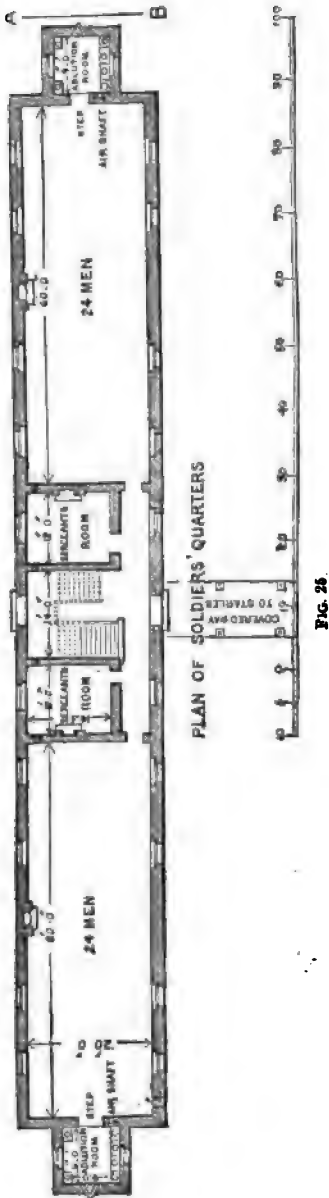


FIG. 26

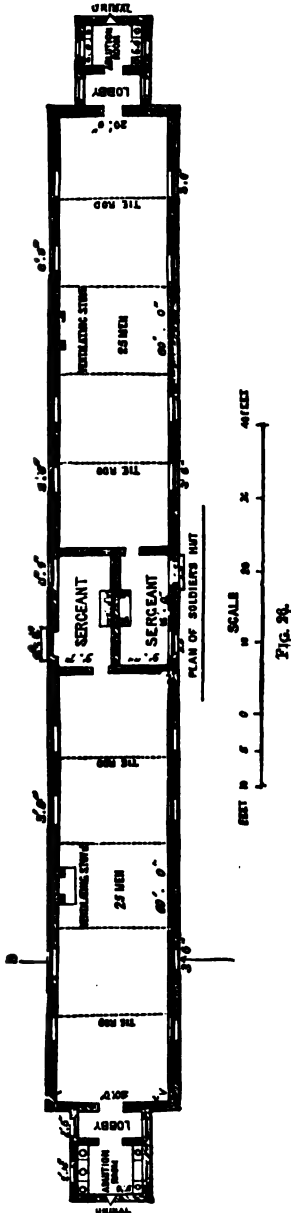


FIG. 28

near the head of the staircase, in the centre of the building; this is, without question, a retrograde step.

Usually two such barrack rooms are placed in a line, the lavatories at the free ends. A house therefore contains a company in four rooms, if it is double storied, and ten such houses will suffice for a regiment. Each room

is provided with two ventilating shafts, also inlets for fresh air between the windows and a ventilating fire-grate (Galton's).

As a rule, the number of windows is half as many as the number of beds; they are on opposite sides of the room, and carried up to within a few inches of the ceiling.

Cavalry Barracks.—In many of the older barracks the men's rooms were placed over the stables. The supposed advantages gained by this arrangement were (1) increased cubic space; (2) less exposure to the men in passing to their stables, and greater convenience.

The Barrack Improvement Commissioners have, however, shown that it is impossible to ventilate satisfactorily a stable accommodating a large number of horses if anything beside the roof is interposed between the stable and the outer air, and that it is equally impossible to keep the air in men's rooms over stables pure and free from stable odour.

The new model troop stable is arranged for forty-eight horses. The length of the stable is 148 ft. 8 in.; breadth, 38 feet; height of side walls to spring of roof, 12 feet; total height, 20 ft. 6 in. Each horse is arranged to have 1,605 cubic feet of air and 100 superficial feet of space. The stalls are 5 ft. 6 in. wide, 9 ft. 6 in. in length, and the width of the passage between the stalls is 14 feet. Over the head of each horse is placed a window 8 ft. 4 in. high by 2 ft. 6½ in. wide; an air brick is placed between every stall 6 inches from the ground, and a course of air bricks is carried around the eaves of the building. The roof is open at the ridge by means of a louvre, which allows 4 square feet of ventilating outlet per horse, and a continuous skylight is carried along on one side of it.

Non-commissioned Officers' Rooms.—Warrant officers and schoolmasters are entitled to two rooms and a kitchen. When possible their quarters will be separated from those of the non-commissioned officers and men. Married senior non-commissioned officers are entitled to two rooms; other sergeants have one room each. The rooms are about 14 feet long, 12 feet in breadth and 10 feet high, giving, when empty, 1,680 cubic feet.

Married Soldiers' Quarters.—Each married soldier is entitled to one room 14 feet by 12 feet, giving 168 superficial and 1,680 cubic feet of space. No man under the rank of sergeant is allowed to marry unless he has completed seven years' service. With the present short service system the number of married soldiers is comparatively few, and generally there is no difficulty in providing them with two rooms each. There are also separate latrines provided for females, and a wash-house adjacent to their quarters.

Guard Room.—This room is about 24 feet long, 18 feet wide, and 14 feet high. Two rooms open out of it, capable of being overlooked from the guard-room—one for prisoners, the other for men who are awaiting trial. It affords 600 cubic feet per man, and is ventilated on the same principle as barrack rooms.

Ablution Rooms.—These are now provided in every barrack, with clean water laid on and basins in the proportion of one to every four men. The basins are either of slate or iron. In many instances baths are also provided. The Barrack Commissioners recommended that one bath be provided for every 100 men.

Kitchens.—These should not be under the same roof as the barrack rooms, but at no greater distance than 100 yards, so as to allow the dinners to be brought hot to the men. Cooking is now frequently done by steam, but ovens are used when the rations are baked. The amount of coal used is generally about 10 lbs. per week for every 7 men.

Ventilation of Barrack Rooms.—In each barrack room ventilating shafts are provided to act as outlets, the sectional area of the shafts being dependent

on the cubic contents of the room, but it is not made larger than one square foot; if more outlet is required, another shaft is put up.

In rooms on the top floor of a barrack a sectional area of 1 inch is allowed for every 50 cubic feet of room space; for floors next below the upper floor a sectional area of 1 inch to 55 cubic feet of room space; and when the barrack consists of three floors, the lower floors have a sectional area of 1 inch to 60 cubic feet of room space.

The movement of air in these shafts is tolerably regular. Each shaft removes about 600 cubic feet per man per hour, the usual current at night being from 8 to 5 feet per second.

These foul-air shafts are carried from one angle of the ceiling to 8 or 4 feet above the roof, and are protected by louvres to prevent the air beating down. The shafts are made of $\frac{3}{4}$ inch deal, very smooth inside, and rebated and grooved together at the angles.

In addition to these shafts there is the chimney, which gives a sectional area per head of about 6 square inches. This removes about the same quantity of foul air as the ventilating shafts, so that each man is provided with from 16 to 18 square inches of outlet area, capable of removing 1,200 cubic feet of air per man per hour.

Inlets for Fresh Air.—All inlets admitting air direct are placed near the ceiling. The form adopted is a perforated air brick of different sectional areas, according to the number of men the room is intended to contain. The sectional area adopted allows 1 square inch for every 60 cubic feet of contents of the room, but if warm air is admitted round the fire-grate and distributed, as will be presently described, 1 square inch to every 120 cubic feet of contents is sufficient.

The air is delivered into the room through valves either louvred or hopper-shaped. In the latter form the air is deflected towards the ceiling. The upper side of this valve is formed of perforated zinc, the area of which is from six to eight times the area of the inlet from the outer air. Area of outer opening = 5 square inches per man.

The outlet and inlet shafts should be placed as far from each other as possible, to enable thorough diffusion of the inflowing air to take place. The best position for the foul-air shafts, however, is at one side or the other of the fire-place and not opposite to it.

Warm air is also provided for by the following plan. Air is admitted through a shaft from the external air to the air chamber at the back of the fire-place. This shaft or tube should contain 1 superficial inch of sectional area for every 100 cubic feet of room space. Care should be taken to draw the air from a pure source. From the air chamber the air is conducted into the room by a shaft and through a louvred opening placed as near the ceiling as possible; the louvres being bevelled upwards so as to cause the air current to impinge against the ceiling. Area of tube, 6 square inches per head; total inlet area = 11 square inches per man. By this system each room is ventilated by itself, and independently of any other room, and 1,200 cubic feet of fresh air per man per hour in a room space of 600 cubic feet per man is provided.

Water Supply.—By decision of the Secretary of State for War, a soldier receives 15 gallons daily. No extra allowance is made for wives and children in a regiment.

BARRACKS IN HOT CLIMATES

The Indian Sanitary Commission have prepared standard plans suitable for different localities in India, and, while the detailed design is left to local

officers, certain general principles are strictly laid down. The number of men to be placed under one roof is fixed at 40 or 50; the number of men in one room to be about 16, but in no case to exceed 24. The barracks are to be two-storied in the plains, and one- or two-storied in the hills, both floors being used as dormitories. Single verandahs of 10 or 12 feet wide surround these rooms. In the plains each bed has $7\frac{1}{2}$ ft. of running wall space, in the hills 7 ft.

The Indian Army Regulations sanction the following scale of superficial and cubic space for European and Native troops in India. *In the Plains*: Superficial space, 90 ft.; cubic space, 1,800 ft.; height of room, 20 ft.; width, 24 ft. *In the Hills*: Superficial space, 60 ft.; cubic space, 600 ft.; height of room, 10 ft.; width, 22 ft.

Married quarters are provided in one-storied blocks. Two rooms (with verandah) are provided for each family. The rooms are 16 ft. by 14 ft. and 14 ft. by 10 ft.

The same principles of ventilation are applied to these barracks as to the barracks at home. The area of the shafts is ordered to be 1 square inch to every 15 or 20 cubic feet; in the lower rooms the shafts are to be built in the walls; in the upper rooms to be in the centre. The number of doors and windows render it unnecessary to provide special inlets for fresh air; outlets should be placed as at home, or, if one-storied buildings be adopted, ridge ventilation may be substituted for shafts.

In all barracks punkahs are used at certain seasons of the year. They assist in ventilation by displacing masses of air. When the weather is dry and hot, tatties are used to cool the air; but they can only be made available for this purpose when the air is moving and dry. The evaporation of water is the great cooling agency.

The temperature may be reduced 10° to 15° by allowing the hot dry wind to blow through wet kuskus tatties. When the air is stagnant thermantidotes may be used, and if the air is dry, thin wet mats made of kuskus grass are suspended in the discharge tubes; or blocks of ice may be placed in the channel near the outlet opening. The latter plan is the only available one when the air is moist.

In India almost every barrack has its plunge bath: the supply of water is in most cases unlimited. Washhouses are also provided on the same plan as adopted at home.

The system of excrement disposal generally adopted throughout India is the 'dry earth' system. The latrines are well kept, and their contents regularly removed. As regards barracks the system answers well, but great care is required to see that the contents are disposed of properly.

The receptacles are emptied into closed carts twice daily; but the natives who perform this duty require close watching, otherwise they are apt to deposit the contents of their carts on the surface, in place of in trenches. The earth supplied for the purpose of deodorisation must be clean and absorbent. Sand is of little use for this purpose.

HUT BARRACKS

The first essential consideration is to secure a dry subsoil, as dry porous soil is absolutely necessary to health, and the ground should be effectually drained to such a depth as will free the subsoil water.

The hut should be so placed as to be free of all surrounding higher ground, and under no circumstances should any part of it be bedded in the ground, otherwise damp is sure to find its way under the flooring.

The ground occupied by a hut should be cleared, levelled, and drained. The hut should be provided with ridge ventilation and projecting eaves to carry off the rain-water from the foundations; it should have the requisite number of windows, and should be raised sufficiently above the ground to allow a free current of air to pass underneath the flooring. In hot climates the roof and sides should be double, if these latter are not protected from the sun by verandahs.

Huts are best placed *en échelon*, so as to receive the full advantage of winds.

Ventilation is effected by openings in the ridge, or outlet shafts may be used, passing through the roof and terminating in louvres and inlets under the eaves.

Warming may be effected by the use of stoves or an open grate. The latter is preferable, as it assists in ventilation. The construction of huts depends on whether they are used for temporary purposes or whether they are intended to be of a more or less permanent character. In the latter case the sides are usually built of brick.

In the German Army the Döcker huts are largely used, and are said to answer well. They have recently been favourably reported on in this country. They are made of wooden or iron frames, covered with a special kind of felt, lined with canvas. They are very portable, and the fastenings are so arranged that they can be put together in a very short time. These huts are well ventilated by windows, cross louvres, and ridge ventilation, and can be easily warmed, if this is desired.

Lord Wolseley recommends that temporary huts on service should be constructed to hold twenty-eight men, and of the following proportions: Length, 32 feet; breadth, 16 feet; height to eaves, 6 feet; height to ridge, 16 feet. The cubic space should be 400 cubic feet per man. Two such huts are placed end to end with one chimney between them.

The roof may be made of felt or tarred calico, secured by strips of wood. In the tropics, if the rainfall is heavy, the roof should be made steep, to throw off the rain.

If the flooring is made of wood, it should be fastened by screws and not nails. This will allow the boards to be taken up, if necessary, and the space beneath cleaned. If the floor is of earth, a little of the surface earth may be removed occasionally and replaced by clean gravel. Ashes from wooden fires, well rammed down, make an excellent floor.

CAMPS

The worst site for a camp is clay soil, or a clay subsoil coming near the surface. Such soils are retentive of water, and keep the atmosphere over them damp. They should therefore, if possible, be avoided. Ground immediately at the foot of a slope is apt to be damp and unhealthy, on account of receiving water from the higher levels. In tropical climates, localities exposed to winds blowing over low marshy ground are unsafe on account of malarial fevers; for the same reason elevated sites on the margin of steep ravines, up which malaria may be carried by air currents, are apt to be unhealthy, as are also deep narrow valleys or gorges covered with dense vegetation.

Ground covered with rank vegetation, especially in the tropics, is unhealthy, partly on account of the amount of decaying matter in the soil, partly because the presence of such vegetation is in itself a mark of the presence of a high subsoil water or of a humid atmosphere. In hot climates, the banks of rivers, especially if the water is stagnant, marsh land, and

TENTS

The essentials of a good tent are—(1) that it should be waterproof; (2) that it should be ventilated by large outlets near the roof; (3) that in the tropics a double fly be provided; (4) that it should be as light as possible; (5) that the colour should not be easily seen by the enemy. The following are the tents used in the British Army on home service :—

The Circular or Bell Tent.—A round tent, with sides straight to one foot high and then slanting to a central pole. Diameter of base, 12·5 ft.; height 10 ft.; area of base, 128 square feet; cubic space, 492 cubic feet. Weight, when dry, including poles, about 72 lbs. and wet about 89 lbs. The canvas is made of linen or cotton. This tent holds from twelve to sixteen men.

The Hospital Marquee.—A quadrangular tent, with two poles and double canvas. Length, 80 feet; breadth, 15 feet; height of sides, 5 ft.; height to ridge, 15 ft.; area of base, about 985 square feet; cubic space, 3,396 cubic feet. Weight, when dry, 512 lbs. and wet, 660 lbs. This tent is provided with a waterproof sheet (weight, 145 lbs.) to lay on the ground, and is intended to hold from ten to sixteen patients; with the latter number there is some overcrowding. Ventilation is by an opening in the roof; the sides also can be raised, if required. This tent is now only used at the base of operations when buildings are not available: it forms no part of the movable field equipment. Lord Wolseley condemns this tent as cumbersome, excessively heavy, and difficult to pitch.

Circular Tent.—A double circular tent, with higher walls and without lining, has been approved for hospital purposes. It weighs about 100 lbs. and is intended to accommodate four sick or wounded men. This tent forms part of the new field equipment.

Shelter Tents.—There is no official shelter tent for the English Army.

In India the following tents are in use :—

British Privates.—With two poles and ridge, double fly. Length 20 ft.; breadth, 16 ft.; height of walls, 5 ft. 6 in.; height to ridge poles, 10 ft. 6 in. Cubic space, 2,873 cubic feet. This tent is used for inland service, and accommodates sixteen healthy men or eight sick.

Mountain Service.—With two poles and ridge. Length, 12 ft.; breadth, 8 ft.; height of walls, 10 in.; height to ridge poles, 8 ft. Cubic space 544 cubic feet. This tent is used for field hospitals to accommodate four sick.

General Service.—With three poles and ridge. Weight, 160 lbs.; length, 14 ft.; breadth, 14 ft.; height of walls, 1 ft.; height to ridge pole, 7 ft. Cubic space, 686 cubic feet. This tent is used for field service, and accommodates 16 British or 20 Native soldiers, or 25 followers.

General Service (small).—With two poles and ridge. Weight, 80 lbs.; length, 8 ft.; breadth, 14 ft.; height of walls, 1 ft.; height to ridge pole, 7 ft. Cubic space, 392 cubic feet. This tent is used for field service, and accommodates 8 British or 10 Native soldiers, or 12 followers.

In the French Army the following are the tents in use :—

The *tente d'abri*, or shelter tent, intended for 8 or 4 men. It is heavy, and is now only used beyond the confines of Europe.

Tente Conique or *Tente à Marabout*.—Intended to hold 16 men. Weight, 129 lbs.

Tente Conique et à Murailles.—Intended for 16 men. This is a circular tent and is specially ventilated at the top. An iron ring 12 in. in diameter receives the canvas which is sewn round it. The opening can, if required,

be closed at the top by a wooden rest which slides on the top of the pole; this opening gives 118 square inches for ventilation.

In the German Army the following tents are in use:—

A conical tent, with a single pole, like the English bell tent. Height of pole, 12 ft.; diameter at base, 15 ft.; weight 88 lbs. Superficial space per man, 12 square feet; cubic space per man, 70 cubic feet. This tent is intended for 15 men.

Shelter Tent.—The German Army has adopted a shelter tent for use on field services; the component parts of the tent-poles and canvas are distributed among as many men (two at least) as are meant to be sheltered by it. The canvas part is carried rolled round the soldier's overcoat, which is strapped down on the top and sides of the knapsack, and in bad weather this tent section may be unrolled and worn as a watertight *poncho* by the bearer.

German Hospital Tent.—This is a rectangular tent. Length, 29 ft. 9 in.; breadth, 24 ft. 6 in.; height, 18 ft. 9 in.; superficial space, 728 square feet; weight, 952 lbs. It is divided by curtains into three parts, a central room for sick and two rooms for attendants, utensils, &c. The tents are made with a wooden framework, and there is a hood for ventilation. Each tent receives only 6 patients. It stands on an area of 58 ft. by 43 ft.

In the field, if no suitable building is available in which operations can be performed, a bandaging tent is erected. It is rectangular in shape; length, 18 ft. 6 in.; breadth, 11 ft. 6 in.; height, 8 ft. It has a single fly, and is made entirely of waterproof canvas.

In the Russian Army, a quadrangular tent 14 ft. square and 7 ft. high is used; there is a centre pole, and four corner poles. Twelve men usually occupy this tent.

The United States War Department have adopted the following tents:—

1. Conical (modified Sibley), 16 ft. 5 in. in diameter at base; wall, 8 ft.; apex, 10 ft.; floor, 212 square feet; air-space, 1,450 ft.; allowance, 20 Infantry or 17 Cavalry; comfortable for camp or slow march with half that number.

2. Common ('I' or modified 'A'), wall, 2 ft.; base, 8 ft. 4 in. \times 6 ft. 10 in.; ridge, 6 ft. 10 in. from ground; floor, 57 square ft.; air-space, 250 ft.; allowance, 4 mounted or 6 foot men. Each Infantryman would have 17 inches to lie in.

3. Wall, 9 ft. square \times 8 ft. 9 in.; to ridge, 8 ft. 6 in.; floor, 81 ft.; air-space 500 ft.; covered by fly or false roof.

Hospital tents are larger wall tents (14 \times 15 \times 4½ ft.; wall, 12 ft. to ridge), that may be opened at each end and thrown together in extension.¹

MILITARY HOSPITALS

In the construction of hospitals the great points to be secured are—(1) purity of internal atmosphere; (2) abundance of pure air and sunlight within the building; (3) facility of administration and of discipline. The realisation of these principles involves the selection of a healthy site for the building; simplicity of plan and construction; a sufficient number of windows properly placed; a certain number and arrangement of wards; proper ward proportions; a suitable number of offices, stores, &c., and easy means of communication throughout the building.² The first of these conditions is met by placing the sick in detached buildings, with such an aspect as will afford the freest air and the greatest light; this is best effected in hospitals built on the pavilion plan, in which the sick can be treated in small detached and

¹ *Military Hygiene.* By A. A. Woodhull, Major, Med. Depart., U.S. Army, 1890.

² *Commission appointed for Improving the Sanitary Condition of Barracks and Hospitals,* 1861.

perfectly ventilated buildings, and where there is no possibility of the air of one ward passing into another.

The ventilation of wards in a military hospital is on the same plan as for barracks, except the dimensions are nearly doubled.

The ward unit is the foundation of the hospital plan, and the ward construction and proportions must be based on the number of cubic feet to be allowed per bed. In wards each man should have at least 90 square feet of superficial space and 1,200 feet of cubic space. This is the amount allowed by regulations at home, but, if possible, a larger space should be given. In tropical climates (exclusive of India) 1,500 feet of cubic space is allowed to each man, or an amount as may be specially authorised for each command.

The *Instructions for the Royal Engineer Department, 1888*, state the size and construction of hospital wards in the United Kingdom to be as follows:—

Ward.—Normal size for 24 beds, 87 ft. \times 24 ft. \times 14 ft. high.

Ward for two beds, 20 ft. \times 18 ft. \times 14 ft. high.

1. The regulations direct that the walls for hospitals shall be constructed on the same plan as those for barracks.

2. The bed space for two beds between two windows must not be less than 9 ft., 3 ft. per bed and 3 ft. between them. The bed space between a window

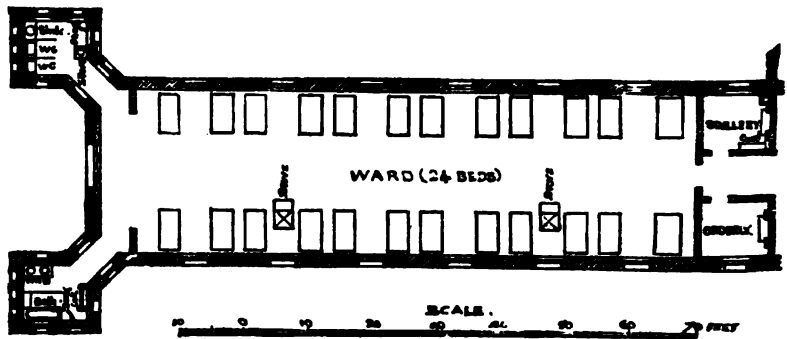


FIG. 27.

and an end wall should be 4 ft. 6 in. With the minimum distance between the windows, their maximum interval width should be 5 ft. 6 in., giving for each bed in the large wards a floor space of 7 ft. 3 in. \times 12 ft., which, with a height of 14 ft., allows 1,218 cubic feet.

The windows of large wards should face east and west. The windows should be 2 ft. 6 in. from the floor to the top of the stone sill, about 10 ft. high, and should run up to within 12 inches of the ceiling; the inner sills to be bevelled to prevent accumulation of dust. Blinds should be provided to the windows of wards.

Doors to large wards to be 4 ft. wide, hung in two, and glazed with a swing fanlight above.

The arrangement of water-closets and urinals is a matter of the greatest importance. The best plan is to throw out from one end of the ward a building to contain the closets, and connect it to the ward by an intercepting lobby. This is the plan adopted in the Cambridge Hospital at Aldershot and in all the new station hospitals. (See fig. 29.)

The following plans show the general arrangements adopted in the construction of military hospitals:—

The Herbert Hospital, Woolwich, consists of four double and three single pavilions of two floors each, all raised on basements. The administration is in a separate block in front. The wards are warmed by two central open

fire-places, with descending flues, round which are air-passages, so that the entering air is warmed. The floors are iron beams filled with concrete and covered with oak boarding.

The Cambridge Hospital at Aldershot is on much the same plan, but the closets and lavatories are thrown out in separate turrets and connected by intervening lobbies.

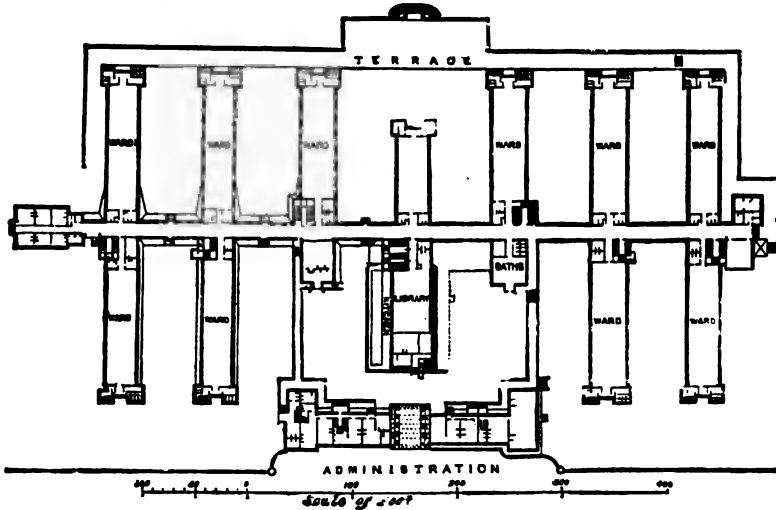


FIG. 28.

HOSPITALS IN INDIA

The *Indian Army Regulations* direct for each sick man from 102 to 120 square feet of superficial area, and from 1,680 cubic feet of space (in the hills) to 2,400 cubic feet (in the plains).

The principle and details of hospital construction are identical with those ordered for home stations.

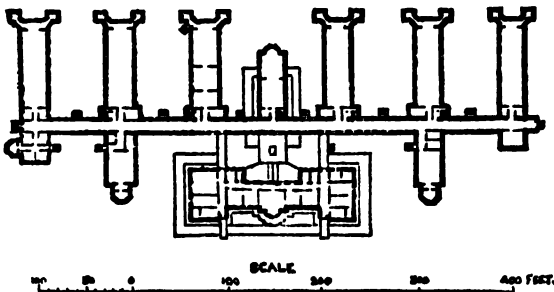


FIG. 29.

HOSPITAL ORGANISATION, &C.

Military hospitals are classified as follows:—

I. In Districts and Commands.

- (a) General Hospitals.
- (b) Station Hospitals.
- (c) Lunatic Hospitals.
- (d) Hospitals on board ships conveying troops.
- (e) Hospitals for soldiers' wives and children.

II. *With an Army in the Field.*

- (a) General Hospitals.
- (b) Hospital Ships.
- (c) Hospitals on the lines of communication.
- (d) Field Hospitals.

Subject to the officer commanding the district or station, the senior medical officer in charge of a hospital commands all officers of the Army Medical Staff and soldiers of the Medical Staff Corps attached to the hospital, as well as all patients in hospital and officers and soldiers of other corps attached to the Medical Staff Corps for duty. He is responsible for the discipline of the whole establishment.

The medical officer in charge of a military hospital is directly responsible for all the duties of the hospital: he will take care that all the instruments, medicines, hospital equipments, clothing and stores held on inventory are in good condition, sufficient according to regulation, and kept in safe custody; that the supplies are of good quality, and that the cooking and distribution of the diets are properly carried out.

The nursing duties in general and station hospitals are carried out by nursing sisters, under the immediate supervision of the superintendent or acting superintendent: they receive orders and instructions relative to the nursing arrangements from the medical officers. Nursing sisters are responsible for the personal cleanliness of the patients in their wards, and that all medicines, diets, &c., are properly issued. They also assist in training, as hospital attendants for nursing duties, the men of the Medical Staff Corps.

Field Organisation.—The organisation for the medical service for troops in the field consists of the following parts:—

1. To each Infantry regiment, regiment of Cavalry, Horse or Field Artillery division of three batteries with ammunition column, and to each Engineer company or troop, is attached a medical officer, to afford such temporary assistance to sick and wounded as may be required on the line of march, in camp, and in action. He will be furnished with a corporal, and a private as orderly from the regiment, and the trained bearers of the corps, in the proportion of two men per company, will be placed at his disposal to render first aid to the sick or wounded soldier.

2. To each brigade of Infantry and Cavalry is attached a bearer company, consisting of three medical officers and sixty-four men of the Medical Staff Corps. In action the bearer company is divided into (1) two stretcher sections, each of one sergeant and sixteen privates, and one of which is under a surgeon-captain; (2) a collecting station, under a sergeant; (3) the ambulances; and (4) a dressing station, under a surgeon-major, assisted by another medical officer.

The *collecting* station will be as close as possible to the fighting line, and, if possible, under shelter. The ambulances rendezvous here, and as they are loaded with wounded move off to the dressing station; having deposited them, they return at once to the collecting station.

The *dressing* station is usually out of range of fire. Any available shelter may be used, and there should be a supply of water at hand; if no building is available a tent must be pitched. After the wounded are dressed and beef-tea, stimulants, &c., given, they are moved in the ambulances of the second line to the field hospitals. Each bearer company is provided with ten four-horse ambulance waggons (when wheeled transport is available), of which four are intended to ply between the collecting and the dressing stations, and six between the dressing station and the field hospitals.

3. To each brigade is attached a field hospital of 100 beds, the *personnel*

consisting of four medical officers, one quartermaster, and forty rank and file of the Medical Staff Corps. This hospital is capable of being divided into two halves of fifty beds each. To each Infantry division is attached, in addition, a field hospital in reserve, and an army corps has a field hospital as well for corps details. A Cavalry division consisting of two brigades has the same provision as a division of Infantry.

For an entire army corps of 85,110 of all ranks, there are provided six field hospitals with the six brigades, a field hospital with each division, or three in all, and one other for the corps details, giving altogether ten hospitals, or 1,000 beds for 85,000 men, or about 8 per cent. of hospital accommodation in the second line.

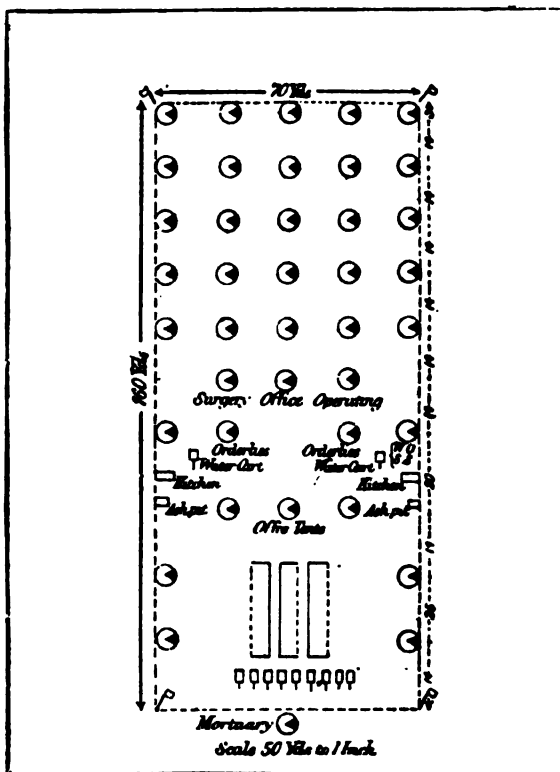


FIG. 30.—Camp for Field Hospital.

On the line of communication stationary field hospitals and general hospitals are established at as many points as may be considered necessary. On an assumed line of 100 miles in length—viz. 50 miles of railway and 50 miles of road—two stationary hospitals and two general hospitals would be required for a field force consisting of an army corps, with a division of cavalry and comprising in the field and at the base and on the line of communications a grand total of 41,815.¹ There are provided 1,000 beds in the field hospitals, 400 beds in the two stationary (200 in each), and 1,000 beds in the two general hospitals (500 in each) on the lines of communication, and 1,000 beds at the base—in all, 3,400 beds for about 8 per cent. of the whole force.

Slight cases would be treated in the field hospitals, but all cases likely to

¹ Army corps, 85,110; Cavalry division, 6,705.

be of a serious nature should be sent to the base. The hospitals in the front should be kept empty as far as possible, in order to meet any emergency and to enable them to advance with the army; if encumbered with sick this is impossible.

A field hospital is a non-dieted hospital—i.e. one in which no hospital diets are issued, but the sick have their ordinary field rations cooked for them: these are supplemented by such medical comforts as are required. Beds or stretchers form no part of the field hospital equipment.

Stationary field hospitals on the lines of communication form the third line of medical assistance. The site for these should be selected near canals, roads, or railways, for the ready removal of the sick and wounded to the base. Each hospital is for 200 patients. They are non-dieted hospitals, but if circumstances permit they may be either partially or wholly dieted.

General Hospitals at the Base.—These are dieted hospitals, and are fully equipped. Nursing sisters form part of the establishment of these hospitals, which are organised in a manner similar to a general hospital in peace.

The hospitals at the base should never be the ordinary buildings of the country adapted for that purpose. Such buildings have always been found unhealthy. Churches should never be used, as they are not only cold, but are subject to exhalation from graves in their proximity, and often beneath them.

All experience shows that the best plan is to erect huts at the base, or wherever hospitals are likely to be of a permanent character. The American Army, during the latter part of the war, discontinued converting old buildings into hospitals. They erected huts capable of accommodating 50 sick men with two rows of beds. The superficial area per bed was 87 square feet; cubic space per man, 1,200 feet.

The huts used at Suakim in 1885 were of wood, the upper half of the walls movable or provided with bamboo chicks or matting. The roof was of cork covered with Willesden waterproof paper, and ventilated by means of metal cowls. Each hut accommodated 12 men, with 850 cubic feet of space. The floor was raised 16 inches above the ground. The Döcker hut (already described) is largely used in the German Army.

Hospital Ships.—The floating hospital accommodation for an army corps consists of three depôt ships, each capable of receiving 200 sick. There are also relieving ships, capable of accommodating 60 sick, employed for the conveyance of those sick who are invalided to England; and in addition despatch vessels fitted with 80 canvas cots, for the removal of the less severe cases, which may be transported by mail packets on their way to England.

On these ships the Admiralty undertakes the lodging, victualling, and conveyance of the sick; the washing is also arranged for by them.

The War Office undertakes to furnish the medical and other attendance necessary for the proper treatment and nursing of the sick, and supplies all articles of personal and hospital clothing (but not bedding or ward equipment), medical and surgical appliances, and hospital utensils.

The fittings of a hospital ship should be as simple as possible and made of iron. Ample ventilation must be provided for, especially between decks; the method of receiving and removing the excreta of dysenteric or febrile patients must be carefully attended to; the supply of distilled water should be ample; if possible, a separate ship should be employed for washing linen, &c.; if the expedition is a large one it would not be difficult to convert a small ship into a laundry. Every possible plan should be adopted to keep the ship as dry, well ventilated, and pure as a hospital on shore.

THE FOOD OF THE SOLDIER

The *Medical Regulations* for the Army direct medical officers to examine from time to time into the quality of the various articles of food; the cooking, to ascertain whether it is sufficiently varied; and likewise the amount and quality of the drinking-water supplied to troops. With an army in the field the principal medical officer will give his advice with reference to rations, clothing, shelter, and all other points affecting the health of the men.

It will be thus seen that a medical officer may at any time be called upon to give his opinion on the quality of the food supplies tendered, on the composition of men's diet, and whether these are sufficiently varied and in proper quantity, and on their cooking and preparation.

The rations of the chief armies are as follows:—

British Soldier on Home Service

1. From the Government he receives 12 ounces of meat (including bone) and 1 lb. of bread 'free.'

2. By a stoppage from his pay (usually 8½d. per diem) he is provided by his company mess with his 'grocery ration.' This sum is expended on the purchase of extra bread, potatoes, milk, vegetables, tea, sugar, &c.

8. In addition to these there are certain individual purchases which the soldier makes at the canteen, all articles being supplied to him at cost price. The sales at various canteens show that ch  ese, bacon, preserved meats, and fish are the articles most in demand.

Nutritive Value in Ounces (Avoir.) of the Free Ration and of the Grocery Ration.

Articles	Quantity taken daily in ounces and tenths of ounces	Water	Proteids	Fat	Carbo-hydrates	Salts	Water-free food
Meat . . .	12 oz., of which one-fifth is bone	7.20	1.44	0.81	—	0.15	2.40
Bread . . .	24	9.60	1.92	0.36	11.81	0.31	14.40
Potatoes . .	16	11.84	0.32	0.02	3.36	0.02	3.72
Vegetables . .	8	7.28	0.14	0.04	0.46	0.06	0.70
Milk . . .	8.25	2.82	0.13	0.12	0.16	0.02	0.43
Sugar . . .	1.33	0.04	—	—	1.29	—	1.29
Salt . . .	0.25	—	—	—	—	0.25	0.25
Coffee . . .	0.33	—	—	—	—	—	—
Tea . . .	0.16	—	—	—	—	—	—
Total quantity .	65.32	88.78	3.95	1.35	17.08	0.81	23.19

Calculating out the amount of carbon and nitrogen in this diet, we have:—

Nitrogen	276	Grains
Carbon in proteids	837	} 4,588
Carbon in fats	454	
Carbon in carbohydrates	3,297	
Hydrogen in proteids	82	
Hydrogen in fats	65	
Sulphur in proteids	82	

This amount may be taken as the very minimum of what is supplied, and is far short of what might be afforded with good management of the messing money. In this diet the nutritive value of the bones, which form one-fifth of

the meat ration, is omitted: these, if crushed and boiled with vegetables, make an excellent and nutritious soup. The nutritive value of bones may be reckoned at one-third that of beef in carbon, and at one-sixth in nitrogen.

The following analysis shows the composition of bones in the ordinary beef ration issued at Netley:—

Analysis of Bone.

		Constituents of Proteids	
Water	12.1	Digestible proteids	10.3
Proteids	24.5	Peptones	1.9
Fat	11.0	Extractives	1.0
Ash	48.6		
Loss	3.8	Total useful	13.2
	100.00	Indigestible proteids	11.3
		Total	24.5

That the former system of purchase by the several companies in a regiment was both wasteful and extravagant has been shown by Colonel Burdett, Assistant Adjutant-General, Aldershot. His efforts to improve the soldier's messing have been most successful, and that without increased cost to the soldier. His plan is now introduced at Aldershot, and gives the greatest satisfaction. The following list shows the variety of messing provided under this system:—

Scale of Messing for the Guidance of Colour-Sergeants.¹

Coffee, 2½ oz. to 10 men (one meal).
 Tea, 1 oz. to 7 men (one meal).
 Sugar for tea, 7½ oz. to every 10 men (one meal).
 Sugar for coffee, 1 oz. per man (one meal).
 Cheese, 1 oz. per man.
 Butter, 1 oz. per man.
 Jam, 2 oz. per man.
 Marmalade, 2 oz. per man.
 Golden syrup, 2½ oz. per man.
 Brawn, 2 oz. per man (breakfast and supper).
 Corned beef, 2 oz. per man (breakfast and supper).
 Potatoes, 1 st. for every 12 men (dinner).
 " 2 st. for supper per company
 Vegetables for soups, 4 lbs. per company
 " " supper, 3 lbs. per company
 Onions for soup, 3 lbs. per company
 " " supper, 2 lbs. per company
 Salt, 2 lbs. per diem (1½ drum) per company
 Pepper, 3 oz. per diem per company
 Mustard, 3 oz. per diem per company
 Herrings, 1 per man.
 Split peas for soup, 1 lb. for 10 men.
 Lentils for soup, 1 lb. for 10 men.
 Barley for soup, 1 lb. for 10 men.
 Curry powder, 5 oz. for soups or curried breakfasts, dinners, or suppers
 per company of 60 men.
 Flour for soup, 1 lb. per diem for company for thickening.
 Celery seed for soup, one packet per diem per company.
 Milk, 1½ pint for every 10 men (one meal).
 " for tripe stew, 2 quarts per company.
 " for porridge, 1 quart for 15 men.
 Oatmeal, 1 lb. for every 9 men.
 Golden syrup for porridge, 3 tins (6 lbs.) per company.
 Bacon, 2 oz. per man.
 Eggs, 1 per man.
 The above scale to be adhered to in making up messing books.

¹ *Report of Soldier's Dietary Committee, 1899.*

The daily nutritive value of a diet provided by Colonel Burdett to the men of his regiment was as follows:—

—	Proteids	Fats	Carbo- hydrates	Nitrogen	Carbon
Breakfast	Ounces 0.9922	Ounces 1.2400	Ounces 5.2663	Grains 69.454	Grains 1637.6
Dinner { Soup	0.9126	0.2992	1.2227	63.882	531.2
Meal, &c. . . .	2.1657	0.9073	6.0503	151.599	1937.7
Pudding and pie . .	0.1765	0.3238	1.1282	12.855	365.2
Supper	1.0401	0.4676	5.2867	72.807	1395.3
Total	5.2871	3.2379	18.9542	370.097	5867.0

In this scale we have—

Proteids	5½ ounces
Fats	3½ „
Carbohydrates	19 „
Nitrogen	370 grains
Carbon	5,867 „

The Committee on Soldiers' Dietary, 1889, came to the conclusion that the Government 'free' ration, supplemented by a messing contribution of from 8d. to 4d. per day, is, under proper management, sufficient to provide an ample diet, and that the chief defects in the soldier's diet are due to insufficient interest being taken in it.

The quantity is judged to be sufficient. In support of this they quote the instance of Pearce's Dining and Refreshment Rooms—an institution supplying 80,000 meals daily to the working classes—where the average amount of meat, without bone, supplied to each man for dinner was 5 oz. uncooked, yielding when cooked about 4 oz.; whereas at Aldershot, where 1,232 cooked rations were weighed, the average amount of cooked meat supplied daily was 7 oz. 1 drachm, exclusive of bone and dripping. When frozen meat is issued they recommend that 10 per cent. increase should be allowed, as there is a greater loss of nutriment when cooked.

The committee also recommended that all ration bread should be baked in 2 lb. loaves, and patent yeast used in place of brewer's yeast: the smaller loaf ensures a proper proportion between the crust and crumb.

British Soldier in India

The constitution and nutritive value of the Indian ration are as follows:—

—	Quantity taken daily	Water- free	Proteids	Fats	Carbo- hydrates	Salts
Meat with bone	Ounces 16.0	Ounces 8.2	Ounces 1.92	Ounces 1.08	Ounces —	Ounces 0.2
Bread	16.0	9.6	1.29	0.24	7.84	0.2
Potatoes	16.0	3.86	0.20	0.02	3.86	0.16
Rice	4.0	3.6	0.32	0.032	3.88	0.082
Sugar	2.5	2.42	—	—	2.41	0.012
Tea	0.71	0.7	—	—	—	—
Salt	0.66	0.66	—	—	—	0.66
Total	55.87	24.04	3.73	1.372	16.99	1.264

In this diet there is—

Nitrogen	256.5 grains
Carbon	4,503 „

The bread and meat are a 'free' issue; the remainder are supplied by the commissariat for a stoppage of 9 pies (about 1*d.*) per diem. In India, as at home, the soldier makes individual purchases of tinned meats, fish, eggs, &c., but the whole amount does not appear to be in excess of a standard diet, necessary to maintain health. Vegetables are very difficult to obtain during the hot weather, and generally there is a deficiency in these.

On active service in the field a special scale is fixed by the Secretary of State, according to the climate and circumstances of the expedition. The following scale is adopted as far as possible:—1 lb. fresh, salt, or preserved meat; 1½ lb. bread, or 1 lb. biscuit, or 1 lb. flour; ½ oz. tea; ½ oz. coffee; 2 oz. sugar; ½ oz. salt; ¼ oz. pepper; ½ lb. fresh vegetables when procurable or 1 oz. compressed vegetables; also ½ oz. of lime juice with ½ oz. sugar, and 2½ oz. rum, when ordered by the general commanding, on the recommendation of the principal medical officer.

In the Ashanti Expedition (1879) the following was the ration issued: *Bread*—1½ lb., or 1½ lb. biscuit, or 1 lb. flour. *Meat*—1½ lb. fresh beef or 1½ lb. salt pork, or 1½ lb. salt beef, or 1 lb. preserved mutton, or ¾ lb. sausage in lieu of half the ration of fresh or salt meat might be substituted in special cases. *Vegetables*—2 oz. rice, or 2 oz. peas, or 4 oz. preserved vegetables, or 4 oz. preserved potatoes, or 1 lb. fresh potatoes. *Groceries*—¾ oz. tea, 8 oz. sugar, ½ oz. salt, ¼ oz. pepper. *Tobacco*—1 lb. per man per month. *Cheese*—4 oz. might be substituted for one-third ration of meat. The ration in Afghanistan was: Fresh meat, exclusive of bone, 1 lb.; bread, 1½ lb., or biscuit 1 lb.; rice or flour, 4 oz.; sugar, 8 oz.; tea, ¾ oz.; salt, ¾ oz.; potatoes, 10 oz.; green vegetables, when procurable, 6 oz.; and if green vegetables were not procurable, then dhall 1 oz. and potatoes 12 oz. were given. One dram of rum per man was also authorised, one half at dinner and the other half in the evening after sunset; ½ to ¾ lb. of tobacco per man per month was also issued.

During General Roberts's march to Candahar the meat ration was increased to 1½ lb.; a tin of Kopf's Erbswurst was also given to each man daily.

In the Suakim Expedition in 1885 the ration for the troops was as follows: Bread, 1½ lb., or biscuit, 1 lb.; fresh meat, 1½ lb., or preserved meat, 1 lb.; tea, ½ oz.; coffee, ½ oz.; sugar, 2½ oz.; salt, ½ oz.; pepper, ¼ oz.; potatoes or fresh vegetables, 12 oz.; compressed vegetables, 1 oz.; occasionally jam or marmalade; Erbswurst, 2 oz.; lime juice, ½ oz.; sugar, ½, and rum ¼ oz. together when ordered. Lime juice was ordered to be invariably given when potatoes or fresh vegetables were not supplied.

On service, bread should be issued as long as possible; if biscuit is issued for more than a week, flour or rice should be added. Salt meat should be seldom required with the present excellent supply of preserved meat in tins. If no vegetables can be obtained, lime juice should be given.

In war the difficulty of cooking often causes the men to be without food for a long time: this tends to exhaust the strength of the soldier. All cooking utensils should be of block tin, and are most easily carried when made round in shape. Nothing is better than Warren's cooking pots: by these, soup, meat, and vegetables can be cooked at the same time; and if there is not time to cook the meal before starting on a march, these vessels can be carried with the baggage, and at the end of the march the food will usually be found ready.

For rapid expeditions, when Transport has to be reduced to a minimum,

the issue of concentrated and cooked foods may be necessary. Men should be able to carry enough for one week and be independent of supply from the base. Dr. Parkes gives the following quantities as sufficient to last a man for seven days:—Biscuit, 2 lbs.; pea or flour meat sausages, 4 lbs.; dried meat, 2 lbs.; sugar, $\frac{3}{4}$ lb.; tea, $\frac{1}{4}$ lb.; cheese, 1 lb.; total, 10 lbs.: this would be lessening day by day, and if carefully used would be sufficient to carry men through any work they might be called on to do during the time. No special emergency ration has yet been fixed for the Army.

In the Franco-German War the Germans used pea sausage (Erbswurst) made by mixing pea-flour and fat pork with a little salt. It was issued ready cooked, but could be made into soup; this Erbswurst contained about 16 per cent. of proteids, 85 per cent. of fat, and 27 per cent. of starch, &c. Knorr's sausage, which is now used, contains 17.5 per cent. of proteids, of which only 2.6 per cent. are indigestible, and about 86 per cent. of fat.

When fresh vegetables cannot be obtained, preserved vegetables may be substituted for them: one pound of uncooked preserved potatoes are equal to 3½ lbs. of fresh potatoes. Preserved vegetables are in every way superior to compressed vegetables. Owing to the high pressure to which the latter have been subjected, a very large proportion of their salts, with part of their albumin, has been expressed, and little remains beyond cellulose (Morache); on this account their antiscorbutic power is not very great. Preserved vegetables require to be well soaked in water before being used, otherwise they are apt to cause diarrhoea.

The issue of a spirit ration on service has been the subject of much discussion; the whole experience in recent wars is against its issue. There is perhaps no point on which there is a more unanimous opinion as this one, that there should be no daily issue of a spirit ration. Dr. Bryden long since pointed out that there was nothing more inimical to the acclimatising process in India than the habitual use of alcohol. But although the daily issue of rum as a ration should be avoided, there are cases in which a ration of alcohol has been found to be productive of the greatest service, even where alcohol in the form of rum and beer may be productive of much evil. The advantage which light red wines possess cannot be passed over. These, well diluted, are most refreshing drinks in hot climates: they should of course be used in moderation, and, for young and 'unseasoned' soldiers, probably total abstinence would be better. After a fatiguing march, red wine may be given with advantage: it has a recuperating effect, and may possibly be a preservative against disease. Alcohol should never be allowed before or during a march, but at the end, and then only in the form already indicated. It was formerly supposed to be a preservative against malaria; that this is not so is now abundantly proved by the experiences gained in India and South Africa. There is no evidence to show that the issue of a daily ration of rum has been productive of any good, and in many cases it has certainly done much harm. It is certainly contraindicated in all cases where cholera and enteric fever are likely to occur. On the other hand, light red wine may be given with advantage, as it contains a large amount of salts and tannin, the latter possibly precipitating and rendering innocuous any organic matter in the water.

The supply of pure water to an army in the field is of the first importance: its quality will depend no doubt on the nature of the soil on which men are encamped; as a rule, healthy geological soils afford good water. The quantities necessary for the use of men and animals will be considered under the head of WATER.

Rations of the French Soldier

The Government furnishes the meat for the soldier's rations at about 85 per cent. under market price ; also his free ration of bread (*pain de munition*) and fuel ; other articles such as white bread (*pain de soupe*) are bought from the funds of his regiment.

The following is the authorised scale of diet :—

	Grammes	Ounces avoird.
Munition bread	750	26·4
White bread for soup	250	8·8
Meat (uncooked)	300	10·6
Vegetables (green)	100	3·5
" (dried)	30	1·1
Salt	15	0·5
Pepper	2	{ 00·73
		{ = 31 grains
Total	1,447	51·00

Deducting 20 per cent. from the meat for bone, the nutritive value of this diet is—Proteids, 4·83 oz. ; fats, 1·25 oz. ; carbohydrates, 18·04 oz. ; salts, 1·12 oz. Total water-free food, 24·74 oz.

If biscuit is issued in place of bread, 19·4 oz. are allowed ; if salt beef is used, 8·8 oz. are issued, or salt pork, 7 oz.

The normal daily ration in time of war is as follows :¹—

Bread	37·27 ounces	} Ration of bread
or Biscuit bread	33·51 "	
or Biscuit	25·92 "	
Meat	14·11 "	} Ration of meat
or Preserved meat	8·82 "	
or Salt pork	8·82 "	
Dried vegetables or rice	3·53 "	
Lard	1·06 "	
Salt	0·55 "	
Sugar	0·74 "	
Coffee	0·55 "	

The nutritive value of this diet is as follows :—Proteids, 5·26 oz. ; fats, 2·86 oz. ; carbohydrates, 19·09 oz. ; salts, 1·86 oz.

Of these, the dried vegetables, rice, salt, sugar, and coffee, form what is known as the ration of *petits vivres*, to which troops are entitled from the day they leave their places of mobilisation for the front.

The ration of brandy is 0·11 of a pint. It is only issued exceptionally by order of the commander-in-chief.

It is also believed that the daily ration includes 'Boissonnet' sausage and preserved beef *au chauffin*.

The Minister of War is authorised to make such additions and alterations in this scale to suit the place and circumstances where the war may be carried on.

Rations of the German Soldier

The rations in time of peace are divided into the smaller ration for ordinary use in garrison and the larger ration for use in camps and in field manoeuvres, &c.

¹ *Handbook of the French Army*, 1891.

	Smaller ration, in ounces avoird.	Larger ration, as supplied for camps, marches, &c., in ounces avoird.
Bread	26·47	85·80
Meat (raw)	5·80	17·65
or Bacon	4·41	6·00
or Smoked meat (only in war time)	—	8·82
Rice	8·18	6·00
or Groats or grit	4·24	6·00
or Peas or beans	8·12	12·00
or Potatoes	53·00	71·00
Salt	—	0·88
Roasted coffee (exceptionally only in war time)	—	1·41
Brandy	—	8·58
or Beer	—	85·80
or Wine	—	17·65
Butter	—	1·76
Tobacco	—	1·41

The nutritive value of these diets is as follows :—

Kind of ration	Proteids	Fat	Carbo- hydrates	Salts
Smaller ration	3·79	0·77	17·27	0·47
Larger ration	4·76	0·95	18·81	0·50

Troops, when travelling by railway or steamer, receive an additional pay of 25 pfennings (=8d.) per man for refreshments. Should the travelling last longer than sixteen hours, the additional pay is doubled.

The daily war ration is now as follows :¹—

Bread	26·50 ounces
Fresh or raw salt meat	18·25 "
or Smoked beef, mutton, ham, bacon, or sausage	7·00 "
Rice or ground barley	4·50 "
or Peas, beans, or flour	9·00 "
or Potatoes	53·00 "
Salt	0·90 "
Coffee roasted	0·90 "
or Raw coffee	1·00 "

This affords from 4·87 to 5·44 oz. proteids, from 0·71 to 8·71 fat, and from 19·61 to 22·41 of carbohydrates.

Rations of the Austrian Soldier

The peace ration consists of the following :²—

Bread	80·89 ounces
or Biscuit	17·65 "
Meat	6·71 "
Suet	·62 "
Wheat flour	6·57 "
or Legumes	2·47 "
or Groats	4·94 "
or Millet	5·29 "
or Pearl barley	4·02 "
or Potatoes	19·77 "
or Rice	3·71 "
Sauerkraut	5·54 "

¹ Dr. W. Roth, *Jahresbericht über die Leistungen und Fortschritte*, 1888.

² Frölich, *Militärmedizin*, 1887.

This contains proteids, 4·84 oz.; fat, 1·74 oz.; carbohydrates, 17·33 oz., and 0·58 oz. of salts.

The ordinary field food ration consists of¹—

Bread	1 portion	30·8 ounces
Rice or preserved vegetables	"	4·9 "
Preserved soup	"	1·8 "
Fresh beef	"	10·6 "
Salt	"	1·0 "
Pepper or paprika	"	0·017 "
Lard or butter for cooking	"	0·7 "
Coffee	"	0·43 "
Sugar	"	0·46 "
Tobacco	"	1·3 "
Rum or brandy	"	0·1 pint
or Wine	"	0·63 "

The nutritive value of this diet is—Proteids, 4·69 oz.; fats, 2·43 oz.; carbohydrates, 17·67 oz.; salts, 1·80 oz.

In the above diet, tea and rum may be substituted for the coffee.

The reserve food supply carried by each man consists of—

Biscuit	1 portion	17·6 ounces
Preserved meat	"	7·0 "
Preserved soup	"	1·3 "
Salt	2 portions	2·0 "
Tobacco	"	2·4 "

This affords proteids, 4·57 oz.; fats, 1·47 oz.; carbohydrates, 13·57 oz.; salts, 1·64 oz.

Rations of the Russian Soldier²

—	Peace ration, in ounces avoird.	Smaller war ration, in ounces avoird.	Larger war ration in ounces avoird.
Rye-bread	48·35	36·15	36·15
or Biscuit	28·91	—	—
Flour	32·65	—	—
Meat	7·24	14·44	21·67
Groats	4·80	4·80	4·80
Butter or tallow	—	1·34	2·72

The nutritive value of these diets is—

Kind of Ration	Proteids	Fat	Carbohydrates
Peace ration	5·86	0·99	24·74
Smaller war ration	3·67	2·58	18·36
Larger war ration	5·79	4·13	18·36

Rations of the Italian Soldier²

The peace ration of the Italian soldier is as follows:—

	Ounces avoird.
Bread	32·40
Meat	7·06 to 10·59
Bacon	0·53
Rice	5·30
Salt	0·53
Sugar	0·71
Roasted coffee	0·53
Wine	8·32

¹ *Handbook of the Military Forces of Austria-Hungary*, 1891.

² Frölich, *op. cit.*

The nutritive value of this diet is—Proteids, 8·99 oz.; fats, 1·34 oz.; and carbohydrates, 21·64 oz.

Rations of the United States Army

The daily ration of a soldier in the United States Army is as follows:¹—

Fresh beef, or other fresh meat	20·00 ounces
or Salt beef	22·00 "
or Pork or bacon	12·00 "
Flour	18·00 "
or Soft bread	18·00 "
Except on fatigue, when	
Hard bread	16·00 "
or Corn meal	20·00 "
Beans or peas	2·40 "
Rice	1·60 "
Sugar	2·40 "
Coffee, green	1·60 "
or Coffee, roasted	1·28 "
or Tea	0·32 "

and Candles, soap, pepper, &c.

By a recent Act, 1 lb. of vegetables is added to this ration. A general order directs the issue of 100 per cent. fresh potatoes, or 80 per cent. fresh potatoes and 20 per cent. fresh onions, or 70 per cent. fresh potatoes and 80 per cent. canned tomatoes or other vegetables.

Diet for Native Indian Soldiers

In the Abyssinian War the native ration consisted of—Rice or atta (flour), 2 lbs.; dhall, 4 oz.; ghee, 2 oz.; salt, $\frac{3}{8}$ oz.; turmeric, $\frac{1}{8}$ oz.; sugar, 3 oz.; and 1 lb. of mutton or goat's flesh (when procurable) twice a week.

In the Afghan War the following rations were allowed:—Atta or rice, 2 lbs.; ghee, 2 oz.; dhall, 4 oz.; salt, $\frac{3}{8}$ oz. Meat was also issued to native troops on payment; also pepper, opium, spices, bhang, &c.

In Egypt, in 1882, the ration for Indian soldiers was $1\frac{1}{2}$ lb. of rice or 2 lbs. of atta, 4 oz. of dhall, 2 oz. of ghee, $\frac{3}{8}$ oz. of salt, 1 oz. of onions, and $\frac{1}{8}$ oz. of pepper. Once a week 1 lb. of meat was issued in lieu of half the rice ration if not antagonistic to caste prejudices.

WATER SUPPLY IN BARRACKS AND CAMPS

It is introduced here only to refer to those points connected with military service: the question of water supply to barracks is so analogous to the requirements of civil life that the same conditions apply. By decision of the Secretary of State for War, a soldier at home receives 15 gallons daily, no extra allowance being made for the soldiers' wives and children in a regiment; the amount required on foreign service varies with the climate and the nature of the work the men are called upon to do.

In stationary camps the quantity required per man for drinking and cooking purposes may be put down at 6 pints in temperate climates. For purposes of ablution the same amount is absolutely necessary. In stationary camps, however, the minimum daily allowance should be 5 gallons for all purposes, washing included; in desert marches as little as half a gallon has been the quantity allowed on special occasions; but, as a rule, from 2 to 3 gallons should be provided, the amount depending on the climate and season of the year.

¹ *Military Hygiene*. By Major A. A. Woodhull. New York, 1890.

For animals the following quantities are required :—

Horse, 6 to 8 gallons (as a rule a horse ordinarily drinks $1\frac{1}{2}$ gallon at a time) ; when not in hard work, 6 gallons is sufficient, but with hard work in the tropics they require from 8 to 12 gallons ; mules and ponies, 6 gallons ; bullocks, 6 to 8 gallons ; camels, 15 gallons ; elephants, 40 to 50 gallons ; sheep or pigs, $\frac{1}{2}$ gallon to 1 gallon. The quantity fixed by the War Office for cavalry horses on home service is 8 gallons daily, and for artillery horses 10 gallons, this latter to include the washing of horses and carriages.

On ships intended for long sea voyages, a distilling apparatus is provided capable of giving $1\frac{1}{2}$ gallon of pure distilled water to each person on board and 10 gallons for each horse or mule. In the Abyssinian Expedition the following was the calculation for the daily expenditure of water per head on shipboard : Elephants, 25 gallons ; camels, 10 gallons ; oxen (large draught), 6 gallons ; oxen (small draught), 5 gallons ; horses, 6 gallons ; mules and ponies, 5 gallons.

In camps it is necessary to guard the site from which water is drawn from any possible sources of pollution : if the supply be taken from a river, that for drinking purposes should be taken from the centre of the stream and at the highest part ; for animals a place should be selected below this ; and for washing clothes, utensils, &c., the lowest part should be used. Water is best given to animals by receiving it first in troughs, as by doing so there is less disturbance of the stream. If the water-supply is derived from wells close to villages, the immediate surroundings of the well should be carefully examined. Stagnant pools should not, if possible, be used as a source of supply, nor sluggish streams passing through dense jungle ; if such water must be used, it should first be boiled and then filtered.

In camps, sentries should be invariably posted over streams and wells from which water for drinking purposes is drawn to prevent irregularities.

When the base of operations is on the sea coast, all water should be distilled if an ample and pure supply is not available. This was done during the Egyptian Campaign on a large scale, and with the best results. Inland, distillation cannot always be resorted to, and shallow or deep wells have to be bored. For this purpose the Norton tube-well is the one most readily available. The well is made by driving a number of tubes into the ground with a 'monkey,' and fixing a pump, by means of a screw, on the top. The yield is about 7 gallons per minute from a depth of about 18 feet. This pump is not suited for sandy soils, as the tube gets easily blocked.

If the Norton tube-well does not yield a sufficient supply, a Bastein pump, having an endless chain and worked by two men at a wheel, is the one generally used : it yields, from a depth of 45 ft., about 2,250 gallons per hour.

The purification of water is important on service, when almost any supply has to be made available at times. In many instances no filters are at hand, and means have to be taken to make the water usable. If it is turbid from finely divided silt, this may be got rid of by the addition of alum in the proportion of 6 grains to the gallon of water. This plan acts well, if there is any calcium carbonate present in the water ; if this is absent, it is advisable to add a little sodic carbonate first to the water before treating it with alum. The water is then allowed to rest, and in a short time will be found to have deposited the greater part of the suspended matters : it may then be filtered and boiled.

In the Ashanti campaign of 1878 the following plan was adopted by Dr. Gouldsbury in the absence of proper filters. Alum was added to precipitate organic matter, the water was then passed through a rough filter, consisting of (1) sponge, (2) sand, (8) charcoal in pieces : it was then boiled, and a few

drops of solution of potassium permanganate added. Water even taken from a hole in a marsh was innocuous after this treatment.

In the Egyptian campaign, wells were dug in close proximity to the fresh-water canal, so as to allow filtration through the soil. The water percolating through was collected and alum added; it was subsequently filtered and boiled.

The simplest form of field-service filter consists of two casks, one placed inside the other, having a space 6 in. all round between them: the outer cask has holes pierced at the bottom, and the inner near the top; the water rises through the filtering medium between the barrels and flows into the inner barrel. It is very advisable to heat the sand or gravel to redness before use, if the space between the barrels is filled with these materials. The filtering media must in any case be changed frequently and purified by washing and heating to redness.

For stationary camps and hospitals on the lines of communication, as well as at the base, the Morris circulating filter appears to be one of the best adapted for military purposes. It is of very simple construction, and can be readily understood from the accompanying figure (fig. 81). The essential feature of these filters is the employment of a fine-grained and of a coarse-grained powder of carbon or other filtering substance. The water having passed through, the fine-grained portion is conducted into a compartment filled with larger grains; its flow is therefore hastened, and by means of a short tube, opening directly from this into the external air, the carbon is kept aerated and more or less dry. There are no joints or fittings to this filter, and it cannot get out of order; it is very strong and capable of resisting the rough usage incidental to field service. The medium employed is Doulton's *manganous carbon*, which is a mixture of animal charcoal and black oxide of manganese.

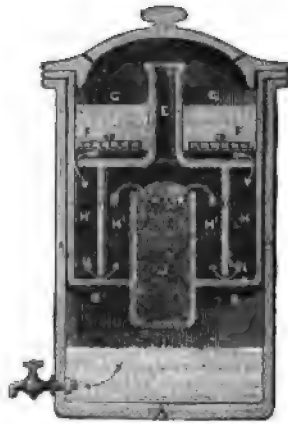


FIG. 81.

In the French Army the Chamberland filter is the one adopted: this consists of cylinders of porcelain through which the water is forced. It renders water clear and bright, and free from suspended matters; it is also said to remove micro-organisms and their spores, rendering the water sterile. The disadvantage of this filter is that pressure is required to force the water through the tubes, and the delivery is slow.

On service, pocket filters are frequently used, but their small size renders their oxidising powers of very short duration: they are useful to remove suspended matters, but have little effect on dissolved organic impurities.

Soldiers should be cautioned not to drink turbid water, and the reason for not doing so should be explained to them. As each man carries a water-bottle (made of enamelled iron and covered with felt) he should be taught to fill it with pure water whenever practicable. If water fit for drinking is not obtainable, the bottle may be filled with cold tea.

In India, during the occurrence of epidemic disease, the *bheesties* (water-carriers) should be paraded every morning, and their *mussacks*, or skins, carefully inspected; under ordinary circumstances this should be done at frequent intervals. The sources from which they obtain water should always be carefully inquired into.

As boiling appears effectually to destroy any organic poison in water, it is

of the greatest importance that all suspicious water should be boiled before use. During epidemics, such as cholera or enteric fever, this is essential, and in such cases it may be necessary to change the source of supply, and where there is any doubt as to the quality of the water this should be invariably done.

MILITARY CLOTHING

In former times a sum of money was granted to colonels of regiments to cover the cost of clothing: this system was not found to answer, and since the Crimean War the Government has taken the supply into its own hands.

The various articles of clothing for the Army are prepared, under Government supervision, at the Clothing Depot at Pimlico, and every care is taken to test the materials tendered by contractors. All clothing for soldiers is now issued in accordance with the *Regulations for the Supply of Clothing and Necessaries*.

The recruit on enlistment receives a free kit. Some of the articles the Government replaces as they become unserviceable; others he is obliged to make good at his own expense, but these are sold at cost price, and a careful man can keep his kit in good order at an annual cost of about 1*l*. The following are the articles of kit supplied to an Infantry recruit:—

Clothing.

2 frocks.	2 pairs of ankle boots (one every six months).
2 pairs of trousers.	1 forage cap.
1 pair of mitts.	

Necessaries.

2 flannel shirts.	1 razor and case.
3 pairs of socks (worsted).	1 hold-all.
1 pair of braces.	1 tin of blacking.
1 hair-comb.	1 blacking-brush.
1 knife and fork.	1 brass-brush.
1 spoon.	1 cloth-brush.
1 mess-tin and cover.	1 polishing-brush.
2 towels.	1 shaving-brush.
1 soap (piece).	1 button-brass.
1 sponge (pipe-clay).	1 kit-bag.

This kit is divided into 'surplus' kit and 'service' kit. The surplus kit is always carried for the men: it consists of one frock, one pair of socks, one shirt, one towel, two brushes, and such articles in the hold-all as he is not likely to require; the service kit is taken on his person or in his knapsack.

Certain articles of clothing are also issued, free of expense, at stated intervals; they vary with the particular arm of the service, and are given in detail in the *Regulations*. The following are those issued to an Infantry soldier on home service:—

1 helmet and bag	Quadrennially
1 tunic	Biennially
1 frock (undress)	Annually
1 pair tweed trousers	Annually
2 pairs of boots (on April 1 and October 1)	Annually
1 forage cap	Annually
1 silk sash for warrant-officers and staff-sergeants	Biennially
1 worsted sash for sergeants	Biennially
1 great coat	Quinquennially
1 pair mitts	Triennially

The following articles of clothing are issued to each man proceeding on active service in cold, temperate, or hot climates :—

In Cold Climates.

Sheepskin coats (for 100 men)	8	Canvas shoes for dismounted, and putties	
Fur caps (per man)	1	for mounted men	1
Woollen comforters	1	Drawers, flannel, per man, pairs	2
Jerseys, blue	1	Belts, flannel	2
Boots, knee, brown leather, pair	1	Mitts, fur-lined	1
Stockings, woollen	2	Pilot coats, each mounted man	1
		Laces for ankle boots, pair	1

In Temperate Climates.

Flannel belts (when not included in the		Waterproof capes (for 100 men)	10
voyage kit)	2	Watch-coats (for 100 men)	8
Canvas shoes for dismounted, and putties			
for mounted men, pair	1		

In Tropical Climates.

Frock-coats of serge or tartan (when not supplied as ordinary clothing	
of these climates)	1
Flannel belts (when not part of the sea kit)	2
Coats, waterproof (for 100 men)	10
<i>For India.</i> —A drill frock and drill trousers are supplied at the public expense.	

In selecting the materials for soldiers' clothing, the chief points to be considered are its permeability, durability, and the property it has of conducting and absorbing heat.

Cotton is durable, does not shrink when washed, is non-absorbent of moisture, conducts heat rapidly away, and has the effect of chilling the body if perspiration is present. Hence it is not the material for the dress or under-garments of soldiers. It is also non-permeable, possessing little more than half the permeability of flannel.

Linen, like cotton, is a good conductor of heat and a bad absorbent of moisture: it soaks up the moisture from the skin, and this evaporating so cools the body as to cause chills. In many respects, however, it is inferior to cotton for underclothing.

'Cellular cotton' has lately been introduced. In the process of manufacture air interspaces are left in the texture. Air being a bad conductor of heat, the cellular cotton is warmer than cotton clothing.

Wool is by far the best material for underclothing: it has very large absorbing properties for moisture, conducts heat very slowly, and thus prevents cooling of the surface of the body after exercise; it is also nearly double as permeable to air as cotton: for these reasons woollen garments are best adapted for ensuring an equable temperature round the body, and should invariably be used in the military service. In all campaigns it has been found the best material, and the best preservative against illness. The non-conducting properties of wool may be, in part, due to the fibres, which contain a proportion of fatty matter, as well as to the large amount of air entangled in the interspaces.

Dr. Jaeger's woollen underclothing is so woven that it is not irritating to the skin, and the arrangement of the constituent hairs provides for the escape of moisture. It is largely used in the German Army.

The disadvantages of woollen clothing are, that the material becomes hard and shrinks on washing, and thus loses, in part, its absorbing pro-

perties. This can, in every case, be obviated by using a soap free from any excess of alkali. The alkali acts on the natural oil of the wool and materially injures it. The addition of a little paraffin to the soap is said to facilitate the removal of dirt.

Soldiers' shirts, made at Pimlico, are manufactured from a mixture of cotton and wool: this material is lighter and cheaper than pure wool, and is said to be more durable; it does not shrink in washing: there should not be more than 80 per cent. of cotton in the mixture.

The colour of the material has an important bearing on the hygienic value of the clothing, and in regard to the absorption of heat exerts more influence than the material itself. The results of experiments made at Aldershot show that white possesses very slight absorptive power compared to other colours, and, next to this in the scale, grey or pale yellow gives the best results: grey is the best colour for soldiers' dress on service, white is least suited for the field, as it soils so quickly; the khaki drill used in India appears to answer well, and, as regards absorption power, corresponds very closely with grey.

All clothing should be made to fit loosely, so as to allow free movement of every part of the body, otherwise mechanical work is increased. In the British Army the underclothing consists of shirts, stockings, and flannel belts. The shirts are made of a mixture of wool and cotton. In hot climates all wool would probably be found a better material; but the collar band should be made of linen to avoid shrinkage, and consequently tightness round the neck. Drawers find no place in the soldier's kit; they are cleanly, and it is as necessary to provide for the warmth of the legs and lower part of the abdomen as for any other part of the body. Many soldiers provide themselves with drawers.

Socks are made of worsted. They are not sufficiently supplied, and as they often cause excess of perspiration they should be frequently washed. It is probable that sore feet are frequently due to this cause. A good sock, kept clean, is a protective against sore feet. Flannel belts are useful to protect the abdomen from chill. A watch must, however, be kept to see that men wear them, especially in hot climates. It is astonishing to find the number of men who neglect to do so.

The tunic—the coat worn by the British soldier—is closely fitting, and to some extent compresses the muscles, and interferes with the free movement of the chest. If loosely made, it does not give the same appearance of smartness to the men. For active service the tunic is made looser, and generally of some thin material (this will depend on the climate); but serge is always preferable to cotton. A loose Norfolk jacket is the pattern usually adopted in India, and this seems to meet all necessary requirements.

Trousers should be large over the lower part of the pelvis. Dr. Parkes recommended the 'peg-top'-shaped trousers as being the best pattern, and if 'putties' are worn with these, they make a most serviceable dress. The putties give support to the leg, and protect it from the bites of insects.

Braces are preferable to a belt round the waist. They form a better means of support, and do not compress any part, which a belt invariably must do. This latter is also said to predispose to hernia.

The great-coat and cape is issued in three sizes, and weighs from 5 lbs. 8 oz. to 6 lbs. 8 oz. They are all made double-breasted, and as long as possible. The cloth is excellent, but it is rather heavy, absorbs a large quantity of moisture, and is difficult to dry. It would be an advantage if the great-coat could be made of a lighter material, and waterproof.

For service in the field a waterproof sheet is an imperative necessity, to

protect against rain or ground moisture. The waterproof sheet should always be used to lie on unless employed to form a temporary *tente d'abri*.

Such articles as sheepskin coats, hoods, gloves, &c., issued for protection against very severe cold, are necessary, and are fully justified by the results following their use.

The head-dress is an important article of the soldier's kit. The essentials of a good head-dress are that it should be light, durable, and comfortable; that it does not press unduly on any part, nor fit too closely on the head. It should admit of a limited amount of ventilation, and its shape should not only serve as a protection to the head, but it should afford as little resistance as possible to the wind.

Helmets are now issued to infantry regiments, artillery, and engineers, and also to departmental corps.

Bear-skin caps are worn by the Guards, Highland bonnets and shakoes by Highland regiments, and a seal-skin cap by Fusilier regiments.

The infantry helmet weighs $14\frac{1}{2}$ oz. It is made of cork, covered with cloth. The weight of the bearskin is 37 oz.

In the cavalry and horse artillery, helmets are also worn, but of a slightly different pattern. They are of excellent shape, but rather heavy. In the Guards and Heavy Dragoons the helmet is of metal, and is partly intended for defence. The weight of the Life Guards helmet is 55 oz., and of the Dragoon Guards 89 oz. The Lancer cap weighs $29\frac{1}{2}$ oz., the Hussar $28\frac{1}{2}$ oz.

In India the same head-dress is worn by all the different branches of the service. Helmets made of bamboo or cork, covered with cotton and provided with puggeries, are now used: they are very light—13 oz.—and afford good protection from the sun.

The 'Tusan helmet' has lately been adopted by some regiments in India. It consists of two bodies, one within the other, forming a complete air-chamber, not only in the crown but in the brim, thereby completely protecting the temples and the nape of the neck. The weight with chin strap and button is only $11\frac{1}{2}$ oz.; with chin strap and spike, 14 oz.

In the French Army a shako, made of pasteboard and covered with leather, is worn. It is very hot.

Leggings are now used by all dismounted branches of the service: they are made of leather and reach from the ankle nearly to the knee. The great advantage of leggings is that at the end of a march they can be at once taken off and cleaned. In India 'putties' are used for the same purpose, and are found most comfortable, affording protection and support. They are worn by the mounted branches also.

The boots are supplied from the Clothing Dépôt at Pimlico, and are in thirty-two sizes, made right and left, and weigh about 40 oz. The sole is wide and the heel low and broad. The leather has to be of a certain quality, and a number are always cut up and examined before a contract is passed. There must be eight stitches per inch for the upper leather, and the thread must be of a certain strength and well waxed. The great fault of this boot is its hardness and the rough way in which it is finished. Once it is moulded by wear to the shape of the foot, it is an excellent boot. In the Russian Army boots made of a soft and pliable leather are issued, and foot-soreness is said to be unknown: the Russians appear fully to understand the advantages gained by this.

The English boot seems to require some other means of fastening it. The number of eyelet-holes cause waste of time in fastening, and the thong is liable to break. The 'Elcho' boot is worn by officers on service: it is a long boot with a slit down the centre; it may be worn under the trousers or

outside, as the slit opens and can be laced. There is no difficulty in taking off this boot when wet, as was so frequently the case with the old pattern.

For service in the field, boots should, if possible, be made impermeable to wet. Dr. Parkes recommended the following receipt which he tried and found effectual.

Take half a pound of shoemaker's dubbing, half a pint of linseed oil, half a pint of solution of india-rubber. Dissolve with gentle heat (the mixture is very inflammable), and rub on the boots. This will last for five or six months; but it is well to renew it every three months.

Army orders direct (1) that soldiers' boots are to be blackened with three coats of ordinary blacking instead of other substances. (2) Boots or shoes in store are to be dubbed, or have neat's-foot oil applied to uppers at least once in four months.

To prevent foot-soreness among men unaccustomed to marching, the feet should be well soaped or greased before putting on the boots, and at the end of the march they should be washed and thoroughly dried.

Astringent lotions, such as alum dissolved in tepid water, have been found useful.

Blisters are best treated by carefully drawing a woollen thread through the lowest point and allowing the fluid to drain, taking care not to tear away the skin.

In the German Army a powder composed of salicylic acid 8 parts, finely powdered starch 10 parts, and pulverised soap 87 parts, is used, it is said, with great advantage. The feet and the inside of the boots are dusted with it. It keeps the feet dry and prevents chafing.

WEIGHTS OF THE SOLDIER'S DRESS AND ACCOUTREMENTS, AND HOW CARRIED

In the mounted branches of the service the weight of the accoutrements and equipment is in great part carried by the horse. When not worn the great-coat is carried in a roll over the shoulder, or more generally in front, on the horse.

The following table gives the approximate weights carried by a horse on active service in the field:—

LIGHT CAVALRY

Table of Approximate Weights carried by a Horse in the Field.

	St.	lbs.	oz.
The rider (say)	10	4	0
Clothes on rider—viz. flannel shirt, drawers, socks, braces, head-dress, tunic, pantaloons, boots, spurs, gloves, and flannel belt	1	1	11
Arms, &c., belts and sword	0	7	0
Carbine	0	7	7
Ammunition, 80 rounds	0	4	8
Saddlery—viz. saddle and bridle complete, breastplate, wallets, shoe-cases, numnah, head-ropes, and carbine bucket	2	9	11
Small blanket under saddle	0	2	0
Kit of rider—viz. clothes-brush, stable-sponge, oil-bottle, pot of grease, horse-rubber, pocket-ledger, field-dressing, horse-brush, curry-comb, flannel shirt, drawers, socks, towel and piece of soap, hold-all with needles and thread, kit scissors, fork, spoon, comb, and forage-cap	0	7	5
One day's reserve rations (sausage), say	0	0	12
Cloak and waterproof sheet	0	9	8
Carried forward	16	11	14

¹ Lord Wolseley, *Soldier's Pocket-book*, p. 18.

	St. lbs. oz.
Brought forward	16 11 14
Mess-tin and strap	0 1 4
Haversack, water-bottle, and pocket-knife	0 1 12
Hoof-picker, nose-bag, picket-peg, heel-rope, and shackle	0 4 1
Hay-net, corn-sack	0 8 12
A fore and hind shoe with nails	0 1 14½
Balance of man's rations, &c. (say)	0 1 0
Balance of horse's forage, &c. (say)	0 6 0
Mallet (when carried)	0 2 0
Total	18 5 9½

The Infantry soldier's kit is divided into the service and the surplus kit, the latter being always carried for the man. The greater part of the service kit is made up of the clothes he wears, with a few duplicate articles and other necessities.

INFANTRY

The following are the weights of the 1888 valise equipment in different 'Marching Orders':—

1. Service Marching Order.

	Lbs. oz.
1 Valise	1 8½
1 Pair of braces and chapes	0 13½
1 Waist-belt	0 12½
2 Pouches	1 3
1 Haversack	0 9½
1 Mess-tin, cover, and strap	1 11
1 Water-bottle and carrier	1 0
2 Coat straps	0 5
1 Frog for side-arm	0 3½
1 Great-coat	4 6
1 Cape	2 0
1 Rifle and sling (magazine)	9 8
1 Bayonet and scabbard	1 1
1 Magazine pouch	0 4
1 Spade	2 9½
Total	27 15½

2. Home Marching Order.

2 Pouches	1 8
Haversack	0 9½
Water-bottle and carrier	1 0
Great-coat and cape, with slings	6 11
Mess-tin, cover, and strap	1 11
Valise	1 8½
Pair of braces and chapes	0 13½
Rifle and sling	9 8
Bayonet, scabbard and frog	1 4½
Waist-belt	0 12½
Total	25 1½

3. Light Service Order.

Same as service marching order, but without valise, braces, and chapes	27 15½
	2 6
	25 9½
Add waterproof sheet	4 4
Total	29 13½

Additional Weight carried on Service.

	Libs.	ozs.
Field kit	5	6
Rations	2	0
Water (in bottle)	1	8
Ammunition (magazine)	6	4
Waterproof sheet	4	4
Total	19	6

In the Cavalry the weight carried appears to be too great: the weight of his clothing and equipment is equal nearly to the man himself. It is a mistake to overweight the horse: the long and rapid marches Cavalry have to make tell especially on the horse when he is overpowered by such a weight as 19 stone, and it is of no less importance to keep the horse effective as well as his rider.

In the case of the Infantry soldier, who carries the weights himself, the greatest care is necessary in their arrangement so as not to detract from his efficiency or to injure his health. Whenever possible, his kit, as far as he can dispense with it, should be carried for him, with the exception of his armament and water-bottle. The advantage of transporting a soldier's knapsack for him is well recognised in India, where longer marches, greater endurance, and efficiency are found to result from this practice.

The chief points to attend to are to so adjust the weights that when carried they fall as near the centre of gravity as possible, and not outside; there must be no compression of the chest, so as to interfere with perfect freedom of respiration, or with the circulation by pressure on the arteries or veins. The weight should be distributed as far as possible, so as to avoid fatiguing one set of muscles.

Soldiers should be taught to carry weights, so as to dispose of them comfortably and then cease to carry them, unless absolutely required to do so.

The present equipment is partly based on the plans recommended by the late Sir T. Troubridge, C.B., but there have been many improvements since it was first introduced. The principle is to use the point of the shoulders and the sacrum on which to distribute the weight. The old framed knapsack has been abandoned, and a valise equipment substituted.

The new valise equipment (pattern 1888) possesses the great advantage that it is light and very simple. In a few seconds the soldier can change from 'marching' to 'service' order by merely detaching the valise from the remainder of the equipment. The pouches are so constructed that whilst the ammunition can be easily got at, liability to loss is provided against. Greater freedom of action is allowed, as the straps under the arms are dispensed with. This valise costs less than the former patterns. It is made of japanned canvas and is carried on the back of the shoulders, and is so arranged that it adapts itself to the width of the soldier's shoulders.

The valise holds the following articles:—Emergency ration; towel and soap; clothes-brush; hold-all complete; housewife, fitted; pocket-ledger; one pair of socks; canvas shoes; and cape carried under the flap.

The ammunition is carried in two pouches; the right hand is the 'expense' pouch containing thirty rounds, the left being the 'reserve' holding forty rounds.

The mess-tin is carried on the top of the great-coat or under the coat, when this is carried on the shoulders, or it may be fastened to the waist-belt; in each case it can be detached without interfering with the rest of the equipment.

The German soldier carries his kit in a cowskin knapsack, which is encircled by the rolled great-coat and to the back of it a camp kettle is strapped.

The ammunition is carried in the two front pouches. The haversack, to which a drinking flask is attached by a clip, is worn at the right side; on the left an entrenching spade is strapped, the bayonet hanging over it. The total weight carried is about 70 lbs.

The helmet is of polished leather with spike and ornaments of brass; it is low, fits tightly on the head, and is rather heavy. His arm is the 'Mannlicher' repeating rifle. The ammunition is made up in magazines holding five cartridges in each: these fit into the pouch. The following weights are carried by the German Infantry soldiers:—

	Lbs.	oz.
Clothing on the person (with gloves), not including helmet	23	8
Armament and equipment, including helmet, water-bottle (full), coffee-mill, and entrenching tools	42	8
Sundries	1	13
Rations	5	12
Total	73	9

Some of the articles are not always carried by the same man, such as the coffee-mill, hatchet, and spade, so the weight may be lessened to 67 lbs.—average weight carried, 67 to 71 lbs. His rifle weighs 10 lbs. and the bayonet 1 lb. 8½ oz.

In the French Army the soldier wears his coat rather long, but has the skirts buttoned back; his trousers are usually turned up at the bottom over shoes and leggings. His kit is carried in a cowskin knapsack, around which a blanket and portions of a shelter tent are strapped, the whole surmounted by a camp kettle. Two pouches carry his ammunition—one in front and the other in rear of the waist belt. A flask and drinking cup hang at the right side and a canvas haversack at the left. His 'képi,' or shako, is made of leather or pasteboard, and to make it as light as possible it is divested of all unnecessary ornaments. His arm is the 'Lebel' repeating rifle, in which nine cartridges are placed, and can be discharged in succession without re-loading. The total weight carried by the French Infantry soldier is nearly 67 lbs.

The Austrian soldier carries his kit in a cowskin knapsack, around which is strapped his overcoat and on the back a camp kettle. The whole can be immediately detached by withdrawing a pin in the right shoulder-strap. Two pouches in front contain the ammunition, and an additional supply is carried in a cartridge-box of the same material as, and immediately under, the knapsack. The drinking-flask is suspended at the left breast. A brown canvas haversack hangs at the left side, and the entrenching spade is strapped to it, over which again hangs the short sword bayonet by a frog from the waist-coat. For parade purposes he is provided with a tunic and shako, but on service in the field he wears a loose blouse, with pockets, and a field cap, with flats, which can be unbuttoned when necessary.

His arm is the same in every respect as the German soldier. The total weight carried on service in the field is about 60 lbs.

The Russian soldier's equipment consists of two large waterproof canvas satchels slung over either shoulder; that on the right side contains clothing and necessities, while the left one is used as the ordinary haversack. The great-coat is rolled and worn round the body; to the middle of it, at the back, a case containing a spare pair of boots is affixed, and below that the man's portion of a shelter-tent. To the coat-ends a copper camp kettle is fastened

by its handle. A water-bottle hangs over the left haversack and an entrenching spade is strapped by its blade to the waist-belt. The ammunition is carried in two pouches in front. His present rifle is being replaced by the French 'Lebel' rifle. In the field his headdress is a forage cap, made of black cloth, with coloured band. Total weight carried is about 74 lbs.

The Italian soldier carries his equipment in a knapsack made of cowskin : round it is the great-coat and a camp kettle strapped at the back. The waist-belt is worn under the tunic, the skirts of which are buttoned back in front to allow the ammunition pouch to protrude. A water-bottle is worn on the right side, the haversack and sword bayonet at the left side. In the field a white cover is worn over the ordinary shako. His arm is the 'Vitali' repeating rifle. The weight carried is about 75 lbs.

WORK OF THE SOLDIER

The work of the soldier depends mainly on the branch of the service to which he belongs, and therefore it cannot be brought under one general description.

The Artillery have the hardest work, which comprises mostly cleaning horses, guns, carriages, and stables ; the Cavalry have very nearly the same amount of work to get through, although their stable duties consist nearly altogether in looking after their horses ; their movements on parade are more rapid and the distances they cover are greater than the Artillery. The Infantry duties are mostly confined to drills, marches, and fatigue work in barracks.

All these duties when not carried too far have a beneficial effect, but when severe and violent work has to be done hurriedly, the soldier is not placed in the same favourable condition to carry out the work as an ordinary mechanic would be : this is due to the movement of his body being limited, owing to his being more or less burdened with the weights of his accoutrements and his clothes.

With a view to assist in the physical development of the soldier, every recruit is ordered to have a three-months' course of gymnastic training. The exercises last for one hour daily, and are in addition to his ordinary drill. This training is superintended by a medical officer, who is responsible that it is done properly, and who has power to discontinue the exercises if there is any evidence of their acting injuriously, the symptoms indicating the necessity for rest being hurried or difficult respiration, and smallness, inequality, and irregularity of the pulse. The Infantry soldier goes through this course every year, but in the Cavalry, fencing and sword exercise are substituted for it.

During the training the men are carefully examined from time to time, and measurements taken to ascertain what effect it has on their physical development. The regulations lay down that each man's weight must be taken at the beginning and end of each course, and oftener if there is any reason to believe that a loss in body-weight is taking place, care being taken that the weight is recorded under the same conditions, as far as possible, each time. Men should be weighed in their trousers only ; the early morning, and before breakfast, is the best time.

All medical officers consider that, as regards chest measurements, the extent of mobility is of more practical value than the actual maximum and minimum measurements of the chest walls, for it is expansion that shows capacity for sudden or sustained effort. As a rule also the growth of muscle follows on gymnastic training. Measurements should be taken when the muscle is relaxed, and when tense, and over the thickest part.

Much of the success due to gymnastic training depends on the instructor ; if he has patience, and the men are not urged to repeated exertion, but

sufficient rest allowed between the exercises and the work constantly varied, there is little danger of any undue strain on the heart or blood-vessels. The ordinary duties of barrack life may be said to consist in drills, fatigue duties, and marches; in the drills the position is more or less strained, and the nature of his dress and equipment adds to the work the soldier is called upon to do.

In all drills and marches the movements are to a certain extent stiff. The position of 'attention' is not a secure one, as the basis of support is small, and muscular action is necessary to maintain the equilibrium. It is not desirable to keep men long in this attitude, and they are told to 'stand at ease.' In this position the heels in place of being together, as at 'attention,' are further apart, and afford a broader basis of support.

In marching the attitude is stiff; the centre of gravity is constantly shifting from one foot to the other in a constrained way; the lateral movement which is made in ordinary walking is limited; it is certainly more fatiguing than ordinary walking. For this reason, whenever practicable, the order is given to 'march at ease,' in which there is more gradual and not nearly so much loss of muscular strength.

In marching, the heel touches the ground first, and then rapidly the rest of the foot. The heels of the boots, therefore, should be broad and flat. It has been noted that low heels exert a favouring influence on the pace at which a man walks, thereby diminishing fatigue, the rhythm of the steps having an important influence on his speed, and low flat heels having an influence on the rhythm.

The pace should not be too slow. It is necessary to combine the least expenditure of muscular force with the greatest number of steps per minute. This has been estimated by Thurn at 100 steps per minute, and he states that at this rate the leg pendulum traverses its full swing. At a less rate the swing is not completed.

To obviate the unnecessary waste of labour and fatigue incidental to marching, the foot should not be raised from the ground higher than is required to clear obstacles, and it should not be advanced beyond the place where it is to be put down.

In the British Army the length and number of steps in marching are as follows:—

Length and Number of Steps in Marching.

Kind of step	Length	No. per minute	Ground traversed per minute	Ground traversed per hour without halt
	Inches		Feet	Miles
Slow time	30	75	187½	2·1
Quick time	30	116	290	3·3
Stepping out	33	110	303½	3·4
Doubling	33	165	453½	5·157
Stepping short	21	—	—	—
Side step	12	—	—	—
Or when				
Forming four deep . .	24	—	—	—
Stepping back	30	—	—	—

Quick time is at the rate of 116 paces per minute, or 3 miles 520 yards in an hour—roughly 3·3 miles per hour. This rate, however, is seldom accomplished by large bodies of men. If the road is a very good one, in a temperate climate during good weather, a division can march 2½ miles an hour. As a rule, not more than two miles are done, and only half this, if

the roads and weather are unfavourable. Cavalry and Horse Artillery march from four to five miles an hour.

Running drill has been introduced during late years. It is limited to 1,000 yards. The pace must not exceed six miles an hour, and the men are to be gradually trained up to this.

In the French Army the length of step, &c., is as follows :—

French Steps in English Measures (Morache, 1886).

—	Length of step in inches	Steps per minute	Ground traversed per minute in feet	Ground traversed per hour in miles
Pas accéléré . . .	29·5	120 to 135	295 to 332	8·35 to 8·80
Pas maximum (gymnastique) } .	31·5	170	446	5·10

The length of the French step is nearly the same as the English. Under the new regulations the ground traversed per hour is the same.

In the German Army the step is 31·2 inches. There are 112 steps per minute, and the ground traversed is 8·86 miles per hour. In the Austrian Army there are from 115 to 130 steps per minute; the length of the step is 29·2 inches, and the ground traversed 8·22 to 8·65 miles per hour. In the Italian Army the step is the same.¹

In the Russian Army the rate is 116 to 120 paces per minute of 28 inches, covering 2½ miles per hour, and 18½ to 16½ miles per day of 7 to 9 hours.²

The soldier in this country carries, when marching in service order, his valise, which contains his kit, two pouches, greatcoat, haversack, water-bottle, mess-tin, rifle and ammunition, and, carrying this weight in addition to the clothes on his person, the steps are probably as long as possible. The steps must of necessity be shorter in proportion to the weight carried. In India the greatcoat and valise are carried for the men.

It may be convenient to state the amount of work done in marching as so many foot-tons. Using the formula³ adopted by Professor Houghton, F.R.S., and assuming a man to weigh 160 lbs. with his clothes, we get the following table :—

Kind of exercise		Work done in tons lifted one foot					
Walking	1 mile	18·86
"	2 "	37·72
"	10 "	188·60
"	20 "	377·20
"	1 "	and carrying 60 lbs.					25·93
"	2 "	"	"	.	.	.	51·86
"	10 "	"	"	.	.	.	259·30
"	20 "	"	"	.	.	.	518·60

It will be seen from the above table that a march of ten miles, with a weight of 60 lbs., which is about the weight the soldier carries, is a moderate

¹ Morache, *Médecine Militaire*, p. 761.

² *Handbook of the Russian Army*, Major Wolfe Murray, p. 32.

³ The formula is $\frac{W + W' \times D}{20 + 2240}$; where W is the weight of the person; W' the weight carried; 20 the coefficient of traction, and 2240 the number of pounds in a ton. The result is the number of tons raised one foot. To get the distance in feet multiply 5,280 by the number of miles walked. Dr. Houghton has shown that walking on a level surface at the rate of three miles an hour is nearly equivalent to raising $\frac{1}{25}$ th part of the weight of the body through the distance walked.

day's work ; a twenty miles' march under similar conditions is a very hard day's work. Dr. Haughton thinks that walking twenty miles a day without a load, and Sunday being a day of rest, is good work. This is equal to 858 foot-tons.

During marches, regular halts are necessary in order to rest the muscles, and to relieve them from continuous tension. Lord Wolseley recommends a halt of five to ten minutes every hour, and when the march extends for ten or twelve miles, to halt for thirty minutes when half-way ; and this appears to be true economy of labour.

Frequent short halts allow the muscles to rest, and there is economy of force with less fatigue by working for a short period with a short period of rest, than by working for a long period with only one period of rest.

In tropical countries the time of marching should be so arranged that, if possible, the sun should not fall on the backs of the men. This can usually be avoided by using the early morning hours for the march.

Night marches should never be resorted to. In the tropics the soldier cannot sleep during the day in tents, and marching at night destroys the only rest available. All experience shows that night marches sap men's strength and fill the hospitals : this time should be utilised for the purpose which Nature intended it for. The early morning hours are the best : men appear to traverse the ground more easily and to feel the fatigue less. The length of a march in time of peace, which is usually done on home service twice a week during the winter months, is 8 or 10 miles. In India the distance is about the same, and never exceeds 14 miles route-marching. A forced march is any distance which has to be got over in a certain time, say 20 or 80 miles, which has to be accomplished in 24 hours.

In the French Army the length of a march is $12\frac{1}{2}$ to 15 miles ; in the German Army about 14 miles, and if the march is continuous there is a halt every fourth day.

When the distance covered is over 15 or 16 miles, men should halt for dinner, and have an evening meal on reaching camp. Marches should not be too long prolonged, especially at first ; regular halts should be arranged for, and at least on one whole day during the week, with a short march on one other day. The other rules are simple : ample food, good water, and lighten and adjust the loads a man has to carry in every possible way. No spirit ration is necessary, and none should be allowed.

If a malarious tract of country has to be crossed, this should be done in a temperate climate in the daytime, and in the tropics in the afternoon. The poison of malaria is more powerful in the very early morning than at any other time, and when the march can be accomplished without a halt this should be done. If the distance is too great, and a halt is necessary, a dose of quinine may be given, and the men cautioned against exposing themselves to the early morning air more than is necessary. Before leaving camp the men should have a good ration of bread, coffee or cocoa. The men should start on the march at ' quick time,' and in the most open order, for if the ranks close up the temperature mounts up, and the air is vitiated with the products of respiration and transpiration.

If the march is a continuous one and in a tropical country, men should be allowed to unbutton their coats, and be relieved from all superfluous weight. Good water or cold tea should be provided, as any deficiency in the supply of fluids will obviously diminish perspiration, and the temperature of the body will rise. The effete matters got rid of by free perspiration will be retained, and a condition favouring heat-stroke established. In the tropics

an ample supply of fluids is absolutely necessary, and in addition men should be encouraged to bathe their faces and hands when opportunity offers at the halting-places.

At the end of a march men should be dismissed from parade as soon as possible, so as to avoid inducing further fatigue. On dismissal, they should wash their feet and may change their socks: this is the best preventative against foot-soreness.

Exposure to wet and cold in temperate climates tells severely on the soldier. If at night he can obtain dry clothing and comfortable lodgings, he can bear, without much risk, great exposure; but if, as is often the case in this country, he suffers at night as well as during the day, rheumatism, colds, and dysentery follow. In the German Army provision is made against wet, as far as possible; the shelter tent is carried by every two men, and the canvas can be unrolled and worn by the bearer as a water-tight *poncho*. It would be very advisable to issue waterproof sheets to the men, which would afford protection against wet both day and night. They have been found of the greatest benefit by those who have tried them.

ARMY STATISTICS

Statistics are intended to show the effect of military life, and the influence of service in various countries and under different conditions of climate.

Statistics of army disease and mortality were first commenced after the Peninsular War, but these were issued at long and irregular intervals. They gave much information with regard to the topography and climate of various stations, and were continued from 1817 to 1858. The advent of the Crimean War appears to have put a stop to any further issue for a time; in 1859 they were again published, and since that year have been issued annually. These returns show the amount of loss the army incurs annually from disease, not only for the Army as a whole, but for each particular arm of the service, as well as for each station or command at home and abroad.

In order that the results may be compared with those taken from the civil population as well as those given in previously published medical statistics, it is necessary that the classification shall be as far as possible uniform; for this purpose the official *Nomenclature of Diseases* is adopted in the British Army return. Unfortunately, owing to different classifications being adopted in different countries, it is not always possible to compare the sickness and mortality for special diseases occurring in different armies. To obviate the inconvenience arising from this, Dr. Billings¹ suggests that the following diseases should be given separately from any system of classification, whenever they occur among troops—viz. cholera, yellow fever, plague, typhus, relapsing fever, cerebro-spinal fever, diphtheria, tetanus, and specific influenza.

But little information is gained by recording the statistics of disease and mortality as a *whole*; the most important factor in dealing with army statistics is *age*, for unless the mortality is given by age-groups it is impossible to compare the effects of military service with those relating to the civil population. The special points to be determined with reference to Army Statistical Reports are as follows:—

1. The number of admissions into hospital as compared to the number of persons furnishing the sick.

This is obtained by taking the numbers of the body furnishing the sick,

¹ *International Medical Congress Reports*, 1890.

and the sick, and reducing both to a comparable standard : thus a regiment 1,080 strong furnishes 28 sick in a week ; reduce this to a percentage

$$\frac{28 \times 100}{1,080} = 2.18 \text{ per cent.}, \text{ or } 21.8 \text{ per 1,000.}$$

Of course, the numbers furnishing the sick are constantly reduced by the sick entering hospital ; as a rule, an equivalent number usually leave hospital ; if the numbers are very different, a calculation must be made to equalise them.

It is always desirable to express what would be the admissions in a year into hospital, supposing that the strength remained the same, and furnished every week the same number of sick. This result is obtained taking the daily admission per 1,000 and multiplying by 365. In this case = 1,107 per 1,000. Therefore, the whole regiment, or what would be equivalent to it, would pass through the hospital if 28 men were admitted weekly.

There are here, of course, three variables—

- (a) The successive days or weeks.
- (b) The strength of the regiment.
- (c) The number of sick.

But to simplify matters, it is sufficient to take the first and last, and to leave out the varying strength, unless this is considerable.

The admissions into hospital are advantageously considered from the point of view of age, to show whether the younger or the older men are suffering most. For this we require to know the number of men in the regiment at every age :—

18 to 20 years.	28 to 30 years.
20 „ 22 „	30 „ 35 „
22 „ 24 „	35 „ 40 „
24 „ 26 „	40 „ 50 „
26 „ 28 „	

Then reduce to a comparable standard.

As the number of men at a given age are to the number of sick at that age, so is 1,000 for example. Supposing that between the ages of 20 to 22 there are 168 healthy men furnishing 22 sick in one week, while between the ages of 26 to 28 there are 115 men furnishing 7 sick, then,

$$\frac{22 \times 1,000}{168} = 131.1 \text{ per 1,000}$$

and
$$\frac{7 \times 1,000}{115} = 60.8 \text{ per 1,000 ;}$$

therefore, the younger men are more than twice as sickly as the older ones.

2. The deaths in hospital must be considered, as compared with the number of cases treated. In a given time we have to state how many persons have been treated, how many have died, and then to reduce this to a comparable standard. To get the number of persons treated, we may adopt several ways.

(a) Take half of the cases admitted and discharged. Supposing in one week 40 cases were admitted and 10 discharged, then $40 + 10 = 50 \div 2 = 25$, the average number treated.

(b) This may also be got at in the following way. Supposing we have in hospital—

80 Remained	call that R
40 Admitted	„ A
60 Remaining	„ L
10 Died or discharged	„ D

Then to get the average sick treated in the time, add half the remained to the admissions and deduct half the remaining.

$$A + \frac{R}{2} - \frac{L}{2} = \text{number treated,}$$

$$A = 40 + \frac{80}{2} = 55 - \frac{60}{2} = 25.$$

Deduct from the discharged half the remained, and add half the remaining.

$$10 - \frac{80}{2} + \frac{60}{2} = 25.$$

Then reduce the mortality to a comparable standard. Supposing in a hospital 557 are admitted and 287 are discharged in a given time, say 68 days, then $557 + 287 = 794 \div 2 = 397$ cases treated in the time in which the mortality is to be calculated. Then supposing that the mortality has been 15. Reduce this to a percentage

$$\frac{15 \times 100}{397} = 37.8 \text{ per cent., or } 37.8 \text{ per 1,000.}$$

To make this still more intelligible, let us reduce it to a uniform time, say, as given above, 68 days; then if the mortality continued, let us see what it would be in a year.

$$\frac{365 \times 37.8}{68} = 218.1 \text{ per 1,000;}$$

that is, if, instead of 397 men only, 1,000 had been in hospital constantly, 218.1 would have died in a year if the mortality had been the same as in the 68 days.

Instead of the cases treated, the 'sick population' may be taken; that is, the number of patients in hospital on each day as an average.

To get the 'sick population,' add the numbers put down as remaining on each day or on each week, and divide by the number of days or weeks. This, of course, gives the average numbers in hospital on any one day.

The mortality may be calculated on the sick population, by dividing the deaths by the sick population and reducing to a uniform standard of time, say one year.

8. The next point is to determine the number of days a patient is in hospital.

(a) Add all the days together and divide by the cases treated, or

(b) Multiply the sick population by the number of days over which the return extends, and divide by the cases treated. For example—say the sick population is 28, number of days 7, and the cases treated 89, then $28 \times 7 = 161 \div 89 = 4.1$.

Each patient was therefore in hospital rather over four days.

If in any individual case we wish to know the number of days, we must, of course, have the dates of admission and discharge of the individual. A rough estimate may at any moment be made by remembering that if in the above example the sick population and the cases treated had been equal, each man must have been seven days in hospital. If the sick population had been as above, 28, but the cases treated 46, each man would have been half a week in hospital, or 3.5 days.

The returns required to be furnished by the medical officer in charge of a station or general hospital are as follows:—

1. *Daily Returns*.—This a daily state of the numbers admitted, discharged, remaining in hospital, or died. It is sent to the officer commanding

the station for his information. A return of the men admitted and discharged is also forwarded to the officer commanding each corps.

2. *Weekly Returns*.—This return contains the details for seven complete days, including the average strength by corps, the admissions to and discharges from hospital, the numbers remaining in hospital, the deaths, remarks on all important cases, also on any infectious diseases, and on the sanitary condition of the station. All deaths occurring during the period have to be briefly noted, as well as any important cases under treatment. This return is furnished by all hospitals except those for soldiers' wives and children, hospitals on board ship, and hospitals in the field.

8. *Annual Returns*.—This return is completed and forwarded by each officer who is in medical charge of a hospital on December 31. It includes all the details given in the weekly returns, and, in addition, the average number of daily sick, the average sick time to each soldier, and the average duration of each case of sickness is given. It must include the statistics of all the sick of the Regular Forces who have been admitted during the year. With this return is attached a report in manuscript of medical transactions and prevailing diseases, which should show the bearings of sanitary arrangements thereon.

An annual report is also furnished by the officer in charge of the hospital for soldiers' wives and children.

4. *Special Returns.*—During the prevalence of epidemics, special weekly returns are furnished.

5. *Returns on board Ship.*—The statistics of the following classes of troops are required :

- (a) Troops proceeding on service abroad.
- (b) Troops returning home from abroad.
- (c) Troops proceeding from one station to another.
- (d) Invalids returning home.

6. *Return of Troops on Active Service in the Field.*—These include (a) daily state of sick and wounded, (b) special return of officers and men who have received wounds or injuries in battle, specifying as tersely and accurately as possible the kind of wound or injury and the degree of severity. Weekly returns are also required for hospitals in the field.

EFFECTS OF MILITARY SERVICE

The mortality of the army has undergone an enormous diminution during later years. This is probably due in some measure to a closer selection of the men enlisted, to the lesser difficulty in invaliding under the short-service system, and to the comparative youth of the army taken as a whole. But there can be little doubt that this result is also the outcome in a large measure of the sanitary improvements which have of late years been introduced, and which have lessened the death-rate and invaliding both at home and abroad.

The following table shows this decrease.

<i>Mortality per 1,000 in the United Kingdom.</i>								
								From all causes
Mean of 10 years	1861-70	9·45
" "	1870-79	8·18
" "	1878-87	6·52
Mean of 2 years	1886-88	5·75
1889		4·57

This gross mortality must now be compared with that of the civil population at the soldier's age.

In England the gross male civil mortality at the soldier's age is—

		Mortality per 1,000 of population	
		1871-80	1888
From 20 to 25 years of age		7.32	5.4
" 25 to 35 "		9.30	7.2
" 35 to 45 "		13.74	11.7

The soldiers' mortality, taken as a whole, is therefore under that of the civil population, but this is not taking into account the invaliding for which some addition should be made.

As regards the influence of age on mortality, statistics show that between the ages of twenty and thirty-four the mortality is in favour of the soldier; after thirty-five years the proportion is reversed and the civilian mortality is lower. The inference which must be drawn is that military life, if prolonged, has an injurious effect—probably, in some measure, the result of climate and of personal habits acquired in it.

CAUSES OF MORTALITY

As regards the causes of mortality, the disease which is most fatal is phthisis; the number of cases invalided during the year 1889 were 1.72 per 1,000 and the mortality during the same year 1.12, making a total loss by death and invaliding of 2.84 in 1889 against 8.18 in 1888: this is about the same as in the whole male civil population at the soldier's age, but must be considered excessive when we remember that the soldier is especially selected and undergoes a strict examination before he is enlisted. The diminution over former years has been exceedingly large, and there is still evidence of further decrease, so that we may reasonably hope for still further improvement in this direction.

Heart disease and diseases of the circulatory system come next in order of frequency. The mortality is in excess of that of the civil population. There is here also a considerable improvement over former years, due to the close attention given to the dress of the soldier, and to the careful distribution of the weights he has to carry, which are now so arranged as not to press unduly on the chest walls. Excessive smoking has also been assigned as a possible cause, as well as the excessive use of alcohol. The real cause of the 'soldier's heart' appears to be due to sudden and violent exertion, undertaken by lads of immature growth under unfavourable conditions of food and clothing.

The next most fatal forms of disease are those referred to the nervous system; from these soldiers do not appear to suffer more than the civil population at the same age; this class includes apoplexy, meningitis, paralysis, mania, &c., and accounts for about 6.6 per cent. of the total deaths.

Acute diseases of the lungs come next in order. It is extremely difficult to say whether in military life these are more common than among the civil population, but from the crude information we possess, the mortality appears to be less than in civil life.

The next group are the continued fevers. Practically the mortality from these is nearly all due to enteric fever. There has been a general decline during recent years, as a rule, the exception being 1885-86, when the disease occurred among the troops who had recently returned from Egypt. Enteric fever was also prevalent in Dublin during these years: the mortality was 0.24 per 1,000 in 1889 against 0.88 in 1888, and 0.27 in 1887. This mortality is less than that of the civil population.

Other diseases, such as diphtheria, scarlet fever, diabetes, &c., account for nearly 28 per cent. of the total deaths which take place yearly.

Although there has been great improvement in the health of the army during recent years, still much remains to be done. Tubercular diseases, those of the circulatory system, and 'fevers,' all more or less preventable, are still excessive in the mortality returns.

LOSS OF STRENGTH FROM INVALIDING

In 1889 the invaliding at home was 15·89 per 1,000, and the deaths 4·57, together 20·46; for the whole Army 14·45 and 9·17, total loss 23·62 per 1,000.

In the ten years 1879-88, the invaliding at home was 20·57, and the deaths 6·40, together 26·97 per 1,000; for the whole Army 18·06 and 11·82, total loss 29·88 per 1,000. During the seven years 1860-67 there were invalided 87 per 1,000, and a total loss by death and invaliding from disease of nearly 46 per 1,000. Thus the loss by invaliding has been reduced more than one-half compared to what it was in former years. The chief reason for this reduction is the system of short service. Phthisis and respiratory diseases, in round numbers, account for about one-sixth, circulatory diseases for about one-fifth, and nervous diseases for about one-eighth of the invaliding.

LOSS OF SERVICE FROM SICKNESS

On an average, 1,000 soldiers furnish from 800 to 1,000 hospital admissions: the ten years 1879-88 gave 839; 1889, 730·4.

The Cavalry of the Line and the Artillery furnish the largest number, whilst the Household Cavalry and Engineers give the least number; in the latter, many of the men are married, and when admitted to hospital are deprived of their working pay, so they seldom seek admission until compelled to do so. The Cavalry of the Line and Artillery are also subject to accidents incidental to the nature of their work.

A very large number of admissions is the result of venereal disease, of which there is no immediate prospect of any considerable decrease. It is not possible to compare the loss occasioned by sickness with that occurring in other armies, as the conditions of service are not the same.

PREVENTION OF DISEASE

The prevention of disease is the great aim of the military surgeon. To carry out this, he receives special instruction in the general principles of sanitary science, more particularly as applied to large bodies of individuals living under varied conditions in different climates.

The best means to bring about the best results is by attention to what may be called personal hygiene, that is, to bring the body into the highest possible state of physical development, so that there may be the initial strength in the individual to resist those inroads of disease a less vigorous constitution would probably succumb to, as well as to allow of the elimination of the poison; and by a knowledge of the way epidemics spread, by tracing their rise and progress, and by investigating into their origin, we may be able, as far as possible, to remove those causes which appear to influence or favour their diffusion. As the entity of each specific disease is each year becoming more established, the means of prevention are more within our reach and are better understood.

Hitherto, in the majority of instances, we have had nothing but broad facts to guide us, and our knowledge of necessity was most imperfect; but

even so, the results have not been unsatisfactory, incomplete though they are, and justify us to continue in our endeavours to remove the causes which appear most likely to produce disease ; as our knowledge increases, errors will be recognised and guarded against.

It is here intended to state as briefly as possible the rules which should guide us in dealing with this question in military service : outside these limits would involve a discussion on very many complicated questions which are more conveniently treated in another section.

THE SPECIFIC DISEASES

Cholera.—Until R. Koch in 1884 discovered the *comma bacillus* in the intestinal mucous membrane of cholera patients, the parasitic theory did not meet with general acceptance : it is now universally acknowledged that a causal connection does exist between the organisms discovered by him and this specific disease.

In the prevention of cholera the one fact to bear in mind is, that it is spread by human intercourse.

The history of epidemics of this disease in Europe and India are full of examples they never travel faster than man can travel. Hirsch states ' that it is human intercourse which furnishes the media of this communication, is proved on a large scale by observations on the diffusion of this disease, by pilgrimages and military campaigns, both in India and beyond it ; whilst there are other conclusive proofs furnished in innumerable instances from the smaller circles of diffusion.'¹

The Vienna Congress in 1884 decided that there were no facts to show that atmospheric causes could bring on cholera. On the contrary, they stated that ' all facts go to show that in free air the generative principle of cholera rapidly loses its morbid character.'

The localisation of cholera is a marked feature in its history, and fully bears out the conclusions arrived at by the Vienna Congress. It will seize on one side of a street, leaving the other side free ; or attack one side of a barrack, or camp, thus furnishing the strongest evidence against its being air-borne ; no well-established instance of the diffusion of this disease by atmospheric influences has ever been recorded, while we have numberless instances where it has followed in the track of human intercourse.

These two conditions once acknowledged, that cholera is a specific disease and that it is spread by man's agency, we shall have little difficulty in framing the lines on which preventive action should be based : briefly, these are to isolate the poison, and to keep men in the highest possible state of health, so as to fortify them against an attack.

With reference to soil as a factor in cholera epidemics, we have to note that the disease has attained its widest diffusion on permeable damp soils and rocks capable of receiving and retaining moisture. The influence of the soil *per se* is very slight ; the disease appears to depend most on the physical condition of the soil itself, and especially with reference to its permeability to water and air ; and such soil, resting on a rocky bottom, wherever the latter has disintegrated by the weather, or broken up or covered by detritus, appears to favour the diffusion of cholera. Such knowledge teaches us to select *dry* sites for camps or barracks.

In selecting men for a campaign in India, there are certain individual predispositions which it is well to note. A certain immunity seems to exist among the old soldiers compared with new arrivals in India. Bryden has

¹ *Geographical and Historical Pathology*, New Sydenham Society, 1863.

shown how much more susceptible newly-arrived troops are, if cholera breaks out, and the risk there is in employing such troops on service.

The following table is given by Bryden :—

—	Lucknow 1864	Lucknow 1869	Benares 1865	Meerut 1869
Mortality of new troops from cholera . . .	27	42	9	105
„ old „ „ . . .	11	11	—	6

As regards race, there is almost complete unanimity that the negro race is especially prone to cholera.

Dr. Christie shows that on the East Coast of Africa the mortality amounted to 6·5 per cent. of the population among the Hindoo coolies, 10 per cent. among the Arabs, but 25 per cent. among the Negroes. In the United States Army the epidemic of 1866 gave a mortality of 7·7 per cent. among the white troops, and 185 among the black. This liability to cholera by the negro race shows that they should not be employed in campaigns where there is any likelihood of this disease occurring.

In India, Europeans suffer much more severely than natives. Although European troops are more liable to attacks than native troops, the mortality related to the sickness is slightly greater among natives than Europeans (Hirsch). Bryden's observations, however, do not confirm this view, but show that European troops give a larger mortality than native. Bryden has also pointed out that among natives of Hindoostan there is a certain immunity among the dwellers in the plains as compared with hill-men. This should be recognised in selecting men for a campaign in any countries where there is liability to cholera.

Nothing predisposes more to the propagation of disease in the tropics than density of population. There should be no overcrowding, and whether in camp or barracks there should be free perflation of air. The men should be spread out as far as possible, and too many should not be placed in one tent or room.

In India, cholera attacks men less frequently in the hills than in the plains, although high elevations afford no security against this disease. The physical condition of men serving in the hills is better, and they are more able to withstand sudden outbreaks of epidemic disease on this account. The ground is also drier, there is less moisture, and seldom any subsoil water near the surface.

In tropical countries all sickness, as a rule, decreases with elevation, and for this reason sites for camps should be on elevated ground where military arrangements admit.

There is abundant evidence to prove that cholera epidemics are due in many instances to the use of impure water. Dr. Simpson,¹ the Medical Officer of Health for Calcutta, in his sanitary reports on that city, shows that any increase of this disease in certain districts can be almost invariably traced to the scarcity of wholesome water, necessitating the use of an impure supply from tanks and wells.

The introduction of the public water-supply into Fort William, Calcutta, has effectually stopped outbreaks occurring there, which formerly were of frequent occurrence.

In India all wells in close proximity to native villages should, during the prevalence of cholera, be looked upon with suspicion, and water taken from them should be boiled before being used for drinking purposes.

¹ *Report on Sanitary Measures in India, 1867-88, p. 147.*

Streams and rivers should likewise be avoided unless their source is known, and in any case the water should be subjected to some process of filtration or boiled before being used.

Another factor in the dissemination of cholera appears to be a contaminated milk supply. The sanitary condition of cowsheds should be attended to, and a plentiful supply of pure water insisted on.

No spirit ration should be issued during cholera epidemics : the disease is most prone to first attack the intemperate men in a regiment. It would appear also that by destroying the natural acidity of the stomach it favours the passage of the cholera germ.

Professor Matthew Hay has pointed out that the reaction of the secretion in the stomach in cats is alkaline, and that the injection of water or neutral saline solutions does not excite an acid secretion. The same is also the case in man. It is known from Koch's researches that the *comma spirillum* flourishes in alkaline but dies in acid media. It is also admitted that something more than a casual connection exists between the *spirillum* and the disease, and therefore it is evident that swallowing water on an empty stomach would be a dangerous proceeding if the water were polluted ; also, that after fasting or in the early hours of the morning before breakfast, the stomach is in such a condition as to afford a suitable resting-place for, or to allow the passage through of the *spirillum*.

If gastric disturbances are prevalent, as shown by men suffering from diarrhoea or dysenteric attacks, the danger from the introduction of cholera is greatly increased. Unripe fruit or any excess of fruit should be avoided, and while ample food is provided, this should be of the best kind and able to be easily digested. There should, however, be no excess.

The possibility of the conveyance of the poison by insect life (flies) should not be overlooked ; the number and kind vary according to the place and season ; special provision should be made to obviate this source of danger. The inspection of all food supplies requires the greatest attention, as the more cleanly the mode of its preparation and the more carefully water, milk, and food supplies generally are guarded against cholera contamination, the fewer will be the dangers of infection therefrom.

No duty should be done until the men have had an early breakfast ; the stomach secretions are alkaline, and are therefore ready to receive the germ : men on sentry duty should not be permitted to fast too long, for the same reason, but have coffee or cocoa served out to them at stated hours. If native cooks are employed in barracks, the strictest supervision will have to be exercised over all food supplies and cooking arrangements.

All evidence shows that over-fatigue predisposes to cholera, therefore long and fatiguing marches should be avoided ; fatigue is perhaps the only internal cause which is well established, and seems to produce a condition which favours an attack ; in cholera times marches should be so arranged that the men have regular halts, and are not constantly fatigued.

When cholera appears in cantonments in India, the rule is that on a case occurring in any building occupied by European troops, such building should be immediately vacated, and the affected individuals isolated as far as possible from those among whom there have been no cases of the disease. If a second case occur among the body of men thus removed, they are again moved. If a third case occur among this particular body of men within a week, they are immediately transferred from the station to a selected camp. If cases occur in several barracks simultaneously, all must be moved into camp. The Regulations order that such buildings are to be vacated as have actually presented cases.

If the disease breaks out in the preparatory camp, the site must be changed. As a rule, cholera in camp is not confined to one part, and the safer plan is to move the whole camp, if any cases occur. The golden rule to observe during cholera epidemics is, to move away as quickly as possible from the infected area: often a very short distance will suffice for this, but as long as cases occur, so long is it necessary to change the site. All marches should be made in the early morning, when the men are fresh, and when the liability to chill is at a minimum.

It has been recommended not to place men in tents or move into camp during the rainy season: but so great are the advantages gained by leaving an infected area, that the rule should be to march under any circumstances, if this can be done.

If on active service cholera makes its appearance in the camps, military reasons may not admit of a change of site; under these circumstances the sick should be segregated to one part of the camp, to leeward if possible, and strict quarantine enforced: no communication should be permitted, and if it can be done, a new source of water-supply should be obtained. The men should avoid damp in every direction—damp tents, damp clothes, &c.—and woollen clothing should be worn next the skin, the great object being to avoid chill, as in tropical countries the abdominal region is very liable to be attacked by cold.

Dr. Tuson has advocated the use of burning sulphur to destroy the contagium of cholera; he recommends sulphur fires to be lighted fifty yards apart round the camp, and to be kept lit during the prevalence of the disease; the fires to be placed especially to windward of the camp. As it is extremely improbable that the air conveys the infectious material at all, and almost certain that it never takes it long distances, it is doubtful whether this method has any practical advantage.

All measures of prevention should be based on the fact that the active cause of the disease is a germ attacking men by way of the intestinal canal; that the active cause of the disease is contained in the evacuations, and that when this reaches the intestines of another susceptible person by the agency of water, milk, food, &c., the disease is transmitted.

The tendency to infection varies in different individuals, and never appears to be very large; disordered digestion appears certainly to increase the susceptibility of a person to be attacked by cholera.

The *materies morbi* of cholera being present in the stools, and as they are the most important, if not the only poison-bearers, the disinfection of them is of the highest importance. The most powerful disinfectant is perchloride of mercury. It should be acidulated. The following formula is recommended by Dr. Parsons:—Mercuric chloride, $\frac{1}{2}$ oz.; hydrochloric acid, 1 oz.; aniline blue, 5 grains; water, three gallons. The discharges should remain in contact with the disinfectant for fifteen or twenty minutes. In camps the stools after thorough disinfection should be deeply buried at a place far removed from any water-supply. De Chaumont recommended that the stools should be mixed with sawdust and burnt, and this latter plan, which is by far the safest, is now capable of being put into use by means of the 'Fæcal Cremator,' which ensures the total destruction by fire of fæcal matters and other hospital refuse.

The Rome Conference recommended solutions of carbolic acid and chlorinated lime (5 per cent. of carbolic acid and 4 per cent. of chlorinated lime) to be mixed in equal proportions with the dejecta and vomit, and subsequently buried. Carbolic acid has one advantage: it readily imparts a most disagreeable taste and smell to water, even when present in a minute quantity, and thus indicates if any water-supply is contaminated by the soakage from cesspools or privies. In cases in which there is no other means of disposal,

it may be permissible for cholera discharges, if very thoroughly disinfected, to pass into rivers; but this is a contingency which should very rarely arise and not be permitted except under circumstances of very great necessity; in no case should this be allowed when the stream is a possible source of water-supply for drinking purposes. In the cholera epidemic of 1865-66 in England, in towns with well-constructed sewers, cholera never attained any large dimensions, neither was there any evidence to show that the system adopted of allowing the disinfected excreta to enter the sewers in any way contributed to its spread.

All articles of clothing, blankets, &c., which have been fouled by the patient, or used by him when suffering from the disease, should be burnt. The bedding, which is usually of straw, should also be burnt. Such clothes, &c., as cannot be so disposed of should be soaked in a solution of mercuric chloride (1 in 1,000) and if possible disinfected by moist heat, and subsequently dried in the open air. If no disinfecting chamber is available, boiling from three to four hours is the next most effectual plan.

Tents in which cholera cases are treated should be disinfected also. The best plan is to soak the tent in a solution of mercuric chloride (1 in 1,000), and subsequently let it be exposed to sun and air until quite dry: this is a safer plan than merely submitting it to fumigation by one or other of the ordinary methods generally adopted. Whatever process is adopted the tent should not be again occupied for ten days, during which time it should remain fully exposed to the air.

The bodies of those who have died from this disease should be cremated if possible; but the means are not always available for doing this, neither has custom altogether sanctioned this method of disposal of the dead. If ordinary burial is resorted to, chlorinated lime or good quicklime should be freely used, and deep burial in all cases insisted on.

It cannot be too forcibly stated that the spread of cholera after its introduction into a place is universally associated with contaminated water, filthy habits, and bad personal and domestic hygiene.

Enteric Fever.—Enteric fever, both as regards its frequency and diffusion, marks widely its difference to all other forms of disease; it knows no geographical limits, and its very universality makes it one of peculiar interest to military surgeons.

The infective agents in enteric fever are present in the intestinal dejecta of patients: whether these infective agents are in the form of a bacillus or a micrococcus is still open to doubt; but the whole epidemiological history of enteric fever indicates that the infective virus is a microbe of some kind, and, moreover, one which can thrive in soil, water, sewage, &c., and is, too, possessed of considerable resisting power against cold, drying, and putrefactive processes generally.

Hence it has rightly become an accepted fact that the disease may be spread by infected linen or bedding, even after the lapse of some time.

The micro-organism commonly associated with the propagation of this disease is a bacillus first discovered by Eberth and subsequently confirmed by Gaffky as existing in the contents of ulcerated Peyer's glands, in the mesenteric glands and spleen of the typhoid sick. These bacilli have never been satisfactorily demonstrated outside the body, although some observers state that they have found them in drinking-water. More recent investigations are tending to raise doubts whether the Eberth-Gaffky bacillus is the only or real cause of enteric fever: the matter is, however, one of much difficulty, and at present by no means finally settled.

The two chief predisposing causes in tropical enteric fever are *age* and

recent arrivals within the tropics. The increased predisposition at an early age is now universally admitted as a character of the disease. Murchison shows that the mean age of 1,772 cases admitted into the London Fever Hospital during ten years was 21·25 years. Dr. Collie¹ gives the statistics of 8,528 cases of enteric fever admitted into the three fever hospitals under the Metropolitan Asylums' Board as follows :—

Age	Percentage of enteric fever
10-14	22·60
15-19	25·00
20-24	26·80
25-29	9·75

The same rule holds good in the tropics. Youth in every climate predisposes to enteric fever, no doubt because the glands of Peyer are in their full functional activity. The relationship between enteric fever and *age* among European troops serving in India is exhibited by the following table :—

Table showing the Death Ratios of the European Army from Enteric Fever at different Ages.

	Under 25	25 to 29	30 to 34
1879-88	5·86	2·27	0·84
1888	5·46	2·86	0·84

As with age, so with length of service in the tropics. With length of service there is diminished liability. Taking the Indian return again as affording the most trustworthy information, we find this connection fully borne out by the following table :—

Table showing the Death Ratios of the European Army at different Periods of Residence in India.

	1st and 2nd years	3rd to 6th year	7th to 10th year
1879-88	6·54	2·14	0·70
1888	6·89	2·77	1·08

A comparison of these tables shows that shortness of Indian service is even more powerful than youth as a predisposing cause of enteric fever.

As regards *Season*, it is noteworthy of remark that in the tropics and sub-tropics it is chiefly the hot months that form the enteric period. This is seen from the following table :—

Table showing the Number of Admissions from Enteric Fever in each of the Four Quarters of the Year 1889.

	Total admission	1st quarter	2nd quarter	3rd quarter	4th quarter
Bengal . .	1,100	178	499	295	188
Madras . .	221	12	28	146	85
Bombay . .	257	33	35	166	23

Elevation appears to have no influence on the occurrence of the disease. This applies not only to the temperate zone but also to the tropics.

The relation between enteric fever and the physical characters of the soil is important, especially in tropical countries.

The fate of the Eberth-Gaffky bacillus in soil is early extinction, particularly in the presence of extreme moisture and great dryness. The formation of

¹ *British Medical Journal*, April 1885.

spores by this bacillus has been questioned, though Eberth, Gaffky, and others maintain that it is easily capable of forming true spores. Unless it be capable of forming spores, and thereby be able to survive antagonistic conditions, it is difficult to reconcile the connection of this particular bacillus with enteric fever outbreaks. We are unable to absolutely affirm that the soil serves as a breeding-place for the entire virus; but presuming that it can assume a spore formation, we are justified in considering that the soil can serve as a habitat for spores or such resting forms as may reach it either directly from the sick, or indirectly as the result of processes undergone by typhoid dejecta deposited or buried in it. The practical bearing of these considerations is to lend some confirmation or explanation of the local and seasonal differences in the distribution of typhoid fever, the dominant factors being a certain degree of soil-heat and level of soil-water.

Enteric fever in India prevails in its most virulent form chiefly in the months of April, May, and June. In these months the upper layers of the soil are at their driest, and have a mean temperature throughout the twenty-four hours of 72° F. As this superficial soil becomes moist consequent on either rain or other causes, or when its mean daily temperature falls below 60° F., then the disease abates.

The maximum range of soil temperature co-existent with disease prevalence is undetermined, but it is probably very high. High levels of subsoil water are co-existent with the moistness of the upper soil layer and consequent mechanical fixation, if not actual destruction, of the *contagium vivum* of typhoid fever, as the result of decomposition or of saprophytic action. This agrees with a minimum prevalence of disease at times of considerable rise in the soil-water.

The condition of loose or porous soil after a fall in the soil-water and consequent access of air to the soil interspaces, to say nothing of increased heat, establishes facilities for the virus (now become potent, whether as spores or not) to be carried into the atmosphere with the upper layer of soil, either as dust or with ascending air currents.

Without going so far as to say that any fluctuations of the subsoil water have no effect as mechanical agents for the introduction into wells of the typhoid spores or virus which may happen to be lodged in the soil, yet, in the light of what we know to be the behaviour of the Eberth-Gaffky bacilli in soil and the general limitations of microbial life to the upper layers of the soil, and the remarkable filtering influence of soil on the passage of bacteria through its interstices, one is forced to think this contingency is rare. If enteric dejecta do gain access to drinking-water and wells from the soil, it is more probably by surface than by deep drainage.

Of course, outbreaks of this disease occur in places through various other circumstances, but they do not vitiate the value or importance of these conclusions regarding soil heat, soil dryness, and soil moisture.

From this brief summary we can fairly well indicate the nature of the preventive measures for enteric fever in tropical and sub-tropical countries. The age of men serving in the tropics, and especially in India, should be increased. At present recruits are sent to India just at the age when they are most predisposed to enteric fever. Owing to the short-service system of enlistment there is some difficulty in doing otherwise, but something will have to be done, for it is impossible to conceive that the State will stand the waste of life entailed under the present system.

The immunity acquired by residence is an important factor in the etiology of this disease. The fact is well established that in India, with lengthened service there, the liability to become affected by enteric fever

rapidly diminishes. This immunity, acquired through acclimatisation, is unquestionably much more perfect in tropical and sub-tropical regions than in higher latitudes (Hirsch). Men, therefore, should be allowed to remain in the tropics, and not constantly changed only to be replaced by others who are more susceptible. With these facts before us, it can hardly be said that the present system of short-service enlistment for the European Army in India is one to be commended; and it is a question whether it should not be replaced by some system better adapted to the requirements and wants of that country. From a sanitary point of view, what is needed is an older army, composed of seasoned troops, who will remain some years in the country and acquire that protection which only residence in a tropical climate can give.

The site of any camp in the tropics should be as high as possible, and the ground dry. The subsoil-water should not be near the surface, as moisture favours the development of the enteric bacilli. If enteric fever breaks out in the camp, the site should be changed at once. Nothing appears to favour the spread of this disease more than occupying the same ground for a lengthened period. Dry sandy soils are particularly favourable for the spread of enteric fever when it has once been introduced, as the dry sand possesses little disinfecting properties. On such sites all excreta should be carefully disinfected, and the trenches used by the men should be deep and narrow.

There are few points in the etiology of typhoid fever so certainly proved as the conveyance of disease by infected water, or by food contaminated with infected water. In this latter class milk has generally served as the carrier of the enteric poison. Many examples in the histories of campaigns could be given to show the part played by water in disseminating this disease, but the fact is so well recognised now that it is unnecessary to enter more fully into the subject here. It is enough to state that the protection of the water-supply from any possible sources of contamination demands our most careful attention. The site from which the water for drinking purposes is taken should be guarded from pollution, and where there is any doubt as to the quality all drinking water should be filtered and boiled.

The same scrupulous attention should be given to the milk-supply, and this should in every case be boiled before distribution; and, indeed, speaking generally, all sources of food-supply should be inquired into, and means adopted to keep them pure and wholesome.

If the disease appears in camp, the next important point to look to is the trenches, which should always be separate for enteric cases, and as far to leeward as possible. The dejecta, after careful disinfection with mercuric chloride solution, should be buried. The stools when first voided are not infectious, therefore they should be disinfected at once. In using mercuric chloride as a disinfectant the solution should be rendered acid, which prevents the mercury being precipitated by the albuminous matter in the stools—the formula used to disinfect cholera stools answers the purpose well. If carbolic acid is used, the solution should be 1 in 80, and it must remain in contact with the dejecta for some hours. Hence corrosive sublimate is more efficacious, and better adapted for military purposes. The trenches themselves should be four feet deep at least, and one-and-a-half feet wide. When the trench is half filled in, it should be closed and the earth well beaten down.

All articles of bedding and clothing which have been soiled should be disinfected. It should not be forgotten that the enteric poison has great vitality, and preserves this for months. Blankets, &c., after being soaked in corrosive sublimate solution, should be disinfected by moist heat. If this is not available, then boiling for three or four hours will generally suffice to disinfect them.

It is not always possible to submit to the action of heat such articles as mattresses, &c., and if such be the case they had better be burned. It is not safe to rely on fumigation only to purify them.

Great care should be taken that the disease is not communicated to the attendants. They should be required to take their food in a separate room or tent, and to wash their hands in some disinfecting solution before partaking of their meals. For reasons previously stated, the attendants on enteric cases should be taken from the older class of soldiers.

The result of all preventive measures really depends on recognising the first cases, and dealing with them at once by isolating them and their attendants, and by carefully disinfecting *all* dejects, clothing, bedding, &c., in the manner already directed. Should the disease become epidemic the camp must be evacuated, and a fresh site, to windward if possible, selected.

Yellow Fever.—Yellow fever is a disease which is very limited in its habitats. Throughout the whole of the yellow fever region there are only three situations at which the disease assumes an endemic character—the West Indies, the Mexican Gulf Coast, and part of the Guinea Coast. From this it has been introduced at several points on the western shores of Europe, but it has never obtained a foothold there.

The disease was at one time believed to be of malarial origin, and there were some who considered that the two agencies which produce yellow fever and paroxysmal fevers were inseparately associated, and are referable to telluric emanations.

The experience of later times has, however, distinctly shown its separation from, and independence of, malaria, and its diffusion by personal intercourse.

Geological characters of the soil do not appear to exert any influence, either in the production or diffusion of yellow fever, but climatic conditions, and more particularly temperature, do exert an important influence upon its spread. Its causation is most closely associated with the presence of fecal and other waste organic matters, such as accumulate around human habitations in densely-populated towns.

As regards season, within the endemic zone yellow fever occurs at all seasons, yet in the cold season it is only sporadic. The greatest prevalence of the disease falls in the period from April to September. The two chief factors on which an epidemic depends are temperature and moisture. An isotherm of 68° F. is necessary for an epidemic to develop, but, once started, it is not dependent for its continuance on a relatively high temperature.

The moisture in the air constitutes the second factor in the production of yellow fever. It occurs only to a slight extent, if at all, in years with little rain, dry weather or long-continued drought being unfavourable to the diffusion of the disease.

It is said to prevail as an epidemic only when the temperature of the dew-point does not fall below 74° F.

As regards the nature of the poison, little is known.

Dr. Domingos Freire has found a micrococcus which he terms *Cryptococcus xanthogenus*, and which he regards as the cause of the disease. His observations have not been confirmed, and this theory has not been accepted. So far no specific organism has been discovered which is admitted as the cause, or has in any way been specially identified with the disease.

A point of special interest in the history of yellow fever is the influence acclimatisation and race have upon its distribution. New arrivals from northern latitudes, and those not yet acclimatised, are most subject to the disease, while the native and acclimatised whites enjoy more or less exemption from attacks. This comparative immunity is only to be acquired after

long residence within the endemic zone where yellow fever occurs. The predisposition to attack increases with the distance from the tropical zone, and for this reason troops, before taking a tour of service in the West Indies, first pass three or four years in one of the Mediterranean stations. A previous attack also confers immunity from another one. Creoles enjoy almost a perfect immunity. The negro race appear to enjoy a congenital protection from attacks of this disease, which immunity is all the more complete the more purely the racial characteristics have been preserved; and this is the case whether they are acclimatised or not.

Yellow fever is especially a fever of towns and cities, and not of country districts. The accumulation of filth arising from defective drainage and general inattention to cleanliness seems to be recognised as being intimately connected with localising causes, and this disease, like cholera and enteric fever, has not inaptly been called a filth disease.

An epidemic outbreak of yellow fever never happens suddenly. A series of isolated cases always precede it for a longer or shorter period, after which the epidemic will usually come rapidly to a head. It will exhibit many fluctuations while it lasts, depending chiefly upon the influx of strangers, and will die out gradually, or it may be suddenly, under climatic conditions unsuitable to its further existence.

Given an area in which yellow fever is endemic, it is obvious we are dealing with the disease at its source. It is not possible to alter the essential characters of the soil, or to introduce other conditions of climate into the place, but it is possible to effect some changes in it which will destroy the power of developing the poison.

As yellow fever haunts the low and filthy quarters of seaport towns, the best results will be obtained by a levelling and draining of the soil, the immediate and effective removal from it of all animal and vegetable refuse, and by providing an efficient system of sewage removal. No sewage should be allowed to flow in tidal harbours or the mouths of estuaries, and in such places the dry-earth system is the only one which can with safety be adopted. Cleanliness, in the widest sense, is absolutely necessary, as well as a pure water-supply, and careful attention to all sources from which food is taken.

When the disease has once broken out, our main object must be to arrest its spread. The infected habitation must be immediately evacuated, and segregation of the sick carried out at a distance which is not already a focus of the disease, nor in a condition to become so. The buildings should be fumigated and re-limewashed, and not again occupied for several days. Complete and thorough disinfection of all dejecta, notably the vomit and alvine discharges, by mercuric chloride solution, as already indicated, and then deeply buried and not allowed to pass into any drain or latrine used by others. The attendants on the sick should be taken from the least susceptible classes of the community—that is, from those on whom such is conferred by a previous attack, or from the negro population.

All bedding and clothing which cannot be thoroughly disinfected by moist heat should be burnt. The bodies of those who have died from the disease should be disinfected by chlorinated lime or good quicklime, and deep burial required in all cases. All communication with the infected area should be restricted, and in the case of troops, entirely prohibited.

Such are the main lines to work upon with regard to the prevention of yellow fever. Like cholera, the poison no doubt is specific, although no particular organism has yet been found. The action is primarily on the large intestines, the discharges from which, in those affected by the disease, are capable of giving rise to the same disease in others.

The origin of the disease is enveloped in obscurity. It has been suggested that the disease poison primarily took its origin from the filth of slave-ships, and dysenteric dejecta deposited on certain harbours and on certain shores at which place the disease still tends to prevail under certain existing conditions. Dr. Creighton points out that the slave-trade is the only one circumstance covering its history, geography, and distribution. The advent of yellow fever coincided with the rise of the slave-trade, and it has ceased in many of the great cities of America after the importation of negroes ceased. The disease is essentially one of places rather than men, confining itself to the sea-coasts and the shores of great navigable rivers; and these facts show that the disease poison exists in the foreshore soil and mud of these parts.

Whatever is the cause, there is now no doubt but that epidemics of yellow fever have of late years diminished both in frequency and degree, and if the hypotheses of Dr. Creighton be true, we may reasonably hope that, now that a cruel slave-trade has ceased to exist, the disease will gradually die out and disappear under a more enlightened hygienic rule.

Malarious Fevers.—Malarial fever in one of its varied forms is the disease most frequently met with on military service. Extending to a broad zone on either side of the Equator, they reach their maximum of frequency in tropical and sub-tropical regions, where they become endemic, diminishing both in frequency and intensity as the higher latitudes are reached.

The exact nature of the malarial poison has been the subject of much discussion. Klebs and Tommasi-Crudeli found bacilli in the marshy ground in a malarial region, and described these organisms as '*malaria bacilli*.' The statement with regard to the morphological characters of these micro-organisms is not satisfactory, and on this account their views have not been accepted.

Laveran¹ has described in detail the appearances of certain organisms which he believes to be the cause of malaria: there are three forms, spheres—spheres with flagella, and crescent-shaped bodies. He considers these bodies to belong to the animal rather than to the vegetable kingdom (*hæmatozoaires*).

The French Academy of Sciences have adopted Laveran's views, stating that 'the discovery constitutes by itself all the pathology of intermittent fever.'²

By far the most common variety of the disease is the intermittent malarial fever; this form is universally distributed in the temperate zone in northern latitudes as well as in the tropics and sub-tropical regions.

The remittent type, as well as the severer forms of malarial fever, are principally confined to tropical and sub-tropical countries. There is no acclimatisation for these diseases; natives as well as Europeans suffer, the latter from the more severe and pernicious forms, while natives, if they remain in their own homes, are more often attacked by the milder disease and suffer less severely.

All observations tend to show that the disease poison is elaborated in the soil during decomposition under the combined influence of heat and moisture.

Predisposing conditions largely enter into the soldier's life, particularly on service in the field, where often the vital powers are lowered and the life has a depressing and exhausting tendency. Such conditions are brought about by exposure, often by improper food given at irregular times, and ex-

¹ *Traité des Fièvres Palustres*, 1884.

² *Comptes Rendus*, 1889, ii. 1091.

cessive labour, incurred by forced marches, &c. These render the system less liable to withstand the influence of the malarial poison.

The production of malaria in soil seems to depend upon the saturation of the ground with subsoil water, and where such a condition exists we find malaria most fully developed; for example, on rice fields, cotton marshes, and the damp alluvial banks of rivers; over irrigated lands where much cultivation is carried on, as in India, and in localities where the subsoil water is near the surface from canals or subterranean springs. Any of these conditions producing a wet soil furnishes an intimate causal connection between it and the production of the disease, and this is all the more intensified when men are exposed to the effluvia from decomposing vegetable masses, and to the results of decay of the vegetable matters in or upon the soil.

Although no race is actually immune to malarial fevers, the negro presents the least predisposition to this disease; he is less frequently and less severely attacked, although he does not enjoy absolute immunity.

In military service the selection of season is most important. Hirsch states that in the regions with moderately-developed malaria there are two maxima, one in spring and one in autumn, with a decrease of the disease in the intervening months. In regions with strongly-developed malaria, the maximum begins in summer, reaching its height at the end of summer, the minimum being winter and spring; while in intensely malarious spots of the tropics, the greatest prevalence occurs during the rainy season. As an example of the benefit gained by the proper selection of season for carrying on military operations in malarious countries, may be cited the Ashanti campaign of 1878. Former expeditions, undertaken during the rainy and unhealthy season, proved most disastrous. In the Ashanti expedition of 1864 the troops marched during the latter part of January; early in March the hospitals began to fill. The rains commenced the middle of March, and by the end of the month hardly an officer was in sound health, and none had escaped fever. In June the whole force had to be withdrawn from the interior, and the expedition abandoned.

Paroxysmal fevers furnished an admission rate of 851 per 1,000, against an average of 115 per 1,000 for the previous five years. Contrasting this with the expedition commanded by Lord Wolseley in 1878, we find the time selected was from October to February, months which were not included in the rainy season on the coast, and where every precaution was taken to avoid keeping men in the interior after the rains set in. The mortality for the whole force disembarked was only 81.4 per 1,000. The expedition was able to accomplish the task it had to perform, and its success was almost entirely due to the care and forethought exercised in the proper selection of the season for campaigning.

As regards the influence of age, the older soldier has the advantage, provided he has not previously suffered from the disease. Young soldiers are most frequently attacked, and appear to suffer more severely.

The time of marching in malarious countries should be the same as in the tropics generally. During the Ashanti War, less harm was found to result from early morning marching than from marching during those hours which would of necessity expose men to the sun. Men should always be provided with a good meal of coffee or cocoa and biscuit before commencing a march. The clothing is of first importance, as the prevention of chill removes an important factor in malarial attacks, particularly in those who have already suffered from paroxysms of this fever. Flannel should always be worn next the skin, and never cotton; it is not only a protection against malaria, but also against dysentery. In damp malarious jungle, cotton is

totally unsuited for clothing. Khaki serge, as used in India, appears to answer all necessary requirements. Every precaution should be taken at the end of a march against men sitting in damp or wet clothes: it is at that time that the most danger is to be apprehended. If possible, a change of underclothing should be provided. Smoking in moderation may be permitted with advantage: it certainly seems to ward off malaria.

The selection of the site for a camp in malarious countries is of importance. Elevation, although it does not afford absolute immunity, gives a certain amount of protection, and the disease diminishes in severity as we ascend above sea-level, therefore, as high a site as is consistent with military requirements should be selected. The greater the altitude also the lower is the temperature, another important factor in the production of malaria. On high ground the soil is drier, and experience shows that increased moisture leads to increased admissions for fever; for this reason damp camping-grounds are to be avoided.

For the same reason marshes, or the banks of rivers, particularly where the water is stagnant, and the neighbourhood of swamps, where generally the processes of decomposition are most active under the influence of very great heat, are hotbeds of malaria. These, of course, should not be occupied if possible by troops. Marshes are only malarious when they are partly covered with water, and present their worst condition when they are undergoing the process of drying up. As long as they are completely covered with water, they do not give off malaria.

In preparing a camp the soil should be disturbed as little as possible. In the tropics, where all the ground has remained fallow for any length of time, it is sure to give rise to malarial fever, particularly if the surface is cut away and the soil exposed to the action of the air. Brushwood and jungle are frequently bad. It is better to avoid a site covered with dense jungle and brushwood, as its removal at first will increase malarious disease consequent on the disturbance of the ground, but ultimately the site may be improved by doing so, and rendered more healthy. If the camp is only to be used for temporary purposes, the ground should not be interfered with. Where removal is necessary, the work should be carried on during the day, and not in the early morning or in the evening.

Trees should be removed with judgment. If they interfere with the free movement of air it may be necessary, to do so, but this will seldom be the case. In the tropics they cool the ground, and may protect from malarious currents. It is said that at some of the hill-stations in India malaria has decreased owing to the removal of the trees for some distance around the cantonments.

The fact that the poison may be introduced into the system by drinking-water should not be overlooked. The malarial poison contained in the water is probably generated by the decaying vegetation, and the fact that the disease, in those places where it is taken in by water, becomes most fatal in the month of October, coincides with the time that there is the possibility of the largest amount of organic matter in the water from the shedding of the forest leaves. Malarial fever thus induced is apt to assume a very virulent form. Mr. Whalley¹ states that the streams which enjoy the most deadly reputation all take their rise in the dense forest, and are overhung for a portion of their course by a thick screen of overarching trees and bushes. Streams which are bordered by sand and boulders are generally innocuous. Unhealthy villages are found mostly along the shallow depressions which

¹ *British Medical Journal*, Nov. 8, 1884, p. 942.

convey the surface water of the forests to the rivers. Dr. E. G. Russell¹ gives many instances of the association of malarial diseases with the use of water draining from jungles or marshes at the base of mountain ranges, and their disappearance on the substitution of a better water-supply.

A water free from vegetable organic matter is, therefore, indicated as the best means of prevention. Vegetable matter alone in water will not produce malarial symptoms, but as malaria is generated in soils abounding in vegetable organic matter, it is reasonable to suppose that the malarial poison will readily enter the water with the vegetable substances.

Surface waters are the most dangerous: water from wells, unless contaminated by surface washings, has never been known to produce malarial fever. If surface water has to be used for drinking purposes, it should be filtered and boiled in every case. It would also be advisable to sink a Norton tube-well, and take the supply from a deeper level.

In camp men should never be allowed to lie on the ground at night. Often a few inches will suffice as a protection against an attack. In the Ashanti campaign, the greatest benefit was found to result from the raised bunks used by the men on which they spread their waterproof sheets. In all the huts used in that campaign provision was made that the men should be well raised off the ground; at each side of a centre passage in the hut, and running its whole length, beds were placed, made elastic by using split bamboos. The floors were made of rammed clay, and raised one foot from the ground. It is advisable to close up the huts and tents as far as practicable after sunset to shut off malaria.

The use of quinine as a prophylactic is a subject on which considerable diversity of opinion exists among medical officers. It was formerly believed that the daily use of quinine in from three- to five-grain doses will avert an attack in those exposed to malaria. The general opinion is certainly in favour of giving it, but its action appears to be temporary only, and its effect does not last after the cessation of the drug. Possibly it is on this account that the prophylactic use of quinine has been considered a failure. Sir A. D. Home, V.C., gives as the result of his experience that 'it may be desirable to state the conclusion come to with respect to it from observation. With regret, and heartily wishing that my opinion may be overthrown by those of others, I have to say that I did not recognise any value in quinine given prophylactically; it neither seemed to ward off attack or to mitigate the severity of malarious fevers in those attacked.'²

The experience of the American War³ appears to teach that when men are temporarily exposed in a specially dangerous locality, quinine should be issued as a prophylactic; but where the station must be occupied for some time, prevention should rather be based on the proper selection of sites and the avoidance of predisposing causes.

Arsenic has also been used with the same object. The writer's experience in India, which has been confirmed by other medical officers, is that for this purpose it is wholly useless, and most certainly does not ward off an attack.

With regard to acclimatisation, there is none. In those localities where the disease is endemic, the only acclimatisation possible would be a complete saturation of the system with the poison. Our endeavour should be that by careful selection of seasoned soldiers, and by maintaining them at the highest possible point of resistance to malarial influences, we may afford such pro-

¹ *Malaria and Injuries of the Spleen*, Calcutta, 1880.

² *Army Med. Dept. Reports*, vol. xv. p. 229.

³ *Medical and Surgical History*, 3rd part, p. 171.

tection for a time as will admit of their residence in those parts where they are required.

Heat-stroke.—This disease may be said to arise from two causes: (1) from the direct rays of the sun, and (2) heat in the shade, combined with stagnant and impure air. Next to temperature, the degree of moisture in the air has a decided influence on the occurrence of this disease. In India the largest number of cases occur from April to July, varying with the early or late setting-in of the rains; the period of most prevalence being greatest when the air is very hot and saturated with watery vapour. During this time the evaporation from the skin is reduced to the lowest point, while the body temperature may be increased either from climate, exhaustion, or fatigue.

If to high temperature and moisture is added stagnation of the air, the conditions liable to produce heat-stroke are increased. All medical officers have noticed the liability to heat-stroke during calm sultry weather, with high temperature and moisture.

Fatigue is an important factor in the causation of heat-stroke; the majority of cases in military service are the result of heat, over-exertion, and insufficient water-supply.

On the march in a tropical climate, the heat developed within the system must be dissipated by transpiration and respiration in order to preserve the normal temperature of the body. The soldier starts at a disadvantage: he is loaded with his kit and armament, so that in addition to the muscular fatigue and extra work done in carrying these, the result of exercise under such conditions is that he is bathed in perspiration, which actually causes a loss of water from the system, without a commensurate lowering of body-heat. If the atmosphere is moist, this further interferes with evaporation; or the want of water to replace that lost by excessive perspiration may cause the skin speedily to become dry. From this we see that the suppression of evaporation consequent on a deficient water-supply causes the temperature to rise and favours the occurrence of heat-stroke. Overcrowding in barracks and tents should be avoided. In overcrowded rooms not only is the air rendered foul, but the temperature is raised.

For the same reason on the march the order should be 'open order.' Nothing fatigues men more than keeping 'close order;' evaporation is checked, the temperature in the ranks goes up, and with it the body temperature. Without ventilation through the ranks the air becomes foul, owing to the giving-off of organic matter and watery vapour.

It is almost needless to add, after what has been already said, that unless under special circumstances, men should march in the early morning, and never during the middle of the day. If absolutely necessary, a night march may be made, but this should be the exception; the early morning hours give the greatest protection with the least fatigue. For reasons stated above, there should be ample provision made for a supply of cold tea, coffee, or water; the rule should be to take small quantities of these liquids at frequent intervals.

Regular and sufficient halts should be given to eliminate the element of fatigue, and advantage taken of any shade, provided it affords free movement of the air. In desert marches men should be cautioned against lying down while halting, as the temperature close to the ground is higher, and favours the occurrence of the disease.

The dress should be light, warm to prevent chills, and loose and open so as to avoid any constriction of the neck or chest. Woollen clothing is best adapted for marching. The colour should be of a light grey or khaki

colour. These colours have slow conducting power, and at the same time the least absorbing power to the sun's rays.

The head-dress must afford ample protection to the temples and back of the neck. It should be light and well-fitting. The ordinary helmet, as now issued to the troops in India, appears to answer all requirements in this respect, but if it had a cover to tie on and fall over the back part so as to protect the nape of the neck, it would be a great advantage. This should be lined with cotton and quilted. The back of the jacket may also have a padded lining over that part which covers the spine.

Severe headache may be induced by glare, caused by the direct or reflected rays of the sun impinging on the optic nerve; this may be regarded really as only a premonitory stage of heat-stroke, and can be prevented by the use of neutral-tinted or coloured glasses, which afford a grateful feeling of coolness and comfort, only to be experienced to be appreciated.

The food issued to the men must be in proportion to the work to be performed, but care should be taken that where the conditions favouring heat-stroke are present there should be no excess of animal food. Alcohol, under no circumstances, should be allowed, as it favours the suppression of the action of the skin and increases the predisposition to heat-stroke. Those who are intemperate are especially liable to be attacked. In no disease is the evidence more clear or more conclusive that alcohol not only destroys all immunity to heat-stroke, but largely increases the liability to attacks. Hence any issue of alcohol should be positively forbidden. There should be no exception to this rule. Tea and coffee should be supplied as far as possible. Tea is not only refreshing, but it also promotes the action of the skin, and thus aids in lessening the susceptibility to heat-stroke. Tea and coffee as beverages are therefore indicated, and should be freely partaken of.

In camps and barracks it is necessary to avoid exposure to the direct rays of the sun as much as possible, and at the same time to give ample ventilation, so as to keep the air pure and wholesome. If the temperature is very high, artificial means may be taken to cool the air; but this is not always easy to accomplish in the hot, sultry days when heat-stroke is most frequently prevalent. During such time, artificial methods for keeping up a continual movement of air by means of punkahs should be resorted to.

On the march, or frequently in cantonments, the early signs of an attack should be looked for. The medical officer will often be able to tell by such premonitory symptoms as stumbling, uncertain gait, and total absence of perspiration, of an impending attack; and, recognising these, he can at once pick out the men and afford timely aid.

Nor must it be forgotten that here again the seasoned soldiers have the advantage. New arrivals in India and the tropics generally are very liable to be attacked. This may partly be due to a disregard of ordinary precautions necessary in a hot climate, especially in avoiding exposure to the sun; but this will not account for the enormous difference in the numbers of new arrivals attacked, and leaves no other conclusion but that residence in a tropical country affords a certain limited immunity to this disease.

Dysentery and Diarrhœa.—Bowel diseases are one of the chief causes for admission to hospital in tropical and sub-tropical climates. Almost every soldier is affected with diarrhœa soon after his arrival in the tropics, which quickly passes off, in the large majority of cases, as he adapts himself to the new conditions of life incidental to the change.

Nor is this condition confined to the tropics. Frequently it has been noticed how new-comers in a place suffer more often from diarrhœa than the permanent residents. This, in some cases, has been traced to the use of

impure water, to which those accustomed to it had become tolerant, but to which new arrivals were susceptible, and diarrhoea followed its use. In India and the tropics generally these diseases assume a much more grave and severe form when they attack those whose constitution has been weakened by the debilitating effects of long residence in a tropical climate; and this very condition predisposes to an attack, so that there is no immunity gained by residence or acclimatisation in the tropics.

Diarrhoea and dysentery in the tropics are intimately connected with malarial fever, and by some it has been considered as the cause of these diseases.¹ Whether this be so or not, there can be no question that malaria, by enfeebling the individual, increases the disposition to attacks from these affections, and in many cases aggravates them when they do occur.

The large amount of sickness and mortality caused by these diseases on active service in all countries renders it imperative that preventive measures should be taken at the outset. Sites for camps should be selected on as dry soils as possible. Damp ground should be avoided, as this favours malaria, which in its turn predisposes to bowel affections. Dense forests, overhung by mists, where vegetation is active and decay of vegetable matter abundant, are always unhealthy. In the tropics old camping-grounds are dangerous to occupy; they are frequently found to be saturated with the excreta of men and animals: if necessity compels their use, they should only be occupied for as short a time as possible, and in any case, if these diseases are prevalent, the site should be changed at regular intervals. This not only provides for removal from an infected soil, but also necessitates a fresh water-supply in most cases, an important factor in the spread of these diseases.

The effectual and careful removal of all refuse to a distance, and its prompt disposal by burning or deep burial, should not be overlooked. In the latter case, lime should be freely used beforehand.

The latrines demand most careful attention. Those used by dysenteric patients should be separate from the others, and the excreta carefully disinfected.

It is also advisable to change the site when it has been in use a short time, filling up the old latrine, and ramming the earth well down. The fact that dysentery can be spread by the healthy and sick using the same trenches, and that foul trenches can give rise to sporadic cases, cannot be too strongly insisted on.

There should be no overcrowding. Density of population favours these diseases, and increases the danger from them, owing to surface defilement and the difficulty of keeping the camp clean and dry. Everything that appears likely to cause putrefaction should be at once removed.

A pure water-supply must be provided, and where this cannot be had the water should be filtered and boiled. Stagnant waters are particularly dangerous; they generally contain a large amount of vegetable organic matter undergoing putrefactive changes. In India an excess of mineral constituents in the water, and finely-suspended mineral matters, have been known to produce diarrhoea.

Food is frequently a cause of bowel affections. In former years the issue of salt meat, not only to men on active service in the field but during peace, was a frequent cause of bowel affections. The thirst produced by this diet led to intemperance on the part of the men, and this again predisposed them to these diseases.

Tinned provisions have in some instances produced the diarrhoea, prob-

¹ *Med. and Surg. History of the War of the Rebellion*, part iii. p. 621.

ably from the tinning being badly done, or from partial decomposition of the meat. In the tropics, also, it has been noted that an excess of animal food will produce diarrhoea.

Bad cooking is not an infrequent cause. The meat may be soddened and hard by being too closely packed in the cooking-vessels. The surface of the meat is overdone; the inside almost raw. This condition induces dyspepsia, which is very liable to cause diarrhoea.

Compressed vegetables are, as a rule, difficult for the soldier to cook; they are hard and indigestible unless previously softened by soaking in water, a process not always easy or available in camp. It is important that men should not go too long without food, and while on guard at night hot coffee should be provided.

When these diseases are prevalent, no fatigues or parades should take place in the early morning, nor should men be allowed to march before they have had a light breakfast of coffee or cocoa and biscuit.

When there are great undulations of temperature men should be cautioned against exposing themselves at night: they should especially avoid the chilling influences caused by dew, and where this is present they should always sleep under cover. Flannel belts are a great protection at night; they should be made of a double fold of flannel, and sufficiently deep to cover the whole abdomen.

It is almost unnecessary to refer to scorbutic dysentery. In former years it was the scourge of armies; it is now a disease almost of the past, and it will hardly be possible for it to recur again in civilised armies. With the present excellent methods for preserving fresh meat and vegetables, and the ease with which they can be packed and transported, it would be a standing disgrace if this should again appear to devastate armies.

Scurvy.—The want of fresh vegetable food, or some particular kind of it, has been shown by overwhelming proof to be the cause of this disease. The fact that a diet of fresh vegetables affords certain means of obviating an outbreak, as well as that it is the best remedy we have for the cure of this affection, establishes beyond doubt that scurvy follows a certain diet invariably.

All evidence proves that it is the absence of those salts whose acids form carbonates, and which neutralise the free acid in the system, that is the essential cause of scurvy. There appears now to be little doubt that these salts are required to keep up the necessary alkalinity of the system.

Overcrowding and impure air have been thought to be important factors in the causation of this disease, and that these predispose to an attack by lowering the general health and nutrition of the body there can be no doubt. Foul air is a powerful predisposing cause for this as well as for other diseases, as it renders the body less liable to withstand them.

As regards food, the chief points to be attended to are to provide an ample ration in proportion to the work which has to be done, and to let this consist of fresh meat and fresh vegetables for as long a time as possible. When preserved meat is issued with preserved vegetables, additional anti-scorbutics should be added to the diet, in the shape of vinegar, pickles, &c., and it would be advisable to supplement this with a bi-weekly issue of one ounce of lime-juice. Compressed vegetables have little anti-scorbutic value. During the process of preparation the salts have been squeezed out of them with the water. They should never be issued in place of fresh vegetables. They are difficult to cook, and not infrequently cause diarrhoea. Preserved peas and beans contain little or no anti-scorbutic properties, and should not be given in lieu of other preserved vegetables. Whenever issued, the quantity

should be sufficient to effect the object desired. Seven ounces of preserved potatoes are equivalent to about one ounce of lime-juice.

Vegetable acids—tartaric, citric, acetic—have been recommended as anti-scorbutics, but the results obtained from their use have been doubtful. Bitartrate of potassium is preferred by many, and has been given combined with vegetable acids. It was used during the Red River expedition as an anti-scorbutic. During the Nile expedition of 1882 jam was issued with the same object, and it is said with good results.

Amchur was provided for the native troops from India in the Egyptian expedition of 1882. It prevented the occurrence of the disease, and was considered a good anti-scorbutic. Amchur is made from the mango, which is dried. About one drachm is equal to one ounce of lime-juice.

The importance of red wines, so much used in Continental armies, should not be overlooked.

From the very time an army takes the field, attention to the state of the men as to scurvy demands careful watching, making the recognition of it an expected thing, one to be specially ascertained at the weekly medical inspections of the medical officers with the regiment, as well as to be held in remembrance at the hospitals to which men with any other kind of illness are sent.

Military Ophthalmia.—Under this term are included grey and vesicular granulations, and purulent ophthalmia; these diseases were formerly prevalent in the Army, and from their extreme contagiousness frequently spread in an epidemic form.

The cause was generally owing to mechanical irritation which produced a discharge of a contagious character; and this under the influences of bad sanitation, such as overcrowding, impure air, and no proper means for ablution, gave rise to a most severe epidemic disease, often ending in the total destruction of the organ.

Inflammation of the conjunctiva, spreading thence to the deeper structures, and often ending in suppuration of the eye-ball, may have its origin in the simplest of all ways. A soldier sleeps under a tent, in a tropical or sub-tropical climate, the sides of which are raised for the purposes of ventilation; the cold wind at night blows over his face and he awakens with swollen eyelids; a number of similarly affected men are crowded together; the simple inflammation becomes one having the virulent character of a specific inflammation of a mucous membrane, and infection may be conveyed in a crowded tent or room, as well as conveyed by other contact. Once the disease, then, is started, any form of overcrowding will intensify it and may in fact produce it. Dr. Welsh¹ states that in Malta he traced the causes in most instances to living in an atmosphere vitiated by the excreta of the breath, skin, and body, aggravated by a warm atmosphere, which had a decided influence. Since the ventilation of barracks has been improved, and the cubic space per man increased; there has been a great diminution in the number of cases admitted, showing evidence of a negative character of the influence of impure air. The same results have been brought about in the Hanoverian Army, where good ventilation has largely reduced the number of cases.

The presence of pus cells in the air of wards occupied by persons suffering from acute eye disease shows how readily this may be transferred from the sick to the healthy, and the infection by means of air has been the most frequent way by which it is propagated, the dried particles of the secretions floating in the air being more infectious than the moist.

¹ *Army Med. Depart. Reports*, vol. xi. 1869.

These are known agents in the dissemination of the virus: any one who has seen the swarms of flies which attack those suffering from diseases of the eye in Egypt can easily learn this, and should seek to obviate this source of infection as far as possible.

Once the disease appears, the most careful attention to ablution arrangements are necessary, and it should be borne in mind that the stages between simple catarrh and purulent ophthalmia are so ill-defined that it is next to impossible to say where one ends and the other commences. The use of the same towels and basins and an insufficient water-supply are the principal means by which the disease is spread, and their use may prove a danger not only to others, but to the man himself by re-inoculation.

Where basins are limited in number, men should be taught not only to throw away the dirty water, but to refill the basin with water, which the next comer should run off before filling the basin for his own use. Towels should be frequently washed in *boiling* water, to which some disinfectant may be added, and then dried in the open air.

To summarise. The chief preventive measures against military ophthalmia in those countries where it is most prevalent are ample air-space and good ventilation, whether in barracks or tents. The importance of pure air cannot be overrated; protection to the eyes from glare and dust by glasses when necessary; perfect cleanliness of the person, and frequent bathing of the eyes in pure cold water; care in the use of basins and towels; avoidance of cold draughts of air at night, and especially exposure to cold winds during sleep; isolation of the sick, who should be treated under the freest ventilation, and have their bedding, towels, &c., carefully disinfected. In addition, there should be close attention to the sanitary surroundings, and a liberal diet provided to maintain a good standard of health.

When this disease is present among the civil population, there should be daily inspection of the men by a medical officer, in order to detect the first case that may appear among the men under his charge.

Venereal Diseases.—This class of disease causes a large amount of inefficiency in the Army. It is impossible to compare the numbers who are affected in the military and naval services with those in the civil community, as there are no morbidity statistics relating to the latter. We have, however, no reason to believe that the civil population is exempt from these diseases in a greater degree than the military and naval services are.

The large amount of inefficiency in the Army and Navy caused by venereal disease was brought prominently to notice shortly after the systematic issue of the *Army Medical Department Reports* in 1859.

In 1864 the first Act of Parliament was passed which had for its object the prevention of these diseases, and subjected those females who were the chief sources of its diffusion to medical treatment in hospital while they were in a state capable of communicating it. The first Act passed was found to be more or less ineffectual, and this was further amended by the Acts of 1866 and 1869, which remained in force until May 1888, when compulsory examination was abolished by resolution of the House of Commons, at which time the Acts practically ceased. They were finally repealed in 1886.

These Acts have given rise to much controversy, and there has been a widespread and active opposition to them. Apart, however, from the saving of much suffering and misery, which they were the direct means of giving rise to, they were no doubt useful as affording a wholesome influence on the class subjected to them, and were the means of raising the moral tone of those towns which were placed under them. It is not likely that these Acts

will ever be passed in the same form again, but it is not unreasonable to hope that the time will come when venereal diseases will be included in the list of those of which notification is required, and that men and women suffering from a dangerous disease should be placed under some supervision and control.

It cannot be denied that since the abolition of the Acts there has been a progressive diminution in the admissions from these diseases in the Army at home, and this has been used as an argument against their reinforcement by those who are opposed to any legislation on the subject. But that this occurred in former years before the introduction of the Acts must also be admitted, although the reason for this being the case is not apparent. The following table shows the admissions for venereal diseases for five years before the introduction of the Acts :—

Year	Ratio per 1,000
1859	422.0
1860	368.9
1861	353.8
1862	329.9
1863	306.8

A similar diminution in the admissions for all venereal diseases during the quinquennial period 1885-89 has also taken place, as shown in the following table :—

Year	Admissions for	
	Primary syphilis	All venereal disease
1885	127.4	275.4
1886	118.8	261.1
1887	107.5	252.9
1888	98.2	224.5
1889	88.5	212.1

There is some evidence that during later years a diminution has occurred among the civil population, and the death-rate during the last quinquennial period, 1885-89, shows a large decrease over previous years. The following table is taken from the Registrar-General's returns for England and Wales :—

Years	Deaths per 1,000,000
1865-69	80
1870-74	80
1875-79	85
1880-84	84
1885-89	73

There is no doubt that, as shown by the above tables, there has been a decline in the prevalence of these diseases since 1885.

The last twenty years has seen many changes in this country. Since the passing of the Education Acts, when school attendance was made compulsory, there has been a gradual but increasing improvement in the moral character of the whole population, and the various sanitary Acts have in no small degree assisted to bring this about. It is evident also that the Contagious Diseases Acts have of themselves been beneficial, inasmuch as they proved to individuals the necessity for medical treatment when diseased, and inculcated the benefits to be gained from cleanly habits. It is seldom that the same abandoned class of prostitutes are to be seen in garrison towns that formerly frequented them, and there is every reason to believe that this is due to the general social improvement of the masses, and not to

the abolition of the Acts, which served their time and purpose, and were in no small measure instrumental in bringing about this change.

On the contrary, we are justified in believing that this diminution in venereal disease would have been greater had the Acts remained in force and their sphere of usefulness been more widely extended. The advantages gained by the passing of these Acts at a time when education was neglected and few sanitary Acts dealing with the surroundings of the working classes were in existence is shown in the following table, which gives the admission ratio per 1,000 in fourteen stations under the Acts, and fourteen stations not under the Acts, since 1860.

Year	14 Stations under the Acts ¹	14 Stations not under the Acts ²
1860-63	129.8	120.6
1864-69	87.1	133.9
1870-73	86.0	107.9
1874-79	88.7	97.4
1880-83	75.6	175.9
1883-84	123.9	174.0

In India, where the same rapid advance in sanitary progress has not been possible, and where the education of the natives has been narrowed by caste prejudices, and especially of the female population, which has always been relegated to an inferior position in that country, there have not been the same influences at work; and consequently we find that the suspension of the Acts has had a directly opposite effect to what has taken place in this country.

During later years the proportion of young men serving in the Army in India is larger. In the ten years 1871-80 the proportion under 25 years of age was 62 per cent.; in the five years 1884-88 it was 74 per cent., while the proportion of married men has sunk from 10.37 in 1876 to 8.84 in 1888. The shorter term of Indian service under the present system of enlistment may also have contributed to the increase in the numbers affected.

But whatever be the cause, the facts are indisputable. Lord Roberts³ has shown by a comparison between the years 1883, when the Acts were in full force, and 1888, when they were suspended, how large the increase of venereal disease has been in the Bengal Army. At no station in 1883 did the number of venereal cases of sick in hospital reach 80 per cent.; at only two stations was it over 25 per cent., while in some stations it was under 12 per cent. In June 1888, at 18 stations the percentage was more than

¹ 14 Stations which came under Acts:—

Devonport and Plymouth
 Portsmouth
 Chatham and Sheerness
 Woolwich
 Aldershot
 Windsor
 Shorncliffe
 Colchester
 Winchester
 Dover
 Canterbury
 Maidstone
 Cork
 Curragh

² 14 Stations never under Acts:—

Isle of Wight
 London
 Warley
 Hounslow
 Pembroke Dock
 Sheffield
 Manchester
 Preston
 Edinburgh
 Fermoy
 Limerick
 Athlone
 Dublin
 Belfast

³ Parliamentary Paper, No. 220, June 13, 1888.

50; at a great many stations it was just under 50, and at the majority of the remainder it was 80 and more.

In 1889 venereal disease was directly the cause of 82·1 per cent. of the admissions to hospital, and 8·4 per cent. of the invaliding in the whole of the Army in India. Taking the three Presidencies together, the admissions from venereal diseases in the ten years 1870-79 were in the ratio of 208 per 1,000; in 1888 they were 371 per 1,000, and in 1889 reached 481 per 1,000. In 1884, when the Contagious Diseases Act was in force, the admissions were 294 per 1,000. The Act was suspended on January 1, 1885, since which date there has been a progressive increase in the admissions for these diseases until 1889, when they attained the proportion noted above.

The following table shows the comparison between the years 1866, before the Contagious Diseases Act was imposed, 1884, while they were in force, and 1889, after they were abrogated:—

Presidency	Average strength	1866. Ratio per 1,000 of average annual strength	1884. Ratio per 1,000 of average annual strength	1889. Ratio per 1,000 of average annual strength
Bengal	42,821	217·7	290·6	491·2
Madras	13,619	231·1	307·7	451·6
Bombay	12,826	—	291·6	481·1

The history of the subject in Calcutta¹ is even more convincing. The Acts were put in force in that city in 1869; they were suspended in part of the city in November 1881, and in the entire city in March 1883. The following figures show the results of this change:—

Cases of Venereal Diseases.—Ratio per Cent. of Garrison.

Year	Primary syphilis	Venereal of all kinds
1868	10·0	25·06
1869	9·0	25·08
1870	6·0	14·4
1871	2·7	8·1 New regiment
1872	5·7	13·9
1873	1·4	7·4
1874	1·4	9·4 New regiment
1875	1·3	10·3 „ „
1876	2·3	12·6 „ „
1877	4·3	10·7
1878	4·0	11·7 New regiment and drafts
1879	2·7	9·3
1880	1·7	12·8 New regiment
1881	8·1	8·7
1882	8·7	14·5 New regiment
1883	10·9	28·0
1884	30·2	58·1 New regiment
1885	15·1	31·6 „ „

From these figures it is apparent that syphilitic disease had sunk from a high ratio, 10 per cent. in 1869, to a low one, 1·7 per cent. in 1880, and for two years it had only been 1·4. In 1883 it rose to 11 per cent., and in 1884 to 30 per cent., while 58 per cent. of the garrison were admitted to hospital for one form or other of venereal disease.

¹ Blue Book, East India (Contagious Diseases), 1888, p. 64.

DISPOSAL OF THE DEAD

I.

BY

SIR T. SPENCER WELLS, BART., F.R.C.S.

II.

BY

FREDERICK WALTER LOWNDES

SURGEON TO THE LIVERPOOL POLICE

DISPOSAL OF THE DEAD

I

WHAT shall be done with a man's body after death? is a question which rather concerns those who come after him than himself. As individuals, we seldom think about the matter. Collectively, we are apt to fall into the same indifference. The world at large leaves the consideration of it to executors, priests, and undertakers. In this respect sanitarians, as a class, are much like other men. Due attention, ample discussion, and endless writing have been given to the most heterogeneous subjects, from the equipment of armies to the bottles of babies, from the draining of palaces to the analysis of pickles, from the diseases of artisans to the games of schoolboys; and having succeeded in the nineteenth century in accomplishing what the Romans did in the infancy of their republic, we have complacently folded our hands, while the evils, which they only shifted, grow again and become as threatening as ever. The fact that in the standard work of Parkes 'the disposal of the dead' occupies only four pages, out of a total of 470, is enough to show demonstratively the proportion of consideration which this branch of sanitary science has attracted.

It is, however, a subject which the increase of population, the demand for living space and bread production, together with the results of bacterial investigation, must force into a conspicuous place on the list of sanitary corrigenda. The apathy of the public must be aroused by scientific activity, and a solution found which, without violating sentiment, will give security to life and health.

All organised beings are in a state of perpetual transformation. It has been so from the beginning, and will go on so to the end. The atoms only, of which they are composed, are unchangeable. They alone never lose their identity, and as there is neither annihilation nor new creation of them, each in turn 'plays many parts.' Those atoms which make up the existing generation have been part and parcel of the generations that have gone before, and will, in future, take their places in similar evolutions of formation and destruction. One generation is not only the successor of another, but is as well its material inheritor.

The compounds of which the various structures consist are not at any two successive moments the same. They are always in a state of demolition and repair. The processes of synthesis and analysis go on simultaneously, so long as life continues. One atom is removed, another substituted, and the substance remains. When that principle which animates a living thing declines or ceases, destruction begins. If it decline only, there is deterioration. When it ceases, as is the inevitable end, accumulation stops, and the material that is becomes subject to the ordinary laws of matter. Chemical reaction prevails, bacterial and cryptogamic agency precipitates the changes, and the atomic particles, set free to enter into new combinations, wait their turn to be appropriated by a fresh series of living beings.

From age to age the earth is covered with fresh vegetation, and peopled with ephemeral creatures. Each generation has the same destiny. All have the same needs, develop the same propensities, and fulfil the functions

assigned to them in devouring each other, or being devoured, and thus preparing the materials for the organisation of that which is to replace them. When they fall short of that purpose, chemical affinities supply the want and complete the process. Life alone, whatever that may be, is never interrupted. Passing from one incorporation to another, its mode of manifestation only varies. The many forms of vegetable life absorb the exhalations and exuvia of animals. They flourish, and in time become the sustenance of other beings, or perish by accident. Sometimes the species of a rival kingdom browse upon their tender parts, sometimes kindred fungi consume their harder structures, sometimes ravaging fire dissipates their lighter elements upon the winds of heaven, and leaves the black carbon on the earth in readiness for other changes. By some means or other, and at some time or other, all vanish. Even the cedars of Lebanon are not eternal, and petrifications are but briefly monumental. In their turn the elephant that has stripped the trees of the forest, and the ox that has ruminated on the herbs of the prairie, fall the prey of the wild beast, and pass through the alembic of his system into the form of appropriable matter. The mighty frame of the tiger succumbs, as readily as the softest mollusc, to the irresistible transmuting power of the invisible bacteridium. Man himself, in his omnivorous supremacy, is no exception to the universal rule. In his material aspect he does as the rest of creation does. He lives to eat and destroy, as much as he eats to live. In life he is tenant at will of a variable proportion of organisable matter; in death he ought to render it back into the common stock for the use of others, without in any way barring the succession. If he interfere artificially with the natural course of events among the elements of his mortal covering, either retarding development or impeding dispersion, it is an act of vicious perversity. It is his privilege, if he choose to use it, to improve the one and to accelerate the other. We shall see how he falls short of this. Civilised he is synthetically but a bungler; analytically, his interference has been almost criminal.

SURVIVAL OF OLD BURIAL CUSTOMS

Given corresponding circumstances of savage life, man is much the same at the present day that he was in the most primitive times. According to climate and situation he seeks his means of existence from the trees, herbs, and fruits that grow about him, by the chase and by fishing. He constructs his habitation in the same simple fashion. He has the same qualities and the same defects, and his social life is filled only with the most material interests. He dies with the vaguest hopes of something better than his present experience, and his carcase meets with little more consideration from his fellows than any other mass of putridity. Respect for mortal remains is not a characteristic. Indifferent about the presence of a recent corpse, he spurns it when it becomes offensive. Without sentiment, he relieves his feeling of embarrassment by getting rid of an encumbrance. The ways and means of doing this are determined by his surroundings. From this circumstance we may trace a curious series of survivals of usages in the dealing with the dead almost identical throughout all known time.

The Syrcanians abandoned their dead to wild dogs; the Kamschatdales keep special dogs for devouring their dead. Some Kaffir tribes remove the dead to spots in the bush in order that they may be eaten by animals, following in this the practice of some of the ancient Asiatics. Among the Scythians the dead body was sewn up in skins of animals and suspended from branches of trees. Some of the North American tribes have a similar custom. They envelop the corpse in a buffalo hide and place it on a plat-

form, constructed upon the top of tall poles. The natives of some parts of Australia cover over the body with leaves and grapes, and either lift it into the fork of a tree, and lash it to the boughs, or elevate it upon poles. The Parsees, as far back as there are any traces of their habits, exposed their dead upon high gratings to be eaten by vultures. The natives of some parts of India, to this day, carry the body to the top of a hill and place it upon a stone slab, returning to take it away when the bones are picked clean. The ancient inhabitants of the banks of the Persian Gulf threw the body into the sea. A similar custom holds still among some American aborigines, who lay the dead body in a canoe, and launch it on a stream or lake away from their dwellings. Burials and burnings were both practised in some form in the very earliest ages. The same thing happens among all the savage peoples discovered in recent times—in North America, Australia, and the islands of the Pacific.

Such burials among the purely animal man were but the expression of an instinct of savage nature, which, from fear of the inanimate mass, or from a desire to hide it, or to secure themselves from being haunted by it, or simply to rid themselves of a nuisance, led them to cover it with tree-branches, earth, or stones. The gorilla, which lives in small troops, one male with a few females and their offspring, does the same. He lays boughs or withered wood upon the carcase and leaves it. Wandering from place to place, and sheltering in caves without implements to build or trench the earth deeply, prehistoric man, whose dim unwritten story begins to shadow forth behind that of the first historic man, could do no more than the gorilla, and gave us the prototype of all subsequent interments.

CAVE BURIALS

Later on, when men had grouped together and become accustomed to live in tents, on the plains, at the seaside, or in their lacustrine constructions or fortified camps, the ancient cave homes were deserted and put to a new use. They were made to serve as depositories for the dead. In their natural state these caves would not hold great numbers or be serviceable for any long time. The massed bodies became insupportable pests. The wise man of the tribe, the priest-medicine-man, took charge of the rites of sepulture. In order to lessen the stench and allow of entrance for some new burial, he lighted large fires before the mouth of the cave, throwing in brands and partially burning what was therein. Here we get the origin of cremation as a means of sanitary purification.

SEPULTURE IN THE STONE AGE

Tomb caves are comparatively rare. Several have been found in Poland. Those known in France number between seventy and eighty. The most interesting are some in the department of the Lozère, carefully examined by Dr. Prunières. That at St. Pierre-des-Tripis had its opening closed by a pile of loose stones. A thick layer of ashes and burnt wood was lying on the platform before the entrance, and there it ceased abruptly. The relics of more than three hundred bodies were gathered up, some of the bones showing traces of the action of fire. They had been thrown confusedly towards the upper end of the chamber. The skulls only were ranged along the sides, and some of them had flint arrow-points still fixed in the bone. Cinders and charcoal were mixed up with the human remains. It was here that Dr. Prunières first saw skulls that had been trephined, with the holes cicatrised.

In those parts of France and of other countries where, from the geological

conformation, there were no natural caves, subterranean excavations were used both for sepulture and habitation. In Africa this practice lasted even after metals were employed; and in the Canaries the people sometimes used the artificial caves for dwellings, while they buried their dead in those that they found in the rocks.

One hundred and twenty of these caves have been discovered in the department of the Maine, distributed in groups. They are for the most part narrow passages, cut out of the soft chalk at the sides of a valley, with a larger cleared space before the mouth of the crypt. One individual has examined more than two hundred bodies taken from them. They were not covered up, but had been laid upon the soil or upon flat stones, which show that they had been exposed to great heat. Calcined bones and ashes, mixed together, were sometimes seen lying by the side of a skeleton that had not been disturbed. Small cutting flints had been thrust into some of the lumbar vertebræ. It is supposed that these must have been the victims of human sacrifice at the death of great personages. Imperfect gravings of the female form on the side walls, apparently intended to represent some divinity, are among the most interesting disclosures of these caves. In one cave near Montereau, which contained more than two hundred skeletons, it was evident that the bones of one generation had been removed to the upper end to make room for other bodies.

Near Arles the excavations took the form of a long oval trench, wider at the bottom than the top, which was closed in with flat stones and earth. The sides were carefully worked, and the largest diameter was as much as forty-five yards.

In other places, and, no doubt, at a later period, the caves, of a hemispherical form, with a narrow neck-like opening, were sunk in the chalky soil. In one of these bottle-shaped holes there were some forty male and female skeletons, with remains of children, all arranged in a sitting posture on the floor, with the faces turned towards the south. Similar well-shaped tombs have been met with in Algeria and Moravia.

The only known instance of a built-up tomb of this period is that at Mont Vaudois. It is a sort of hollow wall, in the thickness of which twenty skeletons were found. A few of them were in rough stone cases, with specimens of bone tools, polished hatchets, and black pottery without ornament; the structure had been gradually enlarged as room was wanted for fresh bodies. Fragments of coal and parts of animals were mixed up with the human remains. One skeleton—that of a young girl—showed marks of burning.

The caverns in the Devonian, Carboniferous, and Magnesian limestone of England have yielded abundant relics of prehistoric fauna, associated with traces of Palæolithic man. Sir Richard Owen has enumerated thirty-seven species of mammalian found in them, of which eighteen appear to be extinct. In the later stone age the only form which survived (and that is now extinct) was the Irish elk. There are in these caves the bones of some wild beasts at present retired to other countries, and with them those of domestic forms which were not part of the indigenous fauna of Europe. The appearance of the remains of the dog, sheep, goat, horse, and other well-known animals furnishes us with the inference that they must have been introduced by tribes migrating from Central Asia into Europe. The occurrence of rude flint instruments in the lower deposits on the floor of these caves, and of a more finished kind in the overlying deposits, demonstrates that they were continuously tenanted by an improved race of man. But in England human remains of this period are very rare.

In Switzerland and Northern Europe, though the midden-heaps and lake

beds reveal an enormous quantity of material once in domestic use, it is the same as with England. Of Neolithic man himself there is but little left, or, perhaps, research has not been carried out with the same perseverance. Prehistoric deposits are essentially the same on both sides of the Atlantic. But the younger alluvia of the American river valleys and lakes, though they no doubt claim a high antiquity, have not supplied the same copious evidence of early man, which gives so much interest to the corresponding European formations.

So man, in his original state, did as other animals do. He deserted his dead companions, and left their remains to be disposed of by a beneficent Nature. When influenced by the most elementary ideas of social life, and possessed of the rudest contrivances of self-help, he took advantage of the means at his command of hiding away his dead, or of cutting out hiding-places for them, sometimes using fire to complete his work. But the contents of these caves and wells do not account for the whole of the population. No doubt we see in them only an expansion of the primary idea of interment applied to leading families. As to the insignificant multitude, we can only suppose them to have had the common lot of a shallow hole and a few handfuls of soil. The earth has left no trace of their mortality.

Certainly, in the second part of the Stone Age the most common mode of sepulture was that of dolmens, or, where the stone necessary for such monuments was not to be had, it was in barrows. So extended was this custom that they are spread over more than one continent, and may be seen along the shores from the Baltic to Morocco; also in Asia and in South America. Two thousand three hundred of them have been counted in France alone. Their use lasted in England and France after the introduction of bronze, and in Africa after that of iron.

The simplest form of dolmen was that of three or four upright slabs of stone, fixed in the ground, and supporting another block upon their upper ends. This group of stones formed a cavity just large enough to contain a human body in a sitting position or doubled upon itself. The whole was loaded over with earth and sods, so as to raise up a hillock, which has generally been washed or worn away. Such one-called dolmens admitted of being greatly enlarged. Constructed in the same manner, and of the same materials, some have a measurement of 200 yards in circumference. Others were elongated, divided into compartments, and ended in a spacious chamber, covered in with an enormous block of granite.

These funereal mounds are common in England. Composed only of stones, they are called cairns. Barrows are earth mounds piled up over a stone chamber, with or without a covered passage leading to it. The round or oval barrows are of a date as late as that of the use of metal implements. Peru and Denmark have dolmens with the same characters as those of France and England. Italy has only one group of them, at Salurnia. They abound in the Crimea and Palestine.

Till recently it was unknown what was done with their dead by the Swiss lake dwellers. Dr. Gross has, however, found on the borders of the lake of Neuchâtel a large construction of the dolmen type, made up of great stones, and comprising several chambers. About twenty bodies had been buried in them, in the usual sitting posture. The skulls resembled exactly those known to have belonged to inhabitants of the lake houses. All the indications are that it was a production of the period of transition from the age of polished stone to that of bronze.

Everywhere the body has been arranged in the sitting or bent posture, with the feet touching the buttocks, the thighs pressing the arms against

the belly and chest, and the head resting upon the knees. It is generally found to have been placed upon a layer of flints spread over the floor of the chamber, and showing that they have been greatly heated. There was, however, no sign of cremation. The fires seem to have been lighted for the purposes of fumigation. Some of the compartments were only large enough for one body, but in such case the remains of the first or second occupant have been displaced to make room for a successor. When this has happened, the bones of the last body only are in the orthodox position: the rest are scattered about without any attention to order.

SEPULTURE IN THE AGE OF BRONZE

It is estimated that it was about four thousand years before our era when what is called the Age of Bronze, with its advance of civilisation, superseded that of polished stone. The metal made its appearance in Europe with some Aryan immigrants from the East. During its early stages the inhabitants kept up their custom of burying in dolmens and barrows. But either from imitation or from hygienic necessity, fire was brought into more general use as a purificator. In the chamber of some tombs the skeleton has been found calcined, and lying on a bed of cinders. The body has been burnt on the spot. In others the ashes have been collected and shut up in receptacles cut out of stone, or in vessels of the pottery characteristic of the period. Every now and then the ashes were seen heaped on one side of the compartment, and covered with gravel.

The Bronze Age began later in Denmark than elsewhere, and lasted longer. The stone chambers of some of the dolmens have been reduced to the size of large coffins, and the bones in them were resting on a bed of burnt flints. It was at this time that the trunks of oak trees, hollowed out, began to be used as coffins. In those examined the human remains had almost disappeared, but the vestments had not been destroyed. The bodies had been buried in the dresses which they were in the habit of wearing. The legs had on bandages instead of trousers, and there was a sort of woollen skirt, mantle, and cap. Besides this clothing the coffins contained bronze swords in wooden sheaths, knives in bronze, wooden cups, and in one instance an arrow point of flint. Skins, mostly of the ox, were occasionally stretched out at one end of the chamber. In the course of time these burials were given up, and the bodies were burnt. As had been done with the body, so it was with the bony residue. Part of the old usage held its own with the people, and the ashes were wrapped up in pieces of woollen stuff. This custom was abandoned in its turn, and urns took the place of cloth.

The divisions of the Age of Bronze into two periods was well marked by the transition from burial of the dead to the practice of burning. In Denmark tombs of this age are very numerous. Those showing the prevalence of burning are the most frequent. The same may be said of Norway, Sweden, and the tract of country now Germany. The process of burning did not make such quick progress in England, except in one of the northern districts. Rolleston says that in some of the barrows several bodies had been laid out in the long axis, upon stones, loaded with wood and grass. All were consumed together. The custom in Russia was to burn the bodies outside the tomb. Burial took place afterwards. Not far from the Lake of Lugano earthen vessels enclosing ashes, burnt bones, and bronze implements have been taken out of a cluster of eight cairns, each built up of six unwrought stones, four for the sides and two for the floor and roof. Similar tombs and contents have been discovered in other parts of Italy. The burning of bodies had become a general custom of this period. Yet there is no evidence as to

the valley of the Rhone. Barrows and dolmens are very rarely met with in that country; yet throughout the time in question it was thickly peopled. It is surmised that after incineration the ashes were thrown upon the water, as in India. Probably the same custom prevailed among the lake populations of Switzerland. We may in this way account for our ignorance of their mode of dealing with the dead.

SEPULTURE IN THE AGE OF IRON

With further appliances available for the purposes of every-day life, and with a step forward in the progress of civilisation, we meet with what some would now call a retrograde movement in regard to the disposal of the dead. Iron was known in Egypt under its earliest recognised dynasties, and was the secret of their success in the marvellous undertakings which now excite our surprise and admiration. About the fifteenth century before our era the priest-king Rameses II., the Sesostris of the Greeks, was engaged in repelling an attack upon his dominions by Etruscans and other enemies from the Mediterranean coasts. In the retreat of these aggressors they took back with them iron and the knowledge of its employment. From them it spread over Europe, passing from Italy and Greece to the country lying north of the Carnic Alps. It began to be common in Gaul about the eighth century before Christ, and several hundred years later in the north of Europe. In all these countries the practice of burning the dead lasted till what is called the Iron Age set in, and even then the custom was slow in losing its hold among the rich. But everywhere, as iron came into use, inhumation took its place besides burning, probably from the increased power in the hands of men using efficient tools and partly also as a matter of economy; for when wood was the only fuel its provision for the pile was always a costly and laborious business.

The second part of the Iron Age is distinctly characterised in Italy by the appearance of cemeteries. Several cemeteries in the neighbourhood of Bologna, the antiquity of which is defined by the objects preserved in them, afford evidence of a small proportion of simple inhumations compared with the incinerations. But the size of these plots of ground is in itself a proof of the purpose for which they were wanted. One at Marzabotto is 700 yards long and 800 wide, and contains examples of various modes of burial. There are quadrangular cases, built up in the earth, of stones adjusted without any intermediate substance. The remains in them have been burnt. In some excavated pits of a conical form, enlarging downwards and lined with stone, skeletons not burned have been buried in considerable numbers. Ashes are there, too, but no trace of combustion. In crypts of another form some of the bodies seem to have been partially burnt, while others were untouched by fire. Thirty tumuli upon the adjacent plain only furnished three cinerary urns, while simple burial in them was almost uniform.

At Certosa, where there are also Etruscan inscriptions, there were many more burials (either in the earth or in wooden cases) than burnings; and in all the tumuli—meaning by that term earth mounds without chambers or galleries—inhumation is the usual mode of sepulture. It is not difficult to follow out corresponding changes in other countries. Their cemeteries illustrate not only the condition of the people, but as well the universal sentiments, and sometimes the eccentricities, which find expression in the mode of dealing with the dead—honour for the brave, family affection, servility for the rich, contempt for the poor. There were always the ostentatious, who wished for distinction even in the tomb; and dissentients, who were not

satisfied with the 'existing state of things.' But all prove one point—that fire, ashes, and charcoal were looked upon as the most certain means of lessening the nauseousness of putrid emanations, and that there was no natural repugnance to the committing of a body to the flames.

Thus in the extensive burying-ground at Hallstadt, near Salzburg, out of a thousand interments, it was found that nearly half of the bodies had been burnt. The burning has been effected away from the tombs, and without removal of the things the people were in the habit of wearing. Signs of the action of fire were visible upon bracelets, still encircling the bones. Some of these remains were found in the earth, others had been placed upon flat stones, and a few were enclosed in unbaked vessels. Three bronze pots had been used for the same purpose. When the bodies had not been burnt they were laid in narrow pits, generally singly, and at a depth of rather more than three feet. The position varied. The most common was that on the back, with the arms at the sides, or crossed on the chest, or passing round the head. In some cases the body was lying on the side, the knees drawn up, and the head resting on the arm as in sleep. Only nine coffins could be recognised, but some of the bodies of a richer class were overlaid with stones, regularly disposed. The interments were for the most part single. In some graves, however, two skeletons have been disclosed, either enlacing each other with their arms, or bound together by a waistband of beaten bronze. Among the other tombs are some with as many as four skeletons. They are totally destitute of ornaments, arms, or utensils. Such must have been the common graves of the poor.

In Hanover, Belgium, and Denmark incineration was for a long time general. It gave way at last to burial, and oak-trunk coffins became the peculiarity of Danish grounds. In certain localities cinerary urns reappeared towards the end of this age. The Gaulish cemeteries prove that, at various times, and at various places, the practice was shifting, and subject to capricious alternations. Cemeteries by degrees grew into greater popularity than the tumuli. In both, ashes and whole skeletons have found a resting place together. The graves are very shallow, and contain one body, often with clothes and all accoutrements, but uncoffined. It was in a few of them that coins were first found.

In a tumulus at Meuley, in the Côte-d'Or, this significant fact was observed. A body with all the indications of this age, surrounded by seven unwrought stones, overlaid a burial of incineration, which had in it none other than bronze objects. And at Waldhausen, in Pomerania, a still more curious discovery has been made. The first skeleton met with in opening a tumulus was simply laid in the earth, with pottery and objects made of iron quite rusted. Beneath it were several cases of stone, each holding an urn filled with calcined bones and bronzes. Below all the searchers came upon blocks of rock covering human remains, flint hatchets, and the rudest kind of earthen vessels.

Thus, for some thousands of years, the same spot had been made use of for burial purposes by a series of generations of human beings, perhaps races some as far as possible from civilisation, and all, though making progress step by step, ignorant, and the mere creatures of instinct and impulse. Yet there had been no desecration. But, age after age, as additions were made to the perishing accumulation, each successor observed his own rites, acted after the fashion of his time, and respected what had been done so long before him in the same spirit and under the same exigency.

EGYPTIAN BURIAL CUSTOMS

Nothing at present is known to discredit the opinion that Egypt was the precursor of every other nation in the civilisation of its people. Whether we accept the longer or shorter estimate of the time at which the earliest of the Memphite dynasties were its rulers, we still are dealing with a period coeval with that of the ages of stone and bronze in Europe. There may have been a Flint Age in Egypt. Flint instruments have been found in Theban tombs and mummy cases, but the tombs and cases themselves, and the marks of high refinement in the other contents, prove that that age has long passed away. All traces of its first barbarous condition are lost, and in speaking of the most ancient monuments of Egypt we have to think of a country where knowledge was advanced, where the conveniences of life were ample, where religious ideas influenced man's action, and of which we have more definite information than can be gathered from the exploration of cairns, tumuli, and cemeteries. The history of its domestic, religious, and mortuary customs is plainly written on the walls of the mastaba and pyramids of the first empire, the subterranean tombs of Abydos, the funeral temples of its highest stage of development, and in the documents and paintings which they have yielded up for our investigation.

In many hot and dry climates the dead body, if exposed to sun and wind, dries up rapidly without putrefaction. This is especially the case in Egypt. There is here the clue to all the peculiarities of the art of the Egyptian embalmer. Without the Egyptian climate there would have been no embalming. The native Egyptian had more than a common horror of decay: it contravened all his notions of the destiny of his body. He prayed that he might not be eaten up by the earth. His earliest effort at preservation was by drying, in which Nature both prompted and aided him.

On the left bank of the Nile is the plain of Sakkârah, which stretches from Memphis to the edge of the desert. It has a substratum of tender limestone, upon which is a layer of sand, varying in depth from some yards over the hollows to a few feet only where the rock tends to rise to the surface. There for 400 years the great city never ceased to deposit its dead. It was the vastest cemetery ever known—from 2,000 to 8,000 yards wide, and extending along the banks of the river for five leagues. Some of the bodies were sunk in pits. How numerous they were is shown by the remains of the crowded mastabas built over them, which in course of time passed from one possessor to another, without even the precaution of erasing the superscription. The millions of poor who must have died during these long years were laid in the sand itself, with no other arrangement than a bed of charcoal and a straw mat as a wrapping. These bodies were simply mummified by desiccation. There was no great lapse of time, however, before priestly and other influences associated rites and ceremonies with this form of interment. New methods of seconding the work of the climate in securing the body from the effects of humidity were devised. All was organised in accordance with the national belief in the body being merely the habitation of an eternal something, which exhaled at the last breath, and assumed a form less material than the frame it had quitted; but representing, as a coloured shadow, the exact image of the individual, trait for trait—child if it had left a child, woman if it had left a woman, man if it had left a man. This shadowy existence has been given the name of the 'double.' It was supposed to have the same wants and passions as its original, and to be a continuation of its life, ordained at some future time, after a series of transformations in some unknown regions, to rejoin the body. If it were recognisable, it became all-

important to preserve this tabernacle from destruction till the appointed time. Such a conception of the associated life of the body and extruded soul explains the supposed necessity for all the elaborate and costly proceedings of embalmment, installation in a suitable lodging, supply of provisions, and offerings to propitiate the new powers for good or evil which it was imagined had been acquired in the invisible world.

The art of embalming was a long time in arriving at the perfection in which we find it in some of the mummies of the later empires. Perrot and Chipiez, in their work '*Histoire de l'Art dans l'Antiquité*,' relate that they once unrolled the body of a woman of distinction who had died in the time of one of the Rameses. When they had removed the light linen bandages and envelopes in which she was closely wrapped, the body lay before them almost in the same state as when it left the workrooms of the *taricheutes* of Memphis. The hair was black, and arranged in fine plaits. The teeth were perfect, and showed between the slightly retracted lips. The delicate nails of both feet and hands had been stained with henna. All the limbs were flexible and scarcely altered in form, the skin soft, firm, and adhering to the flesh. But for the odour of naphtha with which everything had been saturated, and the colour of tar or burnt paper which had spread everywhere, the form might have been contemplated with complete admiration. Passalacqua also states that he disinterred at Thebes a female mummy having her hair in perfect preservation, with all the plumpness and regularity of form of body and limbs, which proved her to have been one of the loveliest of her time. He mentions that the beauty, fine proportions, and sound condition of the body so delighted the Arabs that they took it up several times to show it to their wives and neighbours.

Such a state of conservation is rare, and only seen when the most costly and tedious methods of embalmment have been followed. In these first-class mummies an incision has generally been made in the left groin. The intestines were extracted by this opening. The body was then left, for from forty to fifty or seventy days, macerating in a strong solution of natron. Experiments have been made by Dr. Sacquet in France to ascertain what would be the effect of such steeping in the salts procured from Egypt. The result was that in this temperate climate the fluids of the body exuded were not evaporated, underwent decomposition, and at the end of seventeen days gave out an insupportable stench. But the substance itself was not putrefied. Contrary to all expectation, the natron was found intact, and the flesh of the extremities had become mummified. The dry climate of Egypt was wanting to complete the process. It is uncertain whether in the Egyptian mummies there was any desquamation of the epidermis. The fixedness of the hair and nails would indicate that in those carefully manipulated the surface was kept entire. The essential part of the process was the withdrawal of the fluid and oily parts from the body by the natron bath. The desiccation of the solid parts followed. The matters with which the tissues were impregnated were accessories. Their abundance and quality depended upon the rank and richness of the person. Some bodies, when drying, were filled up with a mixture of aromatic resins, and wound up in complicated bandages and cloths soaked in naphtha. The dexterity with which this was done often gave to the dried mass its natural form. These mummies are brittle and of an olive brown colour. They retain the hair and nails, and have the features recognisable, though the bones of the nose were more or less injured by the perforations made to extract the brain. Almost imperishable when kept from air and humidity, they answer as far as possible to the religious demand of durability.

Another class of bodies, with the flank incision, after being emptied of the viscera, were salted and filled or saturated with bitumen. The use of hot bitumen was especially necessary in low localities, such as the plain of Sakkarah and along the river side. Mummies of this description are dark-coloured, hard, and very difficult to take out of the enveloping cloths. When exposed to air they become covered with a slight efflorescence of sulphate of sodium.

In making an inferior class of mummy the entrails were drawn away by the fundament. Such bodies were either salted only, or salted and stuffed with bitumen, or dipped in a bath of bitumen. They are the mummies most commonly met with, are nearly black, have lost the hair and eyebrows, and most of the face characteristics. The cases in which these bitumenised mummies were put before being consigned to the mastaba pits or subterranean caves have mostly crumbled to fragments. It was this class of mummy that formed an article of commerce during the Middle Ages. Charms and pharmaceutical preparations were made of them. The lot of the poor was to be merely salted and buried in the sand. When anything is left of them it is the skeleton, with sometimes a little adipocere where there had been flesh. The practice of embalming, after being held to with constancy through so many thousands of years, and ultimately brought to a degree of perfection which left nothing to be desired, was abandoned during the fourth or fifth century before our era, when the country fell under the Persian invasions.

ANCIENT PERSIAN CUSTOMS

The Persians were not converted to Egyptian ways, and though, long afterwards, they abandoned the old religion of Zoroaster, they still continued the ancient funeral rites. These were founded upon the idea that the body when putrid became the habitation of demons, and could not be allowed to defile the sacred elements of fire, earth, and water. There has been no change in their way of disposing of the dead; and, sentiment aside, no sanitary or economical objection can be made to it. Near every town or colony the authorities have to erect a high building—the Tower of Silence. It is public property and for the use of all. After some ceremonies in a place near at hand appointed for the purpose, the dead body is conveyed to the door of the tower by the relatives. There they part with it. Men are engaged specially to do the work within the building. No one else enters. They receive the body, convey it to the platform at the top, and there expose it. Vultures are not long in stripping off and devouring the flesh. In about seven days the bones, lying in the open air and dried by the sun, are fit for removal. They are then thrown down a shaft leading to the lower part of the tower, which forms a sort of common ossuary. Under this system, with good management, there is no danger to the health of the living, and the earth is no loser by the hideous voracity of the vultures.

ASSYRIAN CUSTOMS

The ancient Asiatic empires show us how much, in respect to the treatment of the dead, depends upon the geological characters of a country, as well as upon ideas involved in a growing society or borrowed from other nations. The Chaldeans, though imbibing their notions of an after life in a great measure from the Egyptians, and imitating in some matters their funeral customs, had not the means of carrying them out in the same manner. Their beliefs led them to desire a prolonged preservation of the body. They attempted this by an imperfect kind of embalming, and would have secured

their dead in caves and pyramids, if caves and pyramids had been possible (as in the northern part of Mesopotamia) near the site of Nineveh, at the foot of the mountain ranges where the Tigris had its source. The habit of solidly constructing for sepulchral purposes would then have been established over the lower country. But it was in Chaldea that Egyptian influence was first felt; and it was from Chaldea that civilisation spread northward to Babylon and Nineveh, carrying with it the customs originally acquired. Now Chaldea was throughout a flat, alluvial plain. No rocky hill sides were there to excavate. There was earth to bury in, and clay of which to make bricks to build a covering. From the beginning, men were obliged to content themselves with what could be done with clay, and the custom was never lost. Sometimes the body, in its clothing, was laid upon a flat flooring of burnt bricks, and a small brick vault was turned over it, which served to hold the water jars, arms, and ornaments of the deceased. These vaults were never more than six feet high, about seven feet long, and less than six feet wide. Many only contained one skeleton lying upon its side, others four or five, and in one the remains of eleven bodies were found crowded together. The work was good, and the interior remained perfectly dry. The coffins used on other occasions were made of clay, square-sided, or in the form of jars, cylinders, and even slippers. They always had an air-hole for the escape of gases. More studied shapes were given to those of a later period; they were enamelled and had ornamental panels, all being still of clay. On the spots set apart as cemeteries in Lower Chaldea coffins of every kind were piled up vertically, one upon another, and formed an enormous hillock, which covered a great extent of ground, and could be seen at long distances on the plain. Trenches more than thirty feet deep have been cut in some of these accumulations of coffins without coming to the lowest layers. They must have been the work of many generations; for, as the pile mounted, the style of coffin changed, and the best are always at the top. Priests and architects disposed of the places and directed the construction. When the hillock had reached the regulation height it was bricked over and drained. Some of these burial places, closely studded with tombs and piles of built-in coffins, were more than a mile long and nearly of the same width.

BURIAL CUSTOMS IN INDIA

The 280,000,000 inhabitants in India, though split up into many religious sects, are mainly divisible into Hindoos and Mussulmans. Besides these, the Parsees form a large and intelligent class. These hold the same philosophical views and follow the same practices as their ancestors of Persia. Numerous in Bombay, they inhabit a special quarter near the sea shore. They have their towers of silence. Five of them, of different sizes, stand on the high ground called Malabar Hill. The chief of these is a building 100 yards in diameter, with brick walls, whitewashed externally. The interior is a vast amphitheatre divided into three stages. Each stage contains coffin-like recesses radiating from the walls towards the centre. The upper stage is for the bodies of men, the others for women and children. All these compartments are quite open to the sky, and lined with marble and cement. Vultures in flocks come at the time of interment, and in about two hours strip and devour the flesh. The bones are then exposed and left for some days to dry. When dry they are placed in a central compartment, where, under the action of sun and rain, they fall into dust. The water which percolates through this ossuary is filtered through broken sandstone and charcoal as it passes into the drains, which convey it to deep wells, partially

filled with sand so as to avoid pollution of the soil. Mussulmans in India are all buried, and without coffins. A sort of alcove is made at one side of the bottom of the grave, it is said, to give the body room to sit up when it receives the visits of the angels at death, but in reality to secure a good base for a monument.

Hindoos have much less regard for the dead body. Many of the poorest sort are buried without coffins, and the graveyards are frequented by jackals. Along the banks of the Ganges numbers are thrown into the water, and crocodiles replace the jackals of the cemeteries and the vultures of the towers. To avoid encumbering the ground in the neighbourhood of cities, and for other sanitary reasons, the Government are endeavouring to induce all Hindoos to be burnt, and in Bombay, Madras, Calcutta, and other towns, have set up places on purpose for this ceremony. That at Calcutta is the most important: it is a long gallery with arcades, looking over one of the branches of the river. Men are appointed to superintend the operation, and a great many of the dead are burnt. A few hours after death the body is brought to the place upon a brancard by the friends, who leave it at the door, after making the necessary declarations in the office. The body is laid upon the ground and there burnt with wood and dried cow-dung. In about four hours the burning is completed, or supposed to be so, and the ashes and remains are thrown together into the river. The public, women, and children pass without hindrance near the fires, and may be seen gossiping and playing unconcernedly, though the smell of burnt flesh is sickening, and the smoke and faggots scarcely hide the charred limbs. It is a disgusting spectacle, but makes little impression upon the crowds. The cost of a burning is about four shillings.

BURIAL AMONG THE JEWS

If anywhere we are to look for opinion, precept, or example as to the light in which we should regard the dead, and as to what we ought to do with them, it must be among the Jews. Their minute and comprehensive sanitary ordinances have come down to us in their sacred books. Of no other people have we such ample information on their sentiments and practice. We know that a dead body filled them with a horror of uncleanness, and that they speedily put it away. Two sons were not allowed to go down upon the dead. In the climate of Palestine the feeling was natural, sanctioned by authority and acted on. 'Whosoever toucheth a dead body, or a bone of a man, or a grave shall be unclean.' 'Command the children of Israel that they put out of the camp whosoever is defiled by the dead.' 'The dead shall be cast out as dung.' 'Wheresoever thou findest the dead, bury them.' 'The manner of the Jews is to bury.' The ruling idea in all was infection; the almost universal custom was burial. That it should be so was the inevitable result of the circumstances under which they took their origin, and developed. Abraham, their patriarch, came from a land where it was the only thing done with the dead. In the land of his adoption it was the prevailing observance. Their relations with Egypt, and the long abiding of the people in that country, habituated them to the use of tombs and graves. In the desert they could not burn; and, once entered into the promised land, fire was an abomination of the idolaters, and among themselves a mark of ignominy. It is not therefore surprising that associations so general, and so many concurrent inducements, should give rise with them to the custom of interment; that the custom should perpetuate itself; that the precautions with which it was officially surrounded should render it tolerable and inoffensive; and that as in time the idea of the tombs of their fathers tinged the whole

of their national history, mingled with all their family traditions, and found expression in the aspirations of their dying moments, it became a practice which no external influence could explode.

Both inhumation and entombment were of earlier date than the Mosaic record. Men of wealth bought sepulchral caves, which remained to their descendants. The poor were buried on the spot where, in their wanderings, death overtook them. After the disappearance of their lawgiver, when they were more settled and lived as dwellers in cities, civic regulations fixed the graveyards beyond the walls. Such were the places known as the 'graves of the children of the people,' in the valley of Kedron, and the 'Potters' Field,' bought with the money of Judas, to bury strangers in. In such spots the multitude were laid in the earth, and hewn stones or some other things were generally placed over the grave to show that it had been used for burial, so that passers-by might be warned not to touch it, and might not be polluted.

A natural cave, enlarged and adapted by masonry, was the standard type of sepulchre. When all those in the neighbourhood came to be appropriated and filled up, artificial imitations made their appearance. This was what the structure of the soil suggested and supplied. When the means permitted, they were often prepared beforehand, and stood in gardens, or by road-sides, or even adjoining houses. The head of a family often provided space for more than one generation. The successive interments in Sarah's tomb are a chronicle of the strong family feeling among the Jews. It was deemed a misfortune or an indignity not only to be deprived of burial, but, in a lesser degree, to be excluded from the family sepulchre. In some of the tombs the bodies were so crowded as to be in actual contact.

A sepulchre, complete according to Rabbinical notions, was a cavern about six cubits square, or six by eight, from three sides of which are recessed longitudinally several vaults, each large enough for a corpse. On the fourth side the cavern is approached through a small covered court or portico, of a size to receive the bier and bearers. Sometimes other caverns were entered from the sides of the same portico, and stones which could be rolled closed the entrance. It was the custom to have this part whitened once a year, after the rains before the Passover, to caution against defilement. The neighbourhood of Jerusalem is thickly studded with tombs, some of them of great antiquity and extent. In that called the 'Tombs of the Prophets' two parallel passages ten feet high and six feet wide, are carried through the rock for sixty feet, a third diverging and extending forty feet. They are connected by two cross galleries in concentric curves, one at the extreme end, the other in the middle. The outer one is 115 feet long, and has a range of thirty inches at the level of its floor, radiating outwards. Two small chambers, with similar niches, also open into it.

Notwithstanding the strong expressions used in the Mosaic and prophetic writings in regard to the dead, the fears of defilement, and the inconvenience of purification, the Jews were very respectful in their treatment of the body. It was confided by the relatives to the care of women, who, after washing, anointed it with spices in the form of ointment. Spices were also placed in the folds of the grave-clothes, which were much of the same fashion as those worn in life. The head was covered separately, and the whole swathed with bandages. Coffins were but seldom used, and if used were open. Fixed stone sarcophagi had their place in tombs of rank. Fumigations of myrrh and aloes and other fragrant matters were always in abundance unless poverty prevented; and on great occasions the vessels, bed, furniture, and clothes of the deceased were burnt also.

It does not appear that the 600,000 Israelites who came up out of Egypt at the Exodus had contracted during their more than 400 years' stay in the country any partiality for the system of embalming, at that time admirably practised. It is true that they were settled in the pastoral district of Goshen, far away from the centre of civilisation. But during their long employment as herdsmen of Pharaoh's cattle, and their subsequent oppression under new kings, they must have had constant communication with the capital, and every opportunity of learning all about embalmmment, and taking it up if so they thought fit. They had, in fact, experience of it; for Joseph, who when Jacob died was high in authority, showed no reluctance to avail himself of the skill within his reach. To render easy the accomplishment of his father's wish, Joseph 'commands his servants, the physicians, to embalm his father, and the physicians embalmed Israel.' Joseph too, in his last days, had the same desire to be buried with his ancestors, 'so his brethren embalmed him, and he was put in a coffin in Egypt.' These are the only two known instances of the embalmmment of Jewish bodies, and they were at an early period of the sojourn. After-events, and provocation, may have aroused Israelitish prejudices and hatred of all things Egyptian.

Incineration, as a funeral custom, was never regularly adopted by the Jews. The bodies of criminals and unclean things were thrown into fire. Under certain circumstances, as when defeated in war, some of the slain were burnt to avoid insult to the corpses; but in such case the bones were collected afterwards and conveyed to the sepulchres. For a while it became the practice to honour some of the kings after death with many funeral ceremonies, including the burning of the body. But generally, burning of the human body was regarded as aggravating punishment, or a sign of contumely. There are a few allusions in the Prophets to the use of fire in times of pestilence, but they are very indefinite. Isaiah seems to imply that, after the wondrous destruction of the army of Sennacherib, the bodies were consigned to Tophet. Tophet, in the Valley of Hinnom, to the south of Jerusalem, was a spot polluted by Josiah. From this time it became the common laystall and cesspool of the city, into which its sewage was conducted, to be carried off by the waters of Kedron. It is generally believed that, in this loathsome quarter, perpetual fires were kept up for the combustion of the bodies of those who had been executed, carcasses of animals, and whatever town filth was combustible. With such a spectacle before their eyes, the people were not likely to think complacently of a similar destiny for their own flesh, and we may believe Tacitus when he says that it was a point of Jewish custom *condere quam cremare*.

GRECIAN CUSTOMS

The Greeks, shifting in their politics, in their form of government, and in their religion, inconstant to their gods, and faithless to every shade of belief, never adhered steadily to any one way of disposing of their dead. When Greece was occupied by the Pelasgic tribes, and its worship was that of the Dodonæan Jove, the people were not mere barbarians, but their progress was of home growth, and little affected by foreign influence. At that stage of Grecian existence the dead were simply buried. Oriental strangers who settled in the country brought with them the higher fruits of civilisation. The Egyptians taught the art of embalmmment, and for a time it was used in an imperfect manner. The Phœnicians introduced the art of writing and the practice of burning the dead. During the Heroic Age it was an honour for the body to be burnt, and a mark of disgrace to be refused burning. To avoid

pestilence, the slain in battle were also burnt. For the next 400 years we have but little positive information about domestic life in Greece. With a number of small states in various stages of refinement, and every small town insisting upon its independence and regulating its own affairs, funebrial habitudes could have no uniformity. Each community did as it liked with its dead. Some burnt, some buried; none were enthusiastic either for earth or fire. The same licence was taken both for neglect and ostentation. The legislation of Solon brought order out of notorious irregularities, and laid the foundation of the greatness of Athens. The laws were numerous, and contained prescripts on almost all subjects connected with the public and private life of the citizens. The Council of the Areopagus was entrusted with the duty of examining into every man's mode of life, and punishing profligacy. Solon found extravagance in funeral rites rampant, and put restrictions on the pyre. It was usual to throw into it arms, jewels, dresses, cattle. He limited the numbers and cost. Solon himself was burnt, and his ashes are said to have been scattered, by his own direction, round the island of Salamis. The continuance of the practice is noted by successive ceremonies. Alcibiades was burnt during the government of the Thirty Tyrants; Timoleon, about 180 years after; Philopœmen, when the struggle with the Romans was beginning; and Plutarch, the biographer and philosopher, when Greece had fallen to be nothing more than a Roman province. But the pyre was not applied to all. Argesilaus, who lived to the age of eighty, and saw the extinction of Spartan supremacy, was embalmed in wax and splendidly buried in Sparta. The body of Alexander was preserved in honey and perfumes, much in the same way as the Burmese at the present day treat the remains of a pre-eminently holy man. Demosthenes, the orator, too, when dying from the poison he had taken in the temple of Poseidon, to escape from the insults of his conquerors after the Lamian struggle, taunted his persecutor, Archias, with being capable of casting him out unburied. The hero Theseus had been buried in the isle of Scyros. About 470 B.C., in the time of Aristides, by command of an oracle, his bones were disinterred and carried to Athens, where they were deposited with much solemnity in a temple, now known as the Theseum. It is clear that the burning of bodies was never a practice universally resorted to in Greece. The state of the population forbade it. A census, taken by Demetrius, of the male inhabitants of Attica gave the amazing number of 400,000 slaves in the possession of about 80,000 freemen and alien residents. The masters would not have taken the trouble to incinerate the large proportion of these miserable dependents that must have been among their dead. The Greeks, though exacting on the point of physical training, carrying luxury to its highest pitch of refinement, excelling in art, oratory, and literature, infiltrating the world with their philosophical morality, were never very particular in sanitary matters.

ROMAN INTERMENTS AND BURNING

By way of contrast, we find the Romans paying great attention to practical utility. At an early period they buried only; then they took from the Etruscans the habit of burning the dead. Men were free to choose between the two systems, but habitations were more numerous, the population was increasing, and the deaths came in proportion. While they were draining their city and watering the streets, other sources of evil were not overlooked. In the code of the Decemvirs, the famous Twelve Tables, which were considered the foundation of all law, it is decreed, '*Hominem mortuum in urbe ne sepelito neve urito.*' This law banished all sepulchral rites beyond

the walls. The *ustrina* were built there, and such interments as took place were allowed there only. It is not known how many fires were set up. But they were under public control. Those for the rich were apart from the others. The bodies of the poor were treated with no decency. One pile was made to serve for a large number. The place appointed for them was in a wretched suburb, on the Esquiline Hill, inhabited by the outcasts of society. In the slang of the people, the burning-places were called '*culinae*.' The attendants were slaves. Night was chosen for the operation. Body after body was thrown upon the open fire, and long before they were consumed the scorched fragments were raked from the smouldering faggots and cast into a common pit.

The burning of the rich required many assistants, and was attended with great ceremony. Oils and balsams were poured over the body. Incisions were made to be certain of death. Asbestos wrappers were used to separate the ashes from the embers. Locks of hair, cut from the heads of the relatives who were grouped about in mourning garments of white, were enclosed with the body. The next of kin, when all was ready, unclosed the eyes and turning aside put fire to the pyre. When the flames were fiercest, favourite animals, arms, dresses, and other things belonging to the dead, were thrust into the conflagration, and spices, oils, and balsams were spread about in profusion. It was considered a disgrace if the body were not completely consumed. The ashes were sealed up in urns made of marble or bronze, and sometimes of gold or silver. These were arranged in the tombs or niches of columbaria. Many of these are now to be seen by the sides of the roads leading away from Rome. They began to make their appearance after the closing of the city to funerals. Some contain sarcophagi in which lie untouched remains, others the urns and ashes of those whose bodies had been burnt. In the celebrated tomb of the Scipios, the bones of its founder were discovered (1780) in a state of good preservation. Other bodies had been laid there too. The tomb was originally above the surface of the ground, with a lofty threshold, the interior supported by walls hewn in the solid tuffstone rock.

The columbaria were subterranean chambers, also in the rock. A steep staircase descends into a square vault with a central buttress, hollowed out, as well as the walls, into niches for the reception of cinerary urns. Others are more like vaulted passages, with dark prolongations intended only for the remains of slaves. The columbaria were usually constructed by several persons in common, or as a matter of speculation, and single recesses could be purchased, sold, or inherited. The names of the occupants were inscribed on marble tablets over the niches. One of the columbaria, among the trees in the grounds of the Villa Pamfili, has the walls decorated with paintings.

As long as the Republic lasted, the choice between burning and burial was unrestricted. Burning was made compulsory during the Empire. The aristocracy favoured it, and the usual imitative propensity of society had its effect. For a time it became popular. But such obsequies gave a fine occasion for the extravagance of emperors and parasites. They expended fabulous sums upon the pyres and incense. The prodigality excited envy. It was impossible for ordinary subjects to approach or to vie with it. A reaction of feeling set in. The duty was imperative, but to fulfil it, as had become the fashion, was ruinous. Persecution could not prevent the new sect which had sprung up in Rome from worshipping and burying in the Catacombs. Christian sentiment, and evasion of the law, happened to fall in exactly with pagan discontent, and fomented it. Political confusion and

relaxation of civic discipline opened the door to malcontent mourners. Cemeteries were clandestinely put to use, and there was either no will or no power to pursue illegality. The Emperor Constantine looked with more than leniency upon the new doctrines and practices, so that by a concurrence of suppressing causes, before the end of the fourth century, burning the body was no longer a Roman usage. It lingered in the country towns and provinces, but with its decline in the capital begins the history of Christian burial.

CHRISTIAN BURIAL IN ROME

The Christians began to be known shortly after Augustus became emperor. They soon found their way to Rome and made many proselytes. At that time extramural burning of the dead was compulsory ; with sectarian obstinacy, they would not comply. Sentiment, opinion, and expediency were all against it. As Sir Thomas Brown says : ' Christians abhorred this way of obsequies ; and though they stickt not to give their bodies to be burnt in their lives, detested that mode after death.' It jarred with their holding of the doctrine of a resurrection of the actual body. It could not be done in secret, and would subject them to molestation in the performance of their religious rites. Christianity at first was regarded as a Jewish sect ; and the fresh converts clung to their ancient customs. With the zeal of a new faith, they were bent upon open demonstration of a repudiation of all pagan practices. Burning was not only to be put aside for burial, but everything resembling the ceremonies attending burning was to be avoided for a simpler service. Their Church organisation was well defined, and their discipline strict. The first disciples were giving place to bishops, dictators as well as teachers. They were in a difficult position. But persecution taught them the power of combination, and they were enabled to extricate themselves by the device of constructing what we know as the Catacombs. These were their early burying-places, quite distinct from the then disused grounds of the Roman people. They distinguished them by the Greek name of *cœmeteria*. The Roman law acknowledged them, and they came under the regulations relating to family tombs and the joint-stock tombs of the *collegia*. They were necessarily outside the precincts of the city. The earliest are situated between the first and third milestones beyond the Aurelian wall. Some of them were private property, others in the hands of Christian societies got up for the purpose of working them. In both cases, the purchase of a definite area was obligatory, and it is ascertained that they are generally exact as to the extent of the excavations prescribed by the Roman law.

The arrangement of these primary *cœmeteria* was extremely simple. The memory of the Judean caves seems to have dwelt in the minds of the designers. They were nothing more than excavations in the soft rock of narrow passages barely three feet wide and sometimes much less, with recesses in the sides just the length required for a body. A regular society of *fossores* was set on foot, and it held on to its functions till the use of the catacombs was given up. Deacons also were appointed to take charge of the property and direct the allotment of places. At first, accommodation for burying only was required, and they served for no other purpose. But as circumstances altered, objects of veneration accumulated, and persecution pressed, they were used for worship and concealment. The passages were then made narrower and higher, and lateral enlargements gave space for reunions. Communications cut from one property to another ended in forming complicated labyrinths. Light and air were in many cases admitted from above by means of

Luminaria. By degrees the catacombs became very extensive, and spread around the city in a wide circle; exclusive of the smaller ones, there were about twenty-six. An ecclesiastical supervision of these cemeteries is mentioned for the first time about 200 A.D., and after that several of the first series of Popes, or Bishops of Rome, were interred in them. In this, and part of the next century, large chambers were constructed, and sarcophagi introduced, in addition to the closed recesses in the walls. The passages were also excavated in stages, one over the other. Other sects, and the Jews themselves, at last began to use similar cemeteries for their interments. But, when in the fourth century the greater part of the population had passed over to Christianity, and become a power in the State, other ideas came to prevail and the catacombs were less turned to account. Constantine was Emperor and Sylvester was Pope. Constantine openly professed himself a Christian. The reaction was sharp. Persecution ceased, the Christians were protected. The temples of the gods were given over to them, and they were permitted to fill civil and military offices. They soon took advantage of their new powers. The wise prohibitory laws of the ancients were set aside, and burials sanctioned in the churches. Constantine gave to the community the first Christian cemetery within the walls, and filled it with earth from Mount Calvary. Shortly before his death he received baptism; when dead, the remains of the first ruler of the earth who avowed himself a convert to the creed of the Christians were admitted within one of their sacred edifices, leaving in the hands of ecclesiastics the control of sepulchral observances, a fearful weapon of spiritual tyranny, oppressive extortion, and physical injury. For more than the next two hundred years intellectual darkness, religious corruption, and political confusion spread over Rome and the Roman provinces. Alaric overran the city, and Attila drew off his army laden with spoils, at the intercession of the venerable Leo. During the frequent devastations undergone by the city, the catacombs were pillaged, the mausoleum of Augustus, and the tomb of Hadrian were stripped of their contents and converted to other purposes. Under such circumstances, burial customs must have been as much unbridled as other things. Popes were buried in the vaults of churches, and the people had their own way with the cemeteries.

CHRISTIAN BURIAL IN GREAT BRITAIN

In the one hundred years between the death of Constantine and the reign of Honorius, no attempt seems to have been made by the Church of Rome to introduce Christianity, with its rites and customs, into its British province. The natives were split apart into two classes; those living in the towns occupied by the legions, adopting Roman ways and language; and the greater part scattered over the country, speaking their own tongue and in the slightest degree only influenced in their habits by foreign example. But Christianity had found its way among them from Iona, and that it had ably done its work we gather from the fact that on the arrival of the new invaders, under Hengist, their fiercest rage was against the clergy. Priests were slain at the altar, and it was in vain that the people sought shelter in the churches, for the churches were fired. Where there were priests and churches, churchyards and burials were sure to be also. The tombs at Iona and Lindisfarne show this. But the country relapsed. Numbers of the population were driven into the wilds of the West, were massacred, or sold away into slavery. Gildas gives us a glimpse of shrines polluted by heathen impiety, by 'barbarians, hateful to God and man.' It was only in the pontificate of Gregory (596 A.D.) that his missionaries got a hold of the people of Kent. Aidan,

from Ireland, assisted them. The struggle with heathendom was at last successful, and the North-country burnings and tumuli were superseded by the family tomb, with the commemorative 'His jacet.' But the parochial divisions, as they now exist, did not take place in England till near the time of the Roman Conquest. Rural churches had been erected by private individuals, or groups of inhabitants, as necessity suggested, and were served by itinerant ministers. Some of these obtained concessions of tithes and the privileges of baptism and burial, implying a churchyard and the residence of a minister. The Church had long found the secret of making its sacraments useful weapons of domination, and the powers of excommunication and interdiction served them profitably. Everywhere the excommunicated were debarred of a regular sepulture. Corruption in a ground which consecration had thrown into the hands of the clergy was set up as a privilege unfit for those who had died in so irregular a manner. All the artifices of ceremonial and the unctuousness of eloquence were conjoined to make the people look upon canonical interment as the guerdon of prescribed observance. There was but one litany for the dead, rich and poor. It spoke of nothing but the grave. The associations of the churchyard by degrees intertwined themselves with all family ties, and rendered it an object of conservative devotion. Abbeys and ministers were honoured with the obsequies of princes and nobles, and the chancel or the nave of the village church became the ambition of the parson and the petty proprietor. Monumental urns and inverted torches, and the constant iteration of the formula, 'ashes to ashes,' may have kept alive some faint reminiscences of ancient customs; but the sight of the anti-heretical stake in the market place, the quarterings and burnings of the public executioner, the fulminations from the pulpit, and the too literal interpretation of Scripture phrases, all tended to make the thought of fire odious. In this way, without any suspicion of prospective evils, vault interments and churchyard burials came to be ratified as national both in thought and deed.

The Reformation only led the way to a change for the worse. There was neither unity of opinion nor uniformity of action. Henry's Protestants held to the churchyard. Elizabeth's schismatics would have none of it. Puritans scoffed at 'God's acre,' and the right of lying there was less dear to them than the right of digging a pit in some conventicle courtyard. It was not their own choice, but a few of the notorious among them served to illustrate one of the most ignoble ways of disposing of the dead ever practised in England. Cromwell, it is true, was first of all carried to the Abbey with more than regal splendour. But in less than two years he was torn from his grave. His body was hung upon the gibbet at Tyburn, and those of Bradshaw and Ireton hung there with him. Some of us, who were living in the early part of this century, may remember what this apparatus of judicial or political vengeance was. Numbers of gibbets stood along the banks of the river, on lonely heaths, and other conspicuous places. The festering carcase dangled in chains from the cross-bar of a tall upright mast, a terror to the timid and a jesting-stock to the reckless most likely to mount there in their turn. The wind, rain, and vermin mangled the flesh into fragments, the tattered rags and droppings fouled the soil, the stench was horrible, and infected the air around; the sight was humiliating. Yet it was a legal custom, more than tolerated in an unfastidious age. It was the grave unmasked.

The burning of heretics and witches could hardly be called a mode of sepulture. It stood more in the light of a punishment or a mark of superstition and fanatical hatred. Drawing, quartering, and garnishing gates,

bridges, and battlements with the heads, limbs, and dismembered trunks, certainly was a way of disposing of the dead of old standing, and characteristically adopted by the sovereign of whom Macaulay says, 'No one magnanimous or human action is recorded of him.' After the Pretender's defeat in 1745, he made the Scotch Lords Lovat, Balmerino, and Kilmarnock the last examples of it. They offer no sanitary lesson.

In 1665, London had only a trifle over 500,000 inhabitants. It was of no great size, with narrow streets and ill-ventilated houses. A large proportion of its space was taken up by churches and churchyards. These churchyards, in the midst of the population, were the only places used for burial. There was no effective drainage, and the soil must have been saturated with the ooziings of decomposition from the bodies accumulated during the many hundred years of the city's existence. It was not, as now, merely a place of resort for business. It was the home of its traders, who, though opulent and well-housed, supported dirt and foul odours as if they were the natural accidents of town life. Even in times not accounted sickly, the rate of mortality was double what it is at present. It had often been scourged by contagious disease, but in 1665 a pestilence surpassing in horror any that during three centuries had visited the island swept away in six months more than 100,000 human beings. This was a fifth part of its ordinary population; but a much larger proportion of those who had not the means of seeking safety by flight remained to take their chance. The city graveyards, already overcharged, could not receive the dead. But, as burial was the only expedient thought of, pits were dug in the outskirts; and for six months that the plague lasted the dead-carts nightly shot corpses into them by scores. For years these ghastly spots were looked upon with distrust, and left unbuilt. Though the plague itself did not return, one of the best known of them, that over which Little Marlborough Street now runs, proved how dangerous a proceeding this was. During the prevalence of cholera in 1854, in spite of warning, the soil was disturbed, and a great aggravation of the disease in that district followed, doubtless from the use of polluted drinking water from a favourite well. Fortunately, fire came after the plague, and cleared away London from the Tower to the Temple, from the river to the open space of Smithfield. Fifteen hundred houses and eighty-nine churches were in ruins. All were rapidly rebuilt, the churches on the old sites, with their graveyards around them. It is due to the memory of Sir Christopher Wren to state that suburban cemeteries formed part of his plan for the restoration of London. It may be inferred what were the practices of that time, when we find him advancing, as one of the recommendations of his new grounds, that in them 'the dead need not be disturbed at the pleasure of the sexton, or piled four or five upon one another, or bones thrown out to gain room.' He was overruled by the Corporation. The system of intramural burying recommenced. Every inch of ground and every dark nook under a church was disputed, and we may recount a succession of vault interments ever since from Milton's in St. Giles', Cripplegate, to a late Lord Mayor in St. Paul's. But that was not enough. The cry was still for more room. Bunhill Fields was then out of London, almost in the open country. Towards the end of his life Milton had a house there. About the same time, the owners of a large piece of ground adjoining opened it as a cemetery on speculation. Bunyan and Daniel Defoe were buried there. Thenceforth it became the Mecca of the Dissenters. No chapel, no altar was built upon it. Its popularity was something extraordinary. It soon filled, was extended, and filled again and again. For more than 150 years it continued, absorbing generation after generation of Nonconformists,

till at last it swelled high above the road. In some respects it was perhaps better ordered than many others of which it was the forerunner, but it was the type of a series of imitations which grew to be as great a scandal as the parochial grounds.

Our predecessors of the eighteenth century were improving in many respects beyond theirs of the seventeenth; but for burials they were worse off than they would have been in the days of the Tudors and Stuarts. Towns grew bigger; the churchyard only kept within its old boundary walls. It was the same everywhere; the same difficulty, the same indifference. New liberties had been acquired, new interests awakened. Under these circumstances men were tolerant of social miseries, were satisfied with an indifferent police and a heedless parochial administration, lived content with home conditions as they found them, ascribed all calamities to Providence, and in death had no other thought than of going where their fathers had gone before them. What could a man want more than to be buried in his parish churchyard? An admitted right shut out all question of a possible danger. The churchyard was there, had been where it was for hundreds of years, and, for aught that could be seen, might be there for ever. It was the business of the sexton to keep the ground always in working order.

But with settled peace in Europe came time for reflection. Thought was directed to internal affairs. Interest was excited about many matters that had been neglected, or left entirely to local authorities. Advance was made on all points. Social and political changes took place. Education was demanded, and, above the rest, medical education made a great bound. More students came to the schools, and there was more teaching. Anatomy especially had to be more fully demonstrated. More subjects for dissection had to be procured. Body-snatching came to be a lucrative occupation. It suggested new crimes. Not only the pillage of the graves caused anxiety and suspicion among those who had lost relatives, but the discovery of the murders in London and Edinburgh, committed expressly for the supply of bodies to the profession, gave rise to a general feeling of insecurity. The vigilance of those responsible in the medical schools and the Anatomy Bill of 1832 put a stop to the murders. But the robbing of graves for the sale of bodies had made a strong impression on the public. Their first concern was about the security of the dead. Censurable laxity in the management of the churchyards had to be redressed, and in looking into this matter other evils forced themselves into notice. Churchyard literature appeared, and more than met the curiosity of the public. It startled everybody. Mr. G. A. Walker, a London surgeon, published a book in 1839, entitled 'Gatherings in Graveyards.' He related a mass of evidence proving both that the directors were negligent as regards the safe keeping of the bodies under their charge from outside marauders, and that in most cases their malpractices went so far as a systematic violation of the purchased graves for their own profit, taking out the dead from the coffins to make room for fresh interments. He also brought forward the question of the influence of burying the dead in the midst of the living, and gave a detail of the dangers and fatal consequences resulting from the practice. Mr. Sopwith further took upon himself the task of calculating and reducing to demonstration the inadequacy of the space used as burying ground for the number of dead requiring interment.

The first effect of this agitation was to foment into joint action the cupidity of speculative and the sensibility of nascent sanitarianism by the formation of new suburban cemetery companies. Kensal Green and Norwood were attractively laid out, and offered to the public 'with genuine mercantile officiousness.' They were eagerly caught at, and frequented; but, as an

official report states, they 'have done little in improving the last service performed for the dead.'

It became at length impossible for the Government to withhold interference. In 1842, under the ministry of Sir Robert Peel, a Parliamentary Commission was appointed to inquire into the sanitary condition of the labouring population of Great Britain. Mr. (afterwards Sir Edwin) Chadwick was requested by Sir James Graham, Secretary for the Home Department, to conduct a special inquiry into the practice of interment in towns and to make a report thereon. This report was presented to both Houses of Parliament in 1848, and is most elaborate. The evidence and recommendations which it contained formed the base of useful operation. A Board of Health was established. The Commissioners examined a great number of witnesses, printed their statements, and issued a report in 1850, signed by Dr. Sutherland. This report confirmed all that had before been made known as to the state of the burial grounds, their evil influence, and the necessity of authoritative regulation of the practice of interments throughout the kingdom. It also included many new details respecting country places. Two years afterwards, in 1855, an Act, known as the Burial Act, was passed. This Act put it in the power of local authorities to shut up or regulate all the graveyards which 'were conducted in such a manner as to be dangerous to health, or offensive, or contrary to decency.' The onus of providing cemeteries for the people was laid on the parochial boards and borough councils, which were empowered to take ground for the purpose. Sir G. C. Lewis, Home Secretary in 1868, issued regulations for the burial-grounds under the Burials Act. In them the following rules were laid down :—

'(a) The grave spaces for the burial of persons above twelve years of age shall be at least 9 feet by 4 feet, and those for the burial of children under twelve years of age 6 feet by 8 feet, or 4½ feet by 4 feet.

'(b) One body only shall be buried in a grave at one time, unless the bodies be those of members of the same family.

'(c) No unvalled grave shall be reopened within fourteen years after the burial of a person above twelve years of age, or within eight years after the burial of a child under twelve years of age, unless to bury the body of another member of the same family, in which case a layer of earth, not less than 1 foot thick, shall be left undisturbed above the previously buried coffin.

'(d) No coffin shall be buried in any unvalled grave within 4 feet of the ordinary level of the ground, unless it contains the body of a child under twelve years of age, when it shall not be less than 8 feet below the level.'

By this time some 400 local Burial Boards had been constituted. Five hundred Orders in Council had caused the closing and regulating (that is, forbidding all interments except in family graves) of about 4,000 old burial-grounds. These proceedings threw upon the ratepayers a very heavy burden, amounting then to about one million and a half.

In 1867 there was legislation for Scotland, and the Public Health Act of that year enacted that 'any churchyard, cemetery, or place of sepulture, so situated or so crowded with bodies, or otherwise so conducted as to be offensive or injurious to health, may be treated as a nuisance, and shut up or regulated, on satisfactory proof being laid before the Sheriff by the local authority of the place in which the graveyard is situated.' Many cemeteries in various parts of Scotland were in consequence shut up, and companies formed for providing accommodation. These companies, though organised by humane and public-spirited citizens, are now conducted on purely commercial principles. There is sufficient justification in the reports which have from time to time been made as to their condition for the assertion that 'the

duty of the local authorities in respect to their proper use and conservation is either very imperfectly performed or not attended to at all.' Even in Edinburgh, where the matter was investigated by the Health authorities in 1888, most insanitary and unseemly conditions existed. Some of the grosser abuses connected with vault burials and city churchyards had been removed; but a recent writer on the subject declares that the methods of treating the bodies of the poor in that city 'would not be tolerated in any other country in Europe.' He insists upon the necessity of further legislation to put all private cemetery companies under the Government regulations for burial in grounds under the Burial Acts. But experience elsewhere shows that this will only give a partial and short-lasting relief.

For the last forty years, we in England have been living with the question of the disposal of the dead in a state of tentative experimentation. We have been disciplined by Burial Laws and Orders in Council, directed and regulated by Boards of Health and Burial Boards, general and local, superintended by Officers of Health, scrutinised by inspectors, certified by doctors and registrars, lectured and advised by Sanitary Societies and Congresses. We have closed the vaults of our churches, we have tried suburban cemeteries, we have been embarrassed by no extraordinary pestilence, and we have cleared away a great mass of perishable population by emigration; and yet we are in the midst of wide diversities of opinion and no little perplexity. We find that we have had only a temporary respite, and that we have done no more than shift the locality of the evil of burying. The dead are more numerous than ever; many cemeteries are filling to repletion, are no longer isolated, and only suburban in name. The question what we had better do with our dead is as open as ever, and while we drift with the current customs it grows in urgency day by day.

Looking at the matter from a scientific point of view, and apart from sentiment, many alternative methods have been proposed, and some of them tested. But after all, burying, with its increasing difficulties, and in spite of its experienced evils and abuses, is the dominant idea. That it should have adherents, even among those who can see the harm it leads to, is natural. That it has defenders and advocates is but too true, and makes it necessary, in considering the expediency and practicability of other schemes for treating the dead, once more to expose the evils and abuses they are intended to obviate.

MODERN BURIAL

If we were living a nomad life, the question of burial would be of little importance. A dead body would lie alone where it fell, covered with a few feet of soil, and there undergo its natural dissolution. The earth would retain and assimilate its share of the remains. The gases would be dispersed on the winds and imperceptibly disappear. Repeated from time to time, and at distant spots, the survivors would always be out of reach of any harm. But when population is centralised, the very circumstances of civilisation prevent dispersion. Time and money are both wanting. The poor in death are more powerful than the rich—in numbers greater, a majority of the majority, and quite as putrefactive as Dives. It is the accumulation of the dead in living communities that is the source of mischief. A generation of corruptive bodies may last as long as a generation of men, and, instead of giving place the one to the other, the dead take time to decay while the living die. Life, in fact, is on an average shorter than decay, and the second generation of living intrude upon their predecessors. The succession is too quick. In this fact lies the difficulty of cemetery burial, and out of it have sprung its

evils and abuses. The law has failed to make the right of the dead to the grave sufficiently long; thoughtlessness and cupidity have encroached upon this right. So, from this want of protection, and the working of these human failings, the churchyard has become an offence and often a scourge. We have, then, to treat of three points:—(1) The inherent evil of collective burial; (2) the bad management of cemeteries; (3) the shortcomings of the law in relation to them.

(1) *The Evils of Burial*.—The charge made against the practice of burial may be summed up as follows:—Generally, the places used for the purpose are sometimes offensive; this offensiveness is caused by the decomposition of the animal matter deposited in them; the offensiveness is intensified into mischief by the undue agglomeration of decomposing bodies; they predispose to disease, and are a source of disease, by the products of decomposition poisoning earth and water as well as air, and by preserving the germs of infective diseases. Specially, as regards this country, many have always been in a bad state, and are so still. They have never been of adequate size. They do not now furnish the accommodation required.

Burial-grounds are Offensive.—This may be a truism, but it is a truism which everyone would be glad to see falsified. Forty years ago, whoever walked the streets of London knew what was to be met with in passing by certain burial-places. It scarcely seems to be a matter on which to take formal evidence, but all classes of witnesses before the Commissions of which mention has been made began with this point, and bore testimony that the stench proceeding from them was frequently so great that the residents in the neighbourhood were obliged to shut their windows for hours and days together, and complained of being subject to headaches, nausea, sore throats, and diarrhoea. In vain the surface was covered with flagstones and monuments; in vain couch-grass, violets, roses, weeping willows, cypresses, were planted; the soil was found as a black, unctuous, animalised compost, giving out, not the smell of earth, but a material cadaveric something, as impalpable and unanalysable as the scent of musk—a diluted, diffusible poison, which only waits to meet with certain susceptibilities, or a concurrence of circumstances, to show its effects. The only word that can be said about it which is not a condemnation is that by its disgusting character it gives warning of the approach of matters containing noxious properties.

The offensiveness protested against comes from putrefying matter in the graveyards. But we have no right to call decomposition itself an evil, any more than to say so of a volcano. No man in his senses would go to live near the mouth of a crater, and none but an idiot would store up putrefying matters under his window. When a bee dies it is carried away from the hive. Old birds cast out any dead fledglings from the nest. All natural phenomena are in man's favour if he does not interfere with them injudiciously. The rain falls in fertilising drops over the face of the earth, but that is no reason why man should collect it in a cistern to drown himself. The resolution of organic remains into their elements is in the cycle of Nature's operations. It is indispensable for the reproduction of living beings.

As Parkes wrote: 'The body returns to its elements and gradually, and often by the means of other forms of life which prey upon it, a large amount of it forms carbonic acid, ammonia, sulphuretted and carburetted hydrogen, nitrous and nitric acid, and various more complex gaseous products, many of which are very foetid, but which, however, are eventually all oxygenised into simpler combinations.'

Playfair says: 'There are two kinds of changes which animal and vegetable matters undergo when exposed to certain influences. These are known

by the terms of "decay" and "putrefaction." Decay, properly so called, is a union of the elements of organic matter with the oxygen of the air; while putrefaction, although generally commencing with decay, is a change or transformation of the elements of the organic body itself, without any necessary union with the oxygen of the air. When decay proceeds in a body without putrefaction, offensive smells are not generated, but if the air in contact with the decaying matter be in any way deficient, the decay passes into putrefaction, and putrid smells arise.'

Now, man's interference with this natural process by way of burial is due either to ignorance, or want of reflection, or to 'the instinct of affection which most naturally follows even the sadly changed remains of our dearest relatives.' We forget that what is given is 'taken away,' and that, once life has ceased, the body is no longer personal property.

By putting a body in a grave we thwart the purpose of restitution. By using coffins and lead, we go still farther in the way of detention. We 'make more and more deficient the air in contact with the decaying matter, and decay passes into putrefaction and putrid smells arise.'

Intensified by Accumulation.—We create an evil, and, worse still, we aggravate that evil by the insensate habitude of using the same place for the same purpose of burial with incessant iteration. It set a bad example in creating churchyards. Sectarians who objected to the ritual observed in consecrated ground met with no obstacle to filling their own chapel-yards; and others, with no higher motive than gain, took advantage of the free trade in burial to make provision for a public want. Official reports tell us what this ecclesiastical conservatism and independent action ended in. One report to the Board of Health in 1850 says: 'In Bethnal Green burial-ground alone, consisting of an area of about two acres and a half, there have been interred since its opening, in the year 1746, upwards of 56,000 dead bodies. In Bunhill Fields burial-ground, City Road, consisting of an area of less than four acres, there have been interred, from April 1718 to August 1832, according to the registry, which, however, in the earlier years was very imperfectly kept, 107,416 bodies. But in St. Pancras Churchyard, one-half of which has been used as a burial-place for at least six centuries, there have been deposited the remains of more than twenty generations; and in this space of ground, which does not even now exceed four acres, and a large portion of which was considered as full to excess twenty years ago, there have been interred since that period upwards of 26,000 bodies.'

Sir E. Chadwick reports that, 'in the metropolis, on spaces of ground which do not exceed 208 acres, closely surrounded by the abodes of the living, layer upon layer, each consisting of a population numerically equivalent to a large army of 20,000 adults and nearly 80,000 youths and children, is every year imperfectly interred. Within the period of the existence of the present generation upwards of a million of dead must have been interred in those same spaces.'

Dr. Sutherland, in writing to the Board of Health in 1850, says: 'It is not too much to assert that it would require centuries of judicious management to purify the soil of the existing burial-places, so as to make it fit for fresh interments. Some burial-grounds which I have myself visited appear to consist entirely of crushed bones and unctuous animal mould. A few days ago I saw a grave in the process of being opened, in a graveyard in Upper Whitecross Street, belonging to the parish of St. Giles, Cripplegate. It was about six feet deep, and appeared to have been cut out of a 'breccia' of human bones. The grave-digger was breaking or laying bare large fragments with every stroke of the pickaxe, and on the surface there were five

skulls, four of them entire, and the bones of apparently as many skeletons, so fresh that the soft parts might have been just removed.'

In another report in 1851, Mr. Lee, a superintending inspector, states that 'the graveyards of country towns were in general in no better condition than those of the metropolis, and that the practices of interment in use among some of them were even worse.' Of Norwich it says: 'As far back as 1671, it appears from the following extract from Evelyn's "*Memoirs*" of that date that the greater part of the burial-grounds of this town were then full and quite unfit for further interments. Evelyn says: "I observed that most of the churchyards (though some of them large enough) were filled up with earth, or rather, the congestion of dead bodies one on another, for want of earth, even to the very top of the walls, and some above the walls, so as the churches seem to be built in pits." The addition of all who have died in this great city during nearly two centuries that have elapsed since Evelyn wrote has of course made their condition still worse. It is probable that above a quarter of a million of human bodies have been interred since it was said that the earth was raised by "the congestion of dead bodies one above another, for want of earth, even to the very top of the walls, and some above the walls;" and all this in the midst of a dense population. I inspected the whole of the burial-grounds attached to both churches and chapels, and declare without hesitation, of all the thirty-four parish churchyards in the city, that a very large portion of the soil for the depth of many feet consists of decomposed human bodies.'

On the other side of the Atlantic, where the cemeteries are not ancient, the same massing of bodies takes place. Dr. Peacock, of New York, in a paper read before one of the medical societies, April 1889, tells us that 'in the New York City cemetery there are no single interments. The bodies are placed in trenches, dug in regular rows, 45 feet long, 14 feet wide, and 10 feet deep. Each of these pits will hold 150 bodies, which are laid three deep in six rows of twenty-five each. In 1887, 4,158 bodies were buried there; the interments averaging about thirty per day. In the public or poor quarter of Calvary Cemetery a trench is dug 7 feet wide, 10 to 12 feet deep, and of indefinite length, in which the coffins are stowed tier upon tier, making a flight of steps five or more deep, and with not enough earth to hide one from the next. The cemeteries of Newtown, L.I., contain more than 8,500,000 human remains, and receive annually 80,000 bodies of people dying in New York and Brooklyn.'

The state of vaults under churches and in cemeteries is in no respect better as regards crowding. In the vault of St. Peter's, Cornhill, there were 72 coffins; in St. Andrew's Undershaft, 77; in St. Mary-at-Hill, about 150; in St. Mary-le-Bow and St. Bride's, 500 each; in St. Mary's Woolnoth, 1,100; in the vaults of the cemetery, Bayswater Road, 1,120. In the vaults and catacombs of St. Martin-in-the-Fields the number was still larger. Dr. Waller Lewis suffered severely from his examination of these and other vaults, and reported them all as being in a disgraceful condition. 'In St. Mary-at-Hill, some 160 coffins are placed in all possible positions, piled one above another, the lower crushed by the weight of those above. The great majority are broken and decayed; the remnants of mortality falling out between the rows of coffins. Many of the coffins are so fragile, and the piles so much out of the perpendicular, that it is dangerous to approach them. Many of these are of wood only, and a mere touch exposes the skeleton. All are covered with enormous cobwebs and fungi, dirt and filth. In the two farther corners large collections of bones are piled together, without any attempt at order or decency.' Strong leaden coffins will retain all the products of decomposition

for a very long period. In one instance, a coffin that had been in the vaults for sixty-seven years was perfectly air-tight and contained nearly two gallons of a coffee-coloured ammoniacal fluid. But in the wooden coffins decomposition goes on much more rapidly. Three or four years generally suffice to reduce the contents to a dry powdery skeleton. The surrounding air soon becomes charged with the products, and it is impossible to enter some of the vaults without ventilation for twenty-four hours previously. It was a general custom to clear the vaults from time to time by removing the old coffins to make room for others. On one occasion 'it took a week to do it.' The Commissioners could not do otherwise, in view of all the evidence before them, than adopt the conclusion in regard to the physical evils connected with the practice of interment thus expressed by Chadwick: 'That this injurious influence is manifest in proportion to the degree of concentration of putrid emanations, especially in confined spaces.' Happily, this description is no longer applicable to existing churchyards and cemeteries.

Burial-grounds predispose to Disease and are the Source of Actual Disease.—Early writers on the evils of burial thought it necessary to amass voluminous evidence of the fact that emanations from graves would cause illness and death. The world was sceptical. Burial seemed so natural, and was so sanctioned a custom, that it sounded like heresy to speak ill of it. At the present time opinion has changed. Attention has been awakened, and action shows that conviction of the truth has penetrated many minds. People suffer inconvenience, and, having got rid of the delusion that a thing so intangible as a smell can do no harm, move away from cemeteries. They have been told that the diseases of which men die engender morbid germs, that such germs escape from the grave, and are ready for the act of reproduction as soon as they happen to find a suitable living being to breed in. The realisation of the idea of cause and effect was not here quite as easy as in the case of a shadow falling behind an object which intercepts a ray of light, but it is well enough understood to be a motive for action. The first imperfect result of the instinct of self-preservation has been to remove cemeteries to a distance. New evidence is not needed. The old arguments were put together to show that some modification of the way of burial was wanted, and to induce a belief in the necessity of improvement. Considering that burial is essentially wrong, and that it is impossible, as society is now arranged, to overcome the objectionable conditions, and that some other mode of disposing of the dead must be accepted, a fragment of the evidence already used will suffice for present purposes.

When the fear of contagion was vague, and founded upon a notion of something more like a demon than a mushroom, all that could be done was to hold forth about vaporous influence and to give cautions. We were convinced that the phenomena of disease, however capricious they may seem, are obedient to some absolute uniformity, are enchained by that same rigid sequence of cause and effect which is imposed on all remaining nature. But there was a blank. The effect was appreciable, the cause was suspected, but we could not point out definitely how the sequence was effected. We could speak of facts alone with a short empirical knowledge of their succession. But now, thanks to modern research, we have advanced beyond that empiricism. Certain laws of disease have been determined. The sequences of the facts to which they relate are elucidated. We can guide our actions upon them. We have learnt that decomposition is not so purely a chemical proceeding as it appeared to be, that the dissipation of elements is attended with other phenomena. There is life in death. Incalculable numbers of living organisms find their habitat in a decomposing body—some engaged in the

work of destruction, others of a different kind concerned only with the reproduction of the progeny of disease propagators. We have come to know a great deal of their natural history, and especially how much they have to do with the communication and character of disease. We can now trace materially the connection between the dead body in the grave and the living body in his bed. We can fill in the missing link between the disease that has killed and that which is killing. We can demonstrate microscopically the microbic agent which serves to convey and inflict the infection. The gaseous emanations which escape from the graves of bodies dead from infectious diseases in like manner may carry floating in them, and mixing with the air we breathe, the spores of those diseases. They enter the system of those exposed to them, and under favouring circumstances the disease develops itself anew. Dr. Copland, in his evidence before the Committee of the House of Commons, related that one of his patients went to a chapel where the principal part of the hearers, as they died, were buried in the ground or vaults underneath. On going up the steps of the chapel, he felt a rush of foul air issuing from the gratings on each side of the steps; the effect was instantaneous. It produced a feeling of sinking, with nausea and great debility. He went home and to bed. Eight days afterwards he died of malignant fever. In the early part of his illness his wife had slept in the same bed. She caught the disease and died in eight days also, having experienced the same symptoms.

Water Pollution.—It may be affirmed on the authority of Pasteur that no germs can be found, no germs exist, in spring water at its source. But deaths do occur after drinking the water of certain wells. Some people living in a mountainous country, and having very little communication with each other, were in the habit of quenching their thirst at a neighbouring well after the Sunday's attendance at the church of the district. A young man died of diphtheria and was buried in the yard. The drinking continued, and in a short time twenty of these peasants were carried off by the same disease. If asked how are we to account for this accident but on the presumption that the germs of the disease found their way into the water of the well, various explanations, such as a milk outbreak or personal communication, may be imagined, but none which so exactly correspond with the circumstances. That water will carry with it through the ground saline solutions to a considerable distance is shown by experiment. A salt of lithium was sown over a plot of land more than 150 yards distant from a well, the water of which contained no lithium. Repeated examinations were made, but it was only on the eighteenth day that tests proved the solution had percolated through the soil into the well. Animal impurities in water-courses declare themselves to the senses, and instances of contamination are too well known. Where animal matter finds its way, there can be no obstacle to the migration of microbes. In consequence of various investigations in France, a law was passed prohibiting the opening of wells within 100 yards of any place of burial; but this distance is now stated to be insufficient for deep wells, which have been found polluted at a distance of from 150 to 200 yards. In some parts of Germany, the opening of wells nearer than 300 feet has been prohibited. In London it has been found necessary to shut up most of the City wells on account of contamination of the water.

One of the main arguments against the use of ground for burial is that, over and above the noxious emanations which it emits, it has the property of preserving for so long a time the poisonous chemical combinations developed during decomposition and the dangerous organisms placed there in bodies after death from infectious diseases. Many proofs could be given of this fact, but it is well enough exemplified by a remarkable case, cited on

the high authority of Mr. Wheelhouse, of Leeds, where the seeds of scarlet fever germinated after having been buried for thirty years. In a Yorkshire village, part of a graveyard was taken into the adjoining rectory garden. The earth was dug up, and scarlet fever soon broke out in the rectory nursery, and thence spread over the village. Mr. Wheelhouse satisfied himself that there were no other ascertainable causes for the outbreak. It proved to be of the same virulent character as the scarlet fever which, thirty years before, had destroyed the villagers who were buried in the precise part of the churchyard which had been taken into the garden and dug up. Great force is given to this argument by the observations and experiments of Pasteur, to whom we may chiefly attribute the rapid advance in recent years of our knowledge of the dependence of a considerable number of contagious and infectious diseases upon the action of a morbid poison, or of some low form of vegetable or animal organism, each form or variety causing some definite form or variety of specific disease. Through him, and those who have followed his teaching and example, we have become familiar with microbic association and influence. But it is doubtful whether many have thought much of the way in which we preserve these microscopic enemies of the human race and assist them in their ravages. We attempt to destroy them while our patients are alive by the use of antiseptics and disinfectants; and in the same way, and by isolation of the patients, we try to prevent their extension from sick to healthy persons. But as soon as a patient is dead, the body is disposed of in the very way of all others most favourable for the conservation and multiplication of microbes. It is time that the lesson we have learnt should be brought to bear upon the question of burial.

In Pasteur's investigations of the subject of charbon in animals, after having determined the cause of the disease and its facility of propagation, he was at a loss to know how the spores were introduced into the system in cases of spontaneous disease without inoculation. The presumption was that they entered through some of the mucous membranes. Sheep were fed upon herbage sprinkled over with germs. A few died of the disease, and there were indications that inoculation had taken place in the upper part of the throat. Artificial wounds were made in these parts by giving the animals rough, prickly food. The mortality was increased. The chief seat of inoculation was disclosed. The next thing to do was to find the microbes of charbon in fields where sheep had taken the disease spontaneously. To detect them by the microscope was impossible. Earth was taken from the surface over places where diseased beasts had been buried, was washed, and animals were inoculated with the liquid. They died, but some of other diseases as well as charbon, and the charbon cases showed unusual symptoms from the admixture of microbes of different kinds. The charbon microbes were then separated from the rest, and used for reinoculation. The blood from this second series was taken for inoculation, and pure charbon resulted. Charbon microbes were then proved to exist in those places where charbon burials had been made. They were not found in other parts of the field. The probability was that blood and secretions had been left on the ground when the animals were skinned before being thrown into the pit. Yet the charbon germs were proved to be present also over graves where they had been put in whole. Here the observations of Darwin on the formation of mould, made many years previously, came in aid of the explanation of the problem how the germs got to the surface even from deep pits, and are curiously confirmative of the conclusions of Pasteur. In Darwin's paper, read at the Geological Society of London in 1897, he proved that, in old pasture land, every particle of the superficial layer of earth overlying

different kinds of subsoil has passed through the intestines of worms. The worms swallow earthy matter, and after separating the digestible and serviceable portion they eject the remainder in little coils or heaps at the mouth of their burrows. In dry weather the worms descend to a considerable depth, and seek in preference that part of the soil which contains humus, organic matter in a certain stage of decomposition. Returning to the surface, they void their undigested matter. This agency of earth-worms is not so trivial as it might appear. By observation in different fields, Darwin proved, in one case, that a depth of more than three inches of this worm-mould had been accumulated in fifteen years; and in another, that the earth-worms had covered a bed of marl with their mould in eighty years to an average depth of thirteen inches.

Now Pasteur had shown that this earth-mould positively contains the specific germs which propagated the disease; and that the same specific germs are found within the intestines of the worms, where they lose none of their virulence by the process of digestion. But these views are, perhaps, not yet generally known nor accepted.

The parasitic organism, or *bacteridium*, which, inoculated from a diseased to a healthy animal, propagates the specific disease, may be destroyed by putrefaction after burial. But before this process has been completed, the disengaged gases cause lacerations of the tissues and skin, which admit of the flow of fluids charged with living filaments. They then acquire the necessary contact with air and produce germs or spores. These spores will resist the putrefactive process for many years, and remain in a condition of latent life, like a grain of corn, or any flower-seed, ready to germinate and communicate the disease.

In confirmation of what had previously been ascertained, further observations were made in a field in the Jura where diseased cows had been buried a year before, at a depth of nearly seven feet, the surface earth not having been disturbed in the interval, and it was found that the mould contained germs which, introduced by inoculation into a guineapig, produced charbon and death. Pasteur, every time he has made the experiment of feeding sheep upon forage contaminated with the spores of the bacteridium, has seen them die of charbon. Four sheep confined in a fold over the place of burial of diseased animals died in less than a fortnight, while other sheep kept at some little distance in the same field, where no carcasses had been put underground, were free from all symptoms. On another occasion two sheep out of seven died of charbon within six weeks, during which time they were penned for some hours daily on the site of an old pit where infected animals had lain for twelve years. The surface was quite bare of herbage, and the disease must have been contracted by the sheep sniffing, as is their habit, the ground on which they were confined. The mortality has always been greater when thorns, or any pricking substances, were mixed with the food so as to occasion excoriations of the mouth or gullet. In all these cases the morbid appearances at the autopsy were the same as in animals dying of the disease without intentional inoculation.

The earth in the alimentary canal of the worm taken from an infected spot, if used for inoculation, produces the disease. Mould deposited on the surface by the worms, when dried into dust, is blown over the grass and plants on which the cattle feed, and may thus spread the infection. Even after various farming operations of tilling and harvest, Pasteur has found the germs just over the graves of diseased cattle, but not at any great distance. After rains or morning dews, the germs of charbon, with a quantity of other germs, were discoverable upon the neighbouring plants. The

rain, too, flowing in the water-courses may convey the germs to considerable distances.

Pasteur suggests that, in cemeteries, it is very possible that germs capable of propagating specific diseases of different kinds, quite harmless to the earth-worm, may be brought to the surface of the soil ready to cause diseases in man and animals susceptible of them. The same may be said of other burrowing creatures. Rats, mice, moles, ants, and the palmer-worm frequent cemeteries in numbers. Rats especially, attracted by the decaying bodies, find their way down and into the coffins. Some churchyards were formerly completely undermined by these animals. We do not know much of their idiosyncrasies in regard to different microbes, but as they abound in such numbers they evidently have their exceptions.

Another fact of importance which leads to the renouncing much hope of safety from the growth of vegetation is, that such vegetation does not destroy those morbid spores. It may take up and utilise that which its rootlets meet with in the soil, elaborated by the action of the infinitesimal beings there thriving upon the organic matters with which they are in contact. But their spores resist every kind of vegetable assimilation. Plants may serve the purpose of oxidising the foetid organic exhalations of the body, absorbing some of them and part of the carbonic acid. But as regards the living organisms, we believe they do nothing. 'Le grand végétal ne détruit pas ce sans quoi il ne saurait vivre.'

It is true that for the formation of some of the spores they must be favoured with a certain temperature. In cold and damp weather the filaments may entirely perish, without giving rise to any germs. But in the warmer months, when perhaps infectious diseases are most prevalent, they are sure to be found, especially when the interment has been superficial, as it is for human bodies in most cemeteries.

But, after all, it is not that contagion is so much to be dreaded from the inhalation of infectious germs. In the air they are generally sparsely scattered, and the danger of their absorption by the buccal and pulmonary mucous membranes is less than is generally supposed. Even if numerous, there is a great probability that the air will be well nigh cleared of them by coming in contact with the liquids over which, and through which, it must necessarily pass before penetrating into the ultimate lung tissue. The mischief is more likely to happen from the floating grave-dust impinging upon some abraded surface, or open wound, and in this case the purifying power of vegetation could do nothing.

It is also well, in connection with churchyard moulds, to give attention to the conclusion arrived at by Pasteur, that the conjoint action of different kinds of microbes is much more energetic and fatal than that shown when they infect singly. Thus, when inoculations are made at the same time with the vibron of septicæmia and the *vibron pyogene*, the smallest abscess ends in gangrenous infiltration of the tissues, and death follows more quickly than from infection by either of them separately.

(2) *Bad Management and Insufficiency.*—On these points we can appeal to science, past experience, our own experience, and to the calculations of sanitarians. Modern science, as we have explained, has put in a more glaring light than ever what we have to expect from burial, and especially gregarious burial. Notwithstanding the supposed sanctity from episcopal consecration, the constant presence of ministers of religion, and the sentiment which naturally grows up around 'God's acre,' our experience of the long past has shown that overcrowding was inevitable and desecration the consequence. Graveyards became mere tumuli without Assyrian drainage or covering, burial nothing

more than plunging one man's body into the partly decayed remains of others. The last fifty years have made it plain to us that commercial enterprise is no more to be trusted than pastoral trusteeship. The revelations respecting the state of some of the burial-grounds, and the way in which the bodies are treated, show that this is no fiction.

On the point of insufficiency of space, the old grounds speak for themselves. As they were neither widened nor lengthened, they took the only course open to them, upwards. They deepened. Some of the yards in Sheffield had risen six feet above the streets and level of the houses. The burial-ground of the parish of St. Peter and St. Paul, Barnstaple, was on all sides surrounded by dwellings, and the inmates could sometimes see interments taking place above the level of the rooms in which they were sitting at their meals, and within five or six feet of their windows. Two paths, which were originally open and laid out on the surface of the ground, were enclosed with walls on each side from seven to eight feet high. Holes were cut through them in which to thrust coffins below others that had been put into the ground from above. These may serve as specimens of the incongruous expansion of provincial yards.

As to London, it appears from some returns, laid before the House of Commons in 1894, of the extent of the burial-grounds and numbers of burials during the three years preceding, within the diocese of London, that there were then 103 acres occupied by graves, and that the average number of burials was 219 per acre, though in some grounds it was as high as 891 per acre. In some private grounds within the metropolis, at the same time, the average annual number of burials per acre was more than 1,000. The total number of intramural grounds was 218, and in them were buried annually 44,355 bodies, at an average rate of 208 per acre. When Chadwick wrote his report in 1843, the new companies had 260 acres of land, and were burying 3,336 bodies in the year, and the average per acre was very small.

No one can dispute the simple proposition that, if burial be admitted at all, it should be done in such a way that the bodies be laid in the ground separately, and, when once there, remain undisturbed, and that the spot used should be at a distance from any habitations. These are the only conditions upon which burial can be practised with any prospect of safety and propriety. The inconvenience of opening graves, with escape of fœtid gases, is avoided. There is not the indecency of disturbing remains for new interments, and in case of exhumation the danger of mistaking the particular body is prevented. This is so far from being the case about London, that the calculations for one of the cemeteries is that there must be 8,630 graves to every acre, and that every grave shall contain ten coffins. Yet the Kensal Green directors went far beyond this, by actually offering seven acres of their ground adjoining the cemetery in which they proposed to place 1,835,000 paupers in 138,500 graves. Economical considerations seem to forbid the hope that the 'one body one grave' system will ever be adopted. After all, it is only a question of money and waste of land, and those who adhere to burial in preference to any other way of dealing with the dead ought, in reason, to be prepared to make the necessary sacrifice to avoid its evils and abuses. A few sentences will show how the matter stands, and that we could do the wrong thing in the right way if we chose. London at the present time has for its use 2,200 acres of burial land. About a sixth part of this space is taken up with paths, buildings, and plantations, but each nominal acre of new ground is capable of furnishing 1,100 separate graves. The total number of graves in the 2,200 acres would be at this rate 2,420,000, in each of which a body could be laid

by itself. Now, the present death-rate of London for the last few years has been a little over 80,000 per annum. In thirty years, therefore, there would be the necessity for burying 2,400,000 bodies. This number nearly corresponds with that of the available graves in the cemeteries, and they would there find their respective permanent places. At the end of the thirty years the cemeteries would be full. Much of the land would be encumbered with monuments, and held in perpetuity by the families who had built them. The plain graves would be so intermixed with them that there could be no question of restoring the property to other uses. The bodies, therefore, would rest untouched, as they ought to be. But the same conditions would necessitate recommencing the same proceeding for another thirty years. Before the end of a century this renewal of the ground would have happened three times, involving the purchase and setting apart of three times the same space, that is to say, about 7,000 acres of land. In reality, as there must be an increase of population, larger provision would have to be made; but this does not much affect the possibility. In the same way, if we make the calculation for the dead of the whole of England and Wales, we reach high but not unmanageable figures. Multiplying one of the last annual mortality returns by thirty gives a total of 16,118,280. This number of bodies, adults and children, could be accommodated on the same terms, with a little room to spare, in 15,500 acres, which could be portioned out into about 17,000,000 grave spaces. Three times repeated in the hundred years, there would be swallowed up 46,500 acres, the equivalent of, or a trifle more than, one-fourth of the county of Middlesex. At the price paid for the existing cemeteries, the cost would be about five millions and a half, and the consumption of land would not be so great as that for national pleasure grounds. A scheme like this might be carried out by a rich nation like England without much difficulty. But if 'the question is to be solved on a sternly commercial basis,' it is certain the scheme would never be adopted. Then, on the faith of the maxim that 'if a thing be done at all, it should be done well,' we might fairly ask whether its rejection as an impossibility is not a condemnation of the whole system of burial, as one that can only be done badly. These figures, if they do nothing else, serve to give an idea of how much is wanting in the way of space for the seemly storage of our dead, and how still more perplexing a task it will be in the future to obtain it for them.

(8) *The Law imperfect and not enforced.*—The law formerly but little occupied itself with the subject of burial. It was only in 1588 that any record began to be made of the fact of burial. From that time till 1836 the registers were kept by the clergy or by the proprietors of burial-grounds, generally in a very careless manner. The Registration Act of 1836 made the production of a certificate of the cause of death, signed by a registered medical practitioner, imperative before burial. At various times Acts have been passed authorising the formation of suburban cemeteries, and the Public Health Acts have given powers for the suppression of places of sepulture becoming nuisances. In 1863, the Secretary of State issued regulations for the burial-grounds under the Burial Acts, for the proper keeping of the grounds, the division of them into grave-spaces, and for the conservation of the bodies. In 1874 there was further legislation. These Acts have been amended from time to time, but the truth is that if, before any of them were in existence, free trade in burial had been the rule in England, the neglect of restrictions and the defiance of authority had since become so notorious that in 1884 Dr. (now Sir C.) Cameron introduced a bill in the House of Commons for the 'regulation of the disposal of the dead.' In doing so, he pointed out the prevailing abuses. He insisted upon these points: that the rules respecting

the certificates of death were badly observed, especially in regard to the cause of death ; that the regulations of the grave-yards were not complied with, and that, by this reprehensive complicity between all the parties concerned, facilities were afforded for the committal and concealment of crime. The bill, however, did not pass. The evils he exposed have not since been done away with, for only in December 1888 a deputation of the Church of England Burial Reform Association had an interview with the Secretary of State, for the express purpose of urging upon him the necessity of further Parliamentary inquiry into modes of burial, with a view to new legislation. That this demand was not inopportune is made clear by the many facts that have recently been brought to light. Up to the middle of this century, though the number of so-called still-born children amounted to 40,000 per annum, no provision was made for regulating their burial. The law has since been amended in this respect by the Burial and Death Registration Act of 1874, but to what purpose we may judge by one report among many others of the same kind. A midwife was brought before the magistrates of Portsmouth, in September 1888, for contravening this Act in the case of an infant at whose birth she was present, and which survived one hour and a half. She placed the body in a box and took it to the cemetery. The clerk of the cemetery gave the woman a certificate of burial ; and it appeared to be his custom to take the unsupported statement of any midwife that any such ' box ' presented did contain the body of a still-born child. Notwithstanding his liability to a fine of ten pounds for such laxity as to the burial of still-born infants, it was admitted that from 100 to 200 such burials are made at this cemetery in the course of the year. Many of these children are neither registered nor buried. A significant fact is brought out by the returns of the Medical Officer of Health of Glasgow, that while of the deaths of legitimate children under five years old only 20 per cent. were uncertified, no fewer than 52 per cent. of the deaths of illegitimate children of the same age were registered without any medical certificate. With regard to the registration of the cause of death generally, the law is still in a most unsatisfactory condition. Within the last four years, the Registrar-General reported that, out of a total of about half a million deaths, the cause of death was vouched for by medical certificates in a little over 90 per cent. of the cases. His returns revealed this remarkable fact, that in no fewer than 20,194 cases in a single year had persons been buried without any certificate whatever of the cause of death. In Ireland, for one year, 4,289 cases are returned in which the cause of death is set down as ' ill-defined or not specified.' The state of matters is still worse in Scotland. In 1882, no fewer than 9 per cent. of the persons dying in Glasgow were buried without medical certificates, and the Report of the Registrar-General (Scotland) for 1884 notes that as many as 20 per cent. of the deaths in that country were registered as uncertified. Under our present system over 80,000 persons die every year in the United Kingdom under circumstances often suggestive of criminal neglect or worse, and are buried without any attempt being made to verify the cause of their death or to make sure that it has not arisen from criminal negligence or foul play.

In France, Germany, and most other countries on the Continent, the certificate of death is prepared and signed by a public officer whose special business it is to make a personal inspection of the body. The mayor of the *arrondissement* in Paris and of the large communes, appoints as *médecin vérificateur des décès* a physician who is not in actual practice. When a death is announced at the Registration Office, he receives notice to go at once to the house of the deceased. There he inquires into all the circumstances

preceding and attending the death, the nature of the illness, details of the treatment, the names and residence of the medical and other attendants, examines the prescriptions, and is empowered, if he sees any reason to suspect that death was not from natural causes, to uncover the body and make a minute inspection of every part, in order that he may decide as to the necessity for an autopsy. These and many other particulars are entered upon a printed form, which has to be signed by him, and returned to the registrar's office. Upon the report, if satisfactory, the Mayor or his representative fixes the time of interment, and grants the indispensable order authorising it. No burial can take place without this formality and official sanction. In some of the German cities, the regulations for the examination of the body, and filling up of the certificates, and orders for burial, are much more complicated and minute. The following quotation of a passage at the head of No. 2 of the Munich regulations makes more striking the contrast between official indifference in England and the consideration given to those subjects elsewhere:—'In order to preclude the possibility of any one being considered dead who is not actually so; that the spread of infectious disorders be avoided as much as possible; that the quackeries so highly injurious to health may be suppressed; that murders committed by secret violence may be discovered, and the perpetrators delivered over to the hands of justice, recourse must be had to a strict medical examination into the deaths occurring, and a conformable view of the body.' Two medical inspections are insisted upon—one immediately after death, the second before the time of burial is decided on. Both the physicians appointed by the police have to sign the certificate of verification, and the medical attendant of the person is required to certify the cause of death. So comprehensive are the precautions taken, that before the first examination the nurses are not permitted to undress or wash the body. The body is not to be moved from the spot, nor the bedding disturbed. Sir C. Cameron's proposal was to make examination by a Government medical officer compulsory in all cases where no medical certificate had been procured; but it was put aside as too costly and too inquisitorial. Our inquests fall far short of this care-taking; and as to burial itself, every recent inquiry further shows that but slight heed is paid to Acts and regulations. Two facts respecting burial are made clear by what has been stated. The law itself is imperfect, and, such as it is, it is disregarded. There is, therefore, a necessity for further legislation, and more severe supervision of the management of cemeteries is required. Whether burial in earth is to remain the national custom, or whether some other mode of disposing of the dead body be accepted, we require that the proceedings should be exactly defined and legalised. If the 'right' of a citizen be that his body should have occupation of a grave till it has decomposed, as seems to be the case, it should not only be so stated, but all companies and persons concerned in the trade of burial should be compelled strictly to provide the conditions necessary for complying with this 'right.' The sojourn in the ground would be much longer than it is at present. More ample space must be provided, and better security given that this right shall not be infringed upon. Inspectorial visits ought to be more frequent. The examination of twenty-five out of all the English burial-grounds in one year appears to have been thought sufficient by the late Home Secretary; but such scant visitation can have but little effect in controlling the action of the managers. Further, as it appears that a man has the power of ordering the disposal of his body by other means than burial, not illegal or creating a nuisance at common law, his right to do so shall be legally declared, and the means employed should be not merely tolerated, but explicitly admitted and regulated. But the most important

step in the advance that it is desired should be made is in regard to registration. Not only do we want to know for sanitary purposes the fact of death and the nature of the disease causing death, when it is what is called natural, in a much more exact manner than at present, but we want also, for social reasons, a guarantee that no person who has died from other cause than disease, or who is presented for burial unaccompanied by any proper medical certificate, shall be buried without due investigation as to whether that cause was accidental or criminal. At present there is but little security on this point for society. Coroners' inquests, though apparently very numerous, do not go far enough. The coroner is not always informed of suspicious deaths, cannot initiate inquiry, and too much is left to the free will of those interested. The fact and cause of death are really matters of police. A medical certificate of the cause of death and the verdict of a coroner's court ought only to be the supplement of a police certificate of the fact of death. Satisfactory certificates are not to be assured by the means we have now at command. A declaration by a nurse, or some unofficial person, and a paper signed by the medical attendant, or not signed professionally in any way, is too often accepted by registrars. Too often, also, the burial is compliantly accomplished without any certificate at all. This loose way of putting a body out of sight is what ought not in any case to be possible, not even in the case of still-born children. It gives too much facility for the perpetration and concealment of crime. Satisfactory authorisation, justifying the disposal of a body, can only be secured by official inquiry and official signature. For this purpose, special medical officers, not in ordinary practice, ought to be designated, whose business it would be, upon order, to visit and examine any dead body, ascertain the fact and circumstances of the death, and report to the controlling authority upon the necessity of further proceedings by inquest, or the propriety of at once disposing of the body. Medical practitioners, however qualified, are in no way part of the police, and the inquiry should be made independently, but in concurrence with them. Their certificates of the nature of disease have reference to other objects than that of burial. To guard against and detect crime is a magisterial duty, and burial or other means of disposal of a body should only be permitted on a magisterial consent, founded upon the report of his special agent. It is, therefore, not an unreasonable demand when we ask for the legal formation of such a precautionary establishment. To say that it is unacceptable, or inquisitorial, would be absurd. Other nations find it a protective arrangement, and regard it with satisfaction. The benefit would soon appear and be acknowledged in England. The Cremation Society of England have already tried the double certificate system, and no difficulty has been found to attend it.

OTHER MODES OF DISPOSAL OF THE BODY AFTER DEATH

We have now to turn to another branch of the subject. The last hundred years have been troubled with three distinct agitations about the dead. The first was the panic raised by the felonious abstraction of bodies from their graves, which lasted till the end of the first thirty years of this century. Legislation, which ought to have been provided long before, when anatomy became a teachable science, put an end to it. Next came, from official disclosures, the disquietude respecting the state of burial-grounds and vaults, and their pernicious influences. For a time suburban cemeteries seemed to meet the difficulty. But before long they began to verify the predictions of the Board of Health Commissioners in 1850, 'that the removal of the existing

evils cannot be secured by joint-stock cemetery companies.' Then followed plans for all sorts of coffins, in stone, marble, granite, slate, porcelain, earthenware, bitumen, asphalt, paper, peat, india-rubber, iron, and glass. Schemes for burying in charcoal, peat, and quick-lime, for plunging in the sea, for enclosing the body in cement, injecting it with antiseptic solutions, desiccating, embalming, electro-plating, and condensing into small cubes by hydraulic pressure, were all tried or suggested, but proved either unsuited to public taste or inapplicable on any commensurate scale. Thirdly appeared that which we are now traversing—the era of proposals for substitutions for burial. The question has turned from pollution to poisoning. If more serious, it is more scientific. The point now to be decided is whether we shall go on perpetuating poisons of which we have cognizance, or whether, by substituting processes more rapidly destructive of the body than burial, we shall check the spread and further the disappearance of zymotic disease. We see our way more clearly in this matter than when we had to deal with smells; and, if we attend to them, the indications we already have will lead to a right and consistent solution. For surely, while we segregate our living small-pox patients and make it a misdemeanour to expose them publicly, we cannot honestly permit them to be treated after death in the very way most likely to multiply and give a chance of future mischief-working to the germs which they contain. Science has not unaidingly alarmed us by its revelations. Its followers have been eagerly seeking the means of enabling us to act rationally and safely. A variety of ways have been pointed out by which the utter destruction of the body and all that it encloses can be effected. For instance, the system of Girard, which reduces a body in a comparatively short time by sulphuric acid, of a certain temperature and density, to a viscid solution (in which, when obtained from charbon sheep, Pasteur has not detected the presence of any germs) does all that is wanted. Its repulsive character, however, and, from its simplicity, the danger of its use by the criminal populations, put it out of consideration, except in veterinary practice. The two processes which have attracted most attention, are free from these objections, and are now undergoing trial, are desiccation and cremation. These are met, on the part of the adherents of burial, by the proposal of Mr. Haden, under what is known as the 'Earth to earth system.' By it, he would hasten all interments, and place the body in as close contact with the soil of a grave as is compatible with the appearance of decency. He calculated that the purifying and transforming power of the earth is sufficient so to 'change the organic into the inorganic, the noxious into the innoxious,' that he should be able by this resolution of the body in five, or at most six years, economically to use the same ground for the same purpose, 'again and again, at such or longer intervals as we please, with no other effect than to increase its substance and to raise its surface.' This is both an admission and a compromise—an admission of the dangers of burial as ordinarily practised by all communities; a compromise founded on a fallacy. The admitted danger would be very slightly mitigated by the compromise. The fallacy lies in considering the bodies spoken of as mere organic matter, and in supposing that all spores of contagious diseases can be destroyed by a basketful of charcoal. Now a body to be buried is not simply dead organic matter. Under ordinary circumstances, it is the abode of myriads of living organisms. These, uncontrolled by life, assist in the process of decomposition, secrete poisons of their own, and are capable of exciting disease if brought by air or water into contact with human beings, a contingency which the periodical reopenings of the graves every five or six years would render very likely. But worse than this, in a considerable proportion the bodies

that are deposited in cemeteries swarm with specific organisms of zymotic diseases, whose spores, often aërobic—that is, defiant of the oxygen in the basketful of charcoal—are almost imperishable in the ground, and ready, after many more years of latency than the stipulated allowance, to revivify and recommence their infective activity. The fact requires no further demonstration. Once accepted, it demolishes all that favours the 'earth to earth' proposal, and shows that with any method of burial short of that causing chemical destruction the most threatening danger would remain as serious as ever. The words of Sir Henry Thompson on this subject are: 'The poisons of scarlet fever, enteric fever (typhoid), small-pox, diphtheria, malignant cholera, are undoubtedly transmissible through earth from the buried body by more than one mode. And thus by the act of interment we literally sow broadcast through the land innumerable seeds of pestilence; germs which long retain their vitality, many of them destined at some future time to fructify in premature death or ruined health to thousands. Especially is this the case in the "earth to earth" system, when the exposure to the soil is instant and complete. The most dangerous elements are no doubt decomposed and rendered less virulent by retention in close coffins for a few years, before contact with the surrounding soil takes place. But the adoption of a system which is designed to hasten dispersion of the elements by any and every channel open in the soil six feet below the surface, so that the same spot may be similarly used after a brief term of years, is fraught with risk to the living. It is vain to dream of wiping out the reproach to our civilisation which the presence and power of these diseases in our midst assuredly constitute, by any precaution or treatment, while effective machinery for their reproduction is in constant daily action.' Recent experiments have led to additional proof of the preservation of the bacilli of typhoid, of phthisis, of yellow fever in the earth, of their latent life until again called into activity, and their terrible potency when cultivated in suitable surroundings.

Desiccation.—The proposal for drying and preserving the body, instead of either burying or burning, comes from America. It is fifteen years since Dr. Bayles, of Orange, N.J., brought the subject before the public. The scheme has recently been taken up by a company of shareholders. It proposes to build a mausoleum in the vicinity of most of the great cities. The building is to be entirely of concrete. The interior plan will resemble that of a library, with its main corridor and diverging halls leading to the different sections. The sepulchres, or compartments, of solid concrete, four inches thick, and large enough to contain a body and casket, are arranged in tiers of six on both sides of the halls. A chapel and family compartments occupy part of the ground floor of the building. A mausoleum one hundred feet by one hundred feet will contain ten thousand sepulchres. The sepulchres are constructed with conduits, so arranged as to bring fresh dry air into them, and conduct it through the casket space by forced drafts to a central furnace in the sub-cellar. To that furnace the gases and vapourised fluids of the body are thus borne and there consumed. The air current makes an entire change in the contents of the casket-space every second. When dry, the bodies are to remain in their separate compartments, as in the catacombs, shut up from the corridor with double doors, the outer one of marble, iron, or bronze, and the inner one of plate glass, through which the body can be seen. A trial of the system was made, commencing in June 1888. Dr. Peacocke, of New York, in a paper printed in the 'Brooklyn Medical Journal,' August 1889, says: 'The desiccation process is now engaging the attention of many sanitarians, and . . . an opportunity was lately afforded me of inspecting and ex-

aming the body of a man undergoing the process. The remains lay in a glass-covered metallic case, having been placed there nine months ago, and at that time weighed 160 or 170 pounds. Judging by the dried-up appearance of the body, I presume that to-day it does not weigh more than sixty pounds. The muscles of the trunk, and especially of the extremities, are shrunken and hard. The integument is dry and leathery to the touch. The countenance looks natural. There is no discolouration of the cuticle, and no evidence of any decomposition. The rapid abstraction of moisture by this method does away with the factors in the production of ptomaines, which might vitiate the result of a chemico-legal examination.' Many favourable opinions are received from America. Nothing is known of this procedure in England, and it is not probable that it will ever be adopted. Practically the expense would exclude it. To replace the cemeteries round about London alone would require, if we take the figures of the promoters, sixty-three acres of concrete buildings, which must be incessantly renewed, since part of the plan is the conservation of the remains in perpetuity. On principle, this is enough to condemn the system, as it would cause us to act as much in direct frustration of one of the primary laws of nature as the Egyptian embalming.

Cremation.—Cremation as a mode of disposal of the dead, though ancient and widely prevalent, has never yet rested upon any sound basis. It has been either the expression of some national belief or superstition, an imposition of priestly domination, the caprice of imperial ostentation, or the impulse of popular panic. The revival of it in England at the present time, and its spread upon the Continent and in America, have quite another character. It is a purely scientific movement. No burial reformers had taken up the cry. They had probed the evils of both private and common graves to the bottom, but they did not condemn the practice of burial. Even official inspectors and commissioners, whose inquiries had given them a fuller knowledge of the subject, were equally possessed with the one idea of burial. The close contact with the miseries and indecencies which it had ended in did not seem to suggest a doubt about it being wrong in principle. Christian teaching, whose founders had adopted it for security, and whose successors had continued it in the interest of their establishment, surrounding it with a haze of mystical doctrines, canonical subtleties, and impressive ritual, had brought them, as well as the nation they represented, into a state of passive assent to its necessity as the only mode of reverently treating the dead. Their advice and action were modelled on these impressions. For a time they seemed to be right. That which had become unendurable close at hand might, perhaps, be harmless a few miles away. But experience proved that if the men of one decade could be contented with Kensal Green, the next must move to Woking, and that beyond Woking there was the probability of encroaching upon another area embarrassed with the same difficulties. The situation was perplexing, but science here stepped in. Men who had reflected upon the relations set up by social conditions between the dead and the living, who, less materialistic than their co-religionists, could divest themselves of false sentiment about remains, and were neither afraid to look at the facts undisguised nor to proclaim their opinions, proposed cremation. They pointed out that the mass of corruptible matter taken away every year by death from among the population, whatever associations hovered about it, was in no way exempt from the universal law of nature, which ruled the dispersion of its elemental constituents; that there was nothing sacred or sacramental in burial apart from what man's ingenuity had invented; that the grave was but a clumsy contrivance to save the feelings of survivors by putting out of sight a grievous spectacle; that, instead of honouring the dead, it was rather dishonouring

their bodies to make them the unconscious and lingering means of harming those whom they had left behind, and on whom, living, they would have shrunk from inflicting the slightest pain; that cremation destroyed nothing more than burial; that it was scientifically analogous to the *eremacausis* of the earth, but that it could be made to bring about the inevitable change and set at liberty the struggling elements of human *exuvie* more quickly, more 'decently' and 'inoffensively.'

These teachings took shape in two papers, published by Sir Henry Thompson in the 'Contemporary Review' for the year 1878. They excited much discussion and criticism, but they really responded to that latent feeling of relief which will always be found in the mind of everyone who has lost a friend, when he is able to persuade himself that the time of the turmoil of decay has passed. There was a promise of relief from that aggravation of sorrow. At the same time a few men of a practical turn of mind were so impressed by the arguments, that they gathered round the author, and in January 1874 formed a society, the object of which was the explanation and propagation of their views, and if possible the revival and perfection of the custom of the cremation of the dead. It was strictly a scientific association. Their primary business was to educate the people, to make clear their legal position, obtain legislation authorising cremation, and to prove the process to be economical and unobjectionably practicable. They had the experience of others to guide them. The burning of Mrs. Pratt, in 1769, on the burying-ground of Tyburn, showed that burning could be done without infringing any law. The two attempts of the French people to legalise the practice proved that an enlightened Government might be approached with some chance of success. The opening of the question in Italy and Germany was encouraging. And there was a lesson in the failure of Mr. Cobbe in 1857. No action was taken after the publication of his pamphlet, and the suggestion, as might have been expected, shared the fate of other vague schemes.

The Cremation Society undertook the spread of information by numerous lectures and publications. These efforts were met by some few prejudiced denunciations; and objections were formulated of at least three kinds, sentimental, religious, and medico-legal. The sentimental outcry came from a class of people so illogical and unreasonable that they were best left to the effect of time and example. Less than twenty years have done a great deal in putting the feeling for the dead upon a just Socratic footing. In that time the apparently valid objections have dwindled to spectral forms. Divines themselves have exorcised the doctrinal and ritual difficulties. They are represented in the Council of the Society. One bishop of the English Church has declared that 'no intelligent faith can suppose that any Christian doctrine is affected by the manner in which, or the time in which, this mortal body of ours crumbles into dust and sees corruption.' Another, a Canon of St. Paul's, said: 'The resurrection of the body from its ashes is not a greater miracle than the resurrection of an unburnt body; each must be purely miraculous.' English, German, and Italian priests have sanctioned cremation by their presence at the process, and have found no obstacle in modifying their ritual to the occasion. The Roman Office for the Dead is equally applicable, and Catholic priests, even in Rome, have chosen cremation as their mode of sepulture. The process has been judicially declared in Great Britain 'not illegal.' The crucial criticism about the 'annihilation of exhumation'-evidence has been neutralised, for the time being, by the action of those who are responsible for cremation. The refusal of the Council of the Cremation Society to deal with any body of which the death is not certified as from

natural causes by two registered medical witnesses shuts out from the crematorium any dead body which might be required as evidence of crime. It only remains for our legislators to legalise and regulate that which they allow to be done here, and actively promote in the Indian part of our Empire, and, by amending the Registration Acts, to give to the public at large security the same as that offered by the cremationists to that section which trusts to them. The necessity for exhumation occurs too rarely now for it to be seriously urged as an argument against cremation, and with the indicated precautions would scarcely ever present itself. Inquiries made by Sir H. Thompson bring out the information that for the last twenty years the mean number of exhumations made in a year throughout England and Wales is only five, and less than one yearly for poison. Practically there are only four common metallic poisons—arsenic, antimony, lead, and mercury—in respect to which the question of exhumation could be usefully raised, and two of these would be found in the ashes as well as in the ground. Some of the vegetable poisons and alkaloids are so rapidly decomposed or so mingled with other generated poisons, in the midst of putrefaction, that post-exhumation evidence of their presence, of a character certain enough for judicial acceptance, is scarcely more available than if the body had been exposed to fire. In every case where there is reason to doubt, either as to the cause of death or as to personal identity, it would be wiser on the part of the authorities to impound the body, or parts of the body, and to use for them desiccation or some of the known means of preservation.

The first step taken by the Cremation Society to assure themselves as to their position was to obtain the opinions of eminent counsel as to the state of the law on the subject. These opinions went to show that there was nothing in the English law forbidding cremation, provided it was conducted in such a manner as not to involve any public nuisance. Lord Chancellor Selborne and Sir William Harcourt confirmed this opinion by letter. Thus fortified, the Council proceeded to action. After an abortive attempt, which caused much delay, to procure in the Great Northern Cemetery a place for the erection of a crematorium, ground was found about a mile from the Woking Railway Station, and the construction of the building was completed early in 1879. The working of the apparatus was satisfactorily tested upon the carcases of animals, but the intention of the Society to answer to the demand of those who wished to cremate relatives was twice obstructed by successive Home Secretaries, who threatened adverse legislation, and exacted a promise from the Council that they 'would act in strict conformity with the wishes and directions of the Government in the matter.' This promise, however, was not binding upon private persons, and Captain Hanham, of Manston House, Dorset, having in his private mausoleum the bodies of his wife and mother, which he wished to burn in compliance with their request, had a crematorium set up in his own grounds, and there carried out their wishes. He himself did not long survive, and his body was burnt in his own furnace, and in the same manner. Although the utmost publicity was given to these operations, the Home Office took no action. Meanwhile, an occurrence took place which solved the legal question independently of any action on the part of that department or of the legislature. A certain Dr. Price attempted to burn the body of his child, not having registered its death. An inquest was held, returning a verdict of natural death. Dr. Price was committed for trial, and indicted first for having prevented the holding of an inquest, and, secondly, for having attempted to burn the body. The case came before Mr. Justice Stephen, at the Crown Court, Cardiff, in February 1884. Addressing the grand jury upon it, the Judge read a charge in which

he entered elaborately into the whole law of the subject. The following quotations from this document made clear the legal position of the Society and set at liberty the hands of its Council:—‘The great leading rule of criminal law is, that nothing is a crime unless it is plainly forbidden by law.’ ‘The burning of the dead has never been forbidden, or even mentioned or referred to, so far as I know, in any part of our law.’ ‘I am of opinion that a person who burns instead of burying a dead body does not commit a criminal act, unless he does it in such a manner as to amount to a public nuisance at common law.’ ‘I have been unable to discover any authority for the proposition that it is a misdemeanour to burn a dead body, and in the absence of such authority I feel that I have no right to declare it to be one.’ ‘I think that to burn a body decently and inoffensively is lawful, or at the least not criminal.’ Dr. Price was acquitted. In the following month, March 1884, the Council of the Society decided upon the execution of their project, and issued a notice that their crematorium could be used by the public under certain regulations. The conditions were expressed in the following terms:—

1. An application in writing must be made by the friends or executors of the deceased—unless it has been made by the deceased person himself during life—stating that it was the wish of the deceased to be cremated after death. They must furnish the name of the medical man who has attended the deceased, in order that he may receive an official communication from the secretary before certifying.

2. A certificate must be sent by a qualified medical man, who, having attended the deceased until the time of death, can state without hesitation that the cause of death was natural, and what that cause was. Another qualified medical man, if possible a resident in the immediate neighbourhood of the deceased, is also required to certify, after independently examining the facts within his reach, that to the best of his belief the death was due to natural causes. To each of these gentlemen is to be forwarded, before certifying, a letter of ‘instructions,’ marked ‘private,’ signed by the president of the Society, calling special attention to the important nature of the service required.

3. If no medical man attended during the illness, an autopsy must be made by a medical officer appointed by the Society, or the cremation cannot take place, unless a coroner’s inquest has been held and has determined the cause of death to be natural. These conditions being fulfilled, the Council of the Society still reserved the right in all cases of refusing permission for the performance of cremation if they thought it desirable to do so. The subject was now fully before the public. It responded immediately. New provincial societies were formed, a large correspondence ensued, and applications were made for the use of the furnace. The Woking Crematorium was employed for the first burning of a human body on March 20, 1885; two other cremations following in the course of the year. During 1886 ten bodies were burned; in 1887, thirteen; in 1888, twenty-eight; in 1889, forty-six; in 1890, fifty-four; in 1891, ninety-nine; and in 1892, 104, with thirty-eight up to May 1893, making a total of 395 since the commencement in 1885. All this had been done with the law remaining as it was at first; for though, in 1884, Dr. Cameron, one of the members for Glasgow, introduced into a bill which he laid before the House of Commons clauses making cremation a legal way of disposing of the body, they met with Government opposition, and the bill was rejected. A minority of seventy-nine members voting with him, however, put an end to all possibility of carrying any prohibitive measures.

Cremation, therefore, as it is practised at Woking, maintains its position

of non-illegality. The opening of a crematorium at Manchester, the erection of a columbarium in Kensal Green Cemetery, and other circumstances, give every reason to conclude that old prejudices are dying out, that the sanitary benefits are becoming appreciated, and that a rapid extension of the practice may in coming years fairly be expected. That such may be the case is most earnestly to be desired in the interests of morals, health, and economy; and it is not unreasonable to anticipate that Government may recognise and regulate cremation, as well as enforce the rule of single-grave burial upon those who still prefer burial in the earth. Without some such manifestation of wise regard to the teachings of modern science, we shall justly incur from the generations to come, not only the feeling of regret we have when we think of the ignorant adoption of earth burial by our forefathers, but a censure for blind, culpable indifference to our own safety and the future well-being of society.

Arrangements at Woking.—The system adopted for the crematorium at Woking is that of Gorini, which was considered best for the site, inasmuch as no supply of gas is required to ensure combustion, but only wood and coal. The receptacle for the body is a flat-bottomed chamber, open at each end, and heated directly from the furnace. When the door separating the furnace from the crematory chamber is opened, the flames play over the body, and all evaporated matter passes through a second chamber and a series of intensely heated flues to the external chimney. Near the bottom of this chimney is placed a grating upon which coke is kept burning, and here every product of combustion is subjected to a still higher temperature before it issues from the upper end of the shaft. The fumes which escape and mix with the atmosphere are purely gaseous. There is no admixture of smoke, and so perfectly is every particle of organic matter consumed, that analysis of a collection made at the mouth of the chimney during the full action of the fire upon a body proves that nothing more escapes into the air than a vapour containing about 4 per cent. of carbonic acid, 16 of oxygen, and 80 of nitrogen. When any black or visible smoke escapes, as it does occasionally on first lighting the fire, before the furnace is heated for the body, it is only a proof of want of care in the stoker.

A chapel or hall, which can be used for religious services, and suitable accommodation of rooms for attendants at the ceremony, have recently been added to the crematory apparatus. The time usually occupied by the process varies from an hour to an hour and a half, after which the ashes are ready for delivery to the friends. Provision has been made on the property, both for the burial of the ashes in the ground and for the preservation of cinerary urns in the chapel. It is still, however, an open question what is best to do with the ashes. Enclosed in urns, they may form a valued and venerated relic, and if buried in churches, or even in cloisters around them, or exceptionally beneath the floors of our cathedrals and abbeys, they would occupy very little space and remain perfectly harmless for generations. Some would have their ashes scattered to the winds. But where? They might be offensive to someone in their flight, and in the end must come to the ground. The earth, then, is their natural destination, and it seems probable that hereafter they will either be buried, or preserved, or taken to some family grave or churchyard, and there laid to commingle with the soil of which in a short time they would become an undistinguishable part, ready for new transformations and incapable of injury to the living.

Cremation Abroad.—Less difficulty has been experienced elsewhere than in England in reconciling public opinion to the substitution of burning for burial as a means of disposal of the dead. America has so far adopted

the practice that seventeen crematories had been erected in some of the principal cities, and the remains of more than 2,800 persons have been cremated in them up to 1891. As the law in some of the States provides only for the disposal of the dead by burial, efforts are being made to have it so altered as to leave the people free choice between cremation and burial. Most of the American crematories are the property of joint-stock companies, which, to avoid all medico-legal objections, have made very strict regulations.

Cremations began in Germany at Dresden in 1874, one of the bodies burnt being that of an English lady. Societies exist in Berlin, Hamburg, and other places, but the greatest activity is shown at the small town of Gotha, where the Siemens gas apparatus has been chosen by the municipal council, and has served for over 800 incinerations.

The subject had been for many years a matter of discussion in Italy. Coletti began the movement in 1857, but political troubles prevented much attention being given to his addresses. In 1869 the Medical Congress at Florence passed a resolution unanimously approving and recommending cremation. A law of sanction was obtained for it in 1873. Baron Keller, dying in 1876, left a sum of money for the burning of his own body, and for the erection of a crematorium in Milan, of which city he had been an inhabitant. Two Gorini furnaces are at work in it, and many bodies are consumed every year. It was at this institution that a case of accidental poisoning was discovered by the stricter certificate required for cremation, which would have escaped notice had the child been buried in the cemetery with the usual certificate of the cause of death. Similar buildings have been constructed at Rome and many other Italian cities, and the return in 1889 gave a total of 788 cremations in Italy—a number largely increased since.

The spirit of doubt and inquiry which had sprung up in the French mind during the latter half of the last century, and which led to the emancipation of the people from so many of the abuses of the old *régime*, was not confined to constitutional questions. There were among the agitators social reformers and sanitarians as well as politicians and adventurers. When the Church fell, all things which had been under ecclesiastical control came in for their share of scrutiny and depuration. Grave-yards in France were in no better condition than those of England. Complaints of long standing had been met by a neglected decree of Parliament in 1765, ordering the cessation of burials in all towns and churches. Under the Directory, considerations of public health led to a demand for a thorough reformation in the matters of funerals and cemeteries. It was not surprising that, with the rage of imitation of what was supposed to be ancient republican manners, among other projects for the safe disposal of the dead cremation should be the leading proposal. It was partially admitted in the Department of the Seine. The administration had the intention of opening *un champ de repos* at Montmartre, where bodies could be either burnt or buried at discretion. The prefect, upon the demand of the parents, conceded authorisation to burn the body of a child. 'He could not, without violating the principle of freedom of opinion, enforce any particular mode of doing what was a religious duty.' But the child was never burnt, for discussion had lingered and was prolonged till the Church, accepting a position as part of the machinery of imperial government, was re-established. After this, mention of cremation ceased. During the early years of the Second Empire the subject was ineffectively revived. Public feeling was not roused till after the succeeding Government had been obliged to accede to the burning of the bodies

left imperfectly buried about Sedan in 1870. Many determined attempts were made to bring this feeling to some practical issue. It was not till 1880, however, that a cremation society was formed. A crematory of considerable size, erected by the Municipal Council of Paris in the upper part of Père la Chaise in 1887, has been brought into use under authorisation of the Burial Law of that year. Unfortunately, cremation is regarded by the City Council as a purely civil act. Their apparatus is devoid of any religious emblem, and was at first unprovided with accommodation for religious service. Though no great difficulty had been met with in obtaining from the legislature a facultative position for cremation, Article 20 of a decree of the Conseil d'Etat distinctly declares that the ashes of a body cannot be deposited anywhere, even temporarily, but in a regularly established place of burial. Nor can they be displaced without an order from the Municipal Council. For the present, those in urns are kept in a large vault at Père la Chaise, arranged for the purpose, till the columbarium is completed. The unclaimed ashes are packed in boxes and laid in the *fosse commune*. Four Gorini furnaces were first set up in the building at Père la Chaise, and many bodies have been burnt in them. In December 1888 the matters escaping from the chimney were examined on an occasion when the bodies burnt weighed 173 kilogrammes. Four kilogrammes of wood were used for burning the bodies, and coke for consuming the smoke. Gases were collected at two different stages of the operation, and upon analysis were found to consist of—

Carbonic acid	3.47	4.16
Oxygen	16.20	16.15
Nitrogen	80.00	79.68
	<u>99.67</u>	<u>99.99</u>

These furnaces have lately been suppressed on account of their imperfect action. A new apparatus, the invention of Messrs. Toiseul and Fredel, has been substituted. In this the gas producer is on a lower level than the chamber for the body, to which the incandescent gases are conducted through a recuperator, heating the air which they take with them. The smoke is not burnt and escapes by the chimney into the open air. From August 12, 1888, to December 1889, 735 bodies were burnt, of which 37 were of persons not sent from the hospitals and dissecting rooms. At one time the men were at work night and day. One of the latest reports states that the number of bodies burnt now amounts to more than 4,000.

Cremation has made considerable progress in the Colonies and in other European States. In most of them, Russia and Turkey excepted, societies have been formed, or the subject has been brought under notice, with every probability of the custom of burning the dead eventually replacing that of burial. After more than sixteen years of earnest and disinterested work in the propagation of knowledge, seeking a revision of our burial laws, obtaining more exact proof of the real cause of death, practically proving the possibility of decently and inoffensively using cremation, and putting at the command of the public the means of employing it without much expense or inconvenience, it is no slight satisfaction to all who, as founders and supporters of the Cremation Society of England, have been so engaged to find that, in spite of difficulties and discouragements, their efforts are likely to end successfully, and that this and other countries will be freed from an ever-threatening danger of modern civilisation, by a general and reverent adoption of what has proved to be the safest and most rational manner hitherto practised of disposing of our dead.

One or more generations of men may disappear before the burial of ashes

instead of burial of the entire body becomes general in our country. Until that time comes, it will be the duty of every medical officer of health not only to make himself acquainted with the bye-laws of the Local Government Board for the sanitary requirements of cemeteries, but to exert his influence in enforcing obedience to those requirements. It is to be hoped that the regulations of the Cremation Society of England will be approved by our Government, and that cremation will only be permitted when such regulations are obeyed and acted on.

II

THE preceding paper on disposal of the dead considers this subject from the point of view of those who see difficulties and dangers in the customary method of interment. This view is in no way universally shared, and it is urged on the other side that, if proper care be exercised in the selection of the site of the burial-ground and in its subsequent management, these difficulties and dangers can be obviated. The destruction of the body by fire gives opportunity for the concealment of crime, from which interment is free.

Under these circumstances, it is desirable that the volume should contain a more detailed account of burial in the earth as a means of disposal of the dead, and of the conditions which should be observed in the management of burial-grounds.

The practice of burial in the earth as a means of disposal of the dead has been put upon its defence by two such eminent surgeons as Sir Henry Thompson and Sir Spencer Wells, who have succeeded in winning to their side a considerable number of followers. They have condemned burial *in toto*, as in every respect bad, polluting the earth, the air, and the water-supply, propagating diseases by the bacilli arising from the buried corpses, and as being altogether indefensible. Mr. Seymour Haden and Dr. Vivian Poore, two other distinguished members of the medical profession, have taken up the defence of burial, and have also a large following. They have condemned cremation as absolutely as their opponents have condemned burial, alleging that the crematory furnaces are much more likely to poison the air than burial in the earth; that cremation is not a perfect process, since it leaves a not inconsiderable weight of ashes as a residuum, and, which is the greatest objection of all, that cremation, by removing all possibility of exhuming the body, has a direct tendency to defeat the administration of justice, and to encourage criminals in their guilty course. Following at a most respectful distance such eminent men, our course must continue to be, as it has been throughout the whole of this controversy, a middle one. In both modes we can see merits and demerits, while in the arguments used by the respective champions on either side we can see errors. Our principal reason for taking any part in the discussion is the experience we have gained of burial, in witnessing the removal of portions of five burial-grounds, one of which we shall describe, with the exhumations of hundreds of corpses (speaking quite within compass), and subsequently of four bodies for medico-legal purposes. Moreover, the city of Liverpool is distinctly one where the burial of the dead acquires a special interest.

Bounded on the west by the river Mersey, it has spread north, south, and east with a rapidity which is almost, if not quite unique. From being a poor fishing village at the commencement of Lord Macaulay's 'History,' with a population of 4,000, and having only one chapel-of-ease, whose churchyard was the only burial-ground, its population rose in the century following to 40,000; while at the present time it is rapidly approaching 600,000. Many intramural churchyards, after being filled to repletion, and some even beyond this, have long since been closed; country churchyards, though considerably increased, are yearly becoming less capable of meeting the demands made

upon their space ; while six extramural cemeteries, varying in extent from fifteen to 120 acres, present appearances of occupation with the dead buried in them which give rise to serious doubts as to whether they will suffice for the present generation. It is true that land could be obtained north, south, and eastwards at the distance of some miles from the city ; but land is required for the living rather than for the dead. And we are perfectly satisfied that before ratepayers will consent to the purchase of large portions of land, bought at a fancy price, they will insist upon the burial of the dead in the future being conducted on reformed principles, with an entire absence of any undue monopoly of the ground by individual purchasers. Moreover, as cremation grows in favour with at least a section of the public, it will be imperative for some burial boards to provide facilities for this mode of disposal of the body in some cemeteries. It is not, however, with cremation but with burial that we have to deal, and we pass on to describe some of the abuses of burial, as we have witnessed them, side by side with burial as it ought to be. For in this, as in other matters, there has been always in this country a tendency to reform—an assertion of sturdy British common-sense which has generally been ready to pioneer the way towards rectifying abuses and supplying in their stead judicious changes.

In 1869 we witnessed the removal of a large portion of St. Ann's Churchyard, Liverpool. This church had been built in 1772 on a morass somewhat difficult of approach and quite extramural to Liverpool as it then was. Rapidly it was surrounded with houses, which were at first tenanted by the wealthy citizens, next by the tradespeople, and, after being divided into several different parts, they gradually passed into the 'slums.' For a period of sixteen years before the removal of the churchyard there had been no interments ; but from its opening in 1772 until its closure in 1858 by Order in Council, a considerable number of bodies had been buried, comprising individuals of all classes above the rank of paupers, who were buried elsewhere. Many of them must have died from infectious diseases, there having been two severe epidemics of cholera during its eighty-one years of usage, and other epidemics of typhus, small-pox, &c., and yet, though the neighbouring houses were mostly separated from the churchyard wall by a very narrow street, and on one side they actually touched the wall itself, the health of the inhabitants was not worse than that of the residents in any other 'slummy' locality. However, there were no reports of any illness caused by the exhumations, which lasted for some months ; and though, of course, steps were taken to limit the number of onlookers to a select few, nothing could be done to prevent the inhabitants from crowding round the gate, trying to see what they could, and watching the departure of the carts containing the coffins. The majority were taken to Anfield Cemetery, the cemetery proper of Liverpool, which contains 120 acres, two-thirds of which have been laid out for burials, and in which a very large number of human remains were re-interred in 1864. The bodies of some individuals were removed by their friends to other burial-grounds. During the whole of the exhumations the health of the men did not suffer, though they complained very much of the effluvia at times, which was most offensive. One accident occurred, resulting in a slight scalp wound, from a falling plank, but this was the only one. During these months we had time and opportunities to notice the following interesting facts.

In some parts the ground was very dry, and here the bodies were found reduced to skeletons, there being no trace of grave-clothes nor of coffin, except the metal handles, plate, &c. We have already mentioned that there had been no interments for sixteen years ; hence it will be seen how long the skeleton will remain even in a dry soil. There had evidently been no lead used in

these cases ; but wooden coffins will remain intact much longer than is generally believed, and we are convinced that it was not until some years after burial that the body was permitted to come in contact with the earth. We see here, however, that the soft parts will disappear in time in a dry soil, leaving only the skeleton, which no doubt will itself disappear in the course of years.

Small bones, evidently those of newly-born infants, were found buried very superficially and in more or less perfect preservation—another fact showing that the disintegration of the bony skeleton is a work of time. In one case we found a skull with the calvaria neatly sawn through, evidently from a *post-mortem* examination. In another, a very perfect specimen of thyroid cartilage was found. In many instances skeletons were found one lying above the other with earth in all the cavities, and only a very thin layer of earth between. There were many opportunities of observing the differences of sex, as shown by the pelvis, and the great stature indicated by the length of some of the bones.

Very different was the sight presented by the vaults and bricked graves. Their construction was good, better indeed than that of many houses for the living. But this superior construction was a source of danger, since it permitted the retention of water, with which some of them were almost filled. When this was removed, the coffins were seen piled one over the other, the lower ones crushed almost out of shape by the superimposed weight, and the small vacant space covered with the *débris* of the outer cases—oak or elm boards covered with what had been black cloth or velvet now a mass of sodden, tattered rags. The lead had in many instances given way at the soldering over the lid of the inner coffin. When this was intact it was not disturbed ; but when moved it was evident that the contents were a mass of liquids and solids only partly decomposed, and the effluvia was sickening in the extreme. Fortunately, the space in which the men worked was ample, and, as only a small portion was done at a time, the danger was minimised. In those cases where the inner shell had given way the sight was extremely repulsive. The contrast between decomposition as it takes place when the body is brought into contact with the earth, as has been described, and when it is tightly boxed up in a coffin which has for years been soldered up in lead, is very much to the condemnation of the latter. This is distinctly a subject of interest and importance to medical officers of health. That a comparatively thin layer of suitable earth is a perfect deodorizer of the effluvia arising from a corpse has been and can be proved beyond all doubt. What is much more to be dreaded is the concentrated gas retained in the inner coffin gradually oozing through minute apertures in the solder of the lead-chinks in the outer case and cracks in the cement into the open air. That it may seriously affect the health of those attending a funeral or passing through a churchyard or cemetery seems to us to be undeniable.

But there is another aspect of this mode of burial which deserves the most serious consideration. All the bodies exhumed from this churchyard were reinterred exactly in the same manner in the cemetery to which they were removed—those taken from graves being buried in graves ; those taken from vaults and bricked graves being placed in similar structures specially prepared for them. A very serious principle is here involved. The churchyard was attached to a district church of the parish of Liverpool ; the cemetery was purchased by the ratepayers. These bodies had, with their imperishable coffins, vaults, and walled graves, occupied the ground in the churchyard for periods varying from sixteen to eighty years. They were then removed to the city cemetery, not only to occupy, but to encumber the

ground for an indefinite period. It is no answer to urge that compensation has been given in the form of an increased sum for the purchase of the vault or walled grave. No amount of money could recoup the ratepayers for such perpetual monopoly of the ground paid for out of the rates; while, if in addition to this such form of burial be, as we contend it is, dangerous to the living, there can be no justification of it whatever. We are not going to suggest that the bodies should have been removed from the lead coffins, and that these latter should have been destroyed: such would be an outrage. But these exhumations of bodies entombed in lead and placed in vaults many years ago are occurring with painful frequency in London and elsewhere. While we write, preparations are being made for the removal of some two hundred bodies, almost all buried in this same way, from a churchyard in the very heart of Liverpool to a suburban cemetery. Similar proceedings are taking place with respect to bodies entombed below churches in the City of London during the last century and the early part of this. In other words, those cemeteries which were rendered necessary by the wholesale closing of intramural burial-grounds in 1853, and which were purchased at an enormous cost, have to receive the remains of the past generation; even of those who died a century ago! And another very serious question arises. Will these cemeteries be any more inviolate than the consecrated churchyards — 'the God's acres' — of the past? If these latter may be bought by city corporations for the purpose of widening a thoroughfare, making a new street, or any other improvement, and if the dead can be removed to a cemetery, why should not this be repeated years hence in the case of the cemetery? For every suburban cemetery forms at once the nucleus of a suburb, village, or town, and it is quite impossible for them to remain suburban long. Will railways be allowed to pass through them? And will local authorities be permitted to acquire this, that, or some other portion for local improvements, the dead being removed to some other distant cemeteries? The prospect is not a pleasing one, and we allude to it only to show what we must press for if burial is to continue as the more general mode of disposal of the dead. It is burial in the earth pure and simple, forbidding the slightest deviation from this, and requiring the use of readily perishable coffins. In this there is nothing which can be called exacting or unreasonable. The law, as laid down by Lord Stowell more than half a century ago, is that every parishioner has a right to be buried in his parish churchyard or cemetery, but that he had no right to have buried with him a huge trunk or chest, descending with him into the grave and remaining till its own decay. That is no part of his right, which, strictly taken, is that his body shall be carried to its parent earth for dissolution, and that it shall be carried thither in a decent and inoffensive manner.

That this legal dictum exactly accords with the sanitary science of the present day must be obvious to all who are engaged in matters relating to health. The Order in Council which virtually prohibited intramural burial after the year 1853 was rendered absolutely necessary by the overcrowded condition of the church- and chapel-yards; the mode of burial in former years having been that known as 'pit burial.' Bodies were buried in layers ranging from six to twenty, a similar number being buried over them with a very slight layer of earth, and the process was repeated until the last layer was within a very short distance from the level of the churchyard. Bodies buried like this were exhumed from the old parish churchyard of St. Peter's Church and removed to Anfield Cemetery, the superintendent of which informed us that the bones were so thickly clustered together as to resemble sticks in a bundle. This would not have been had the soil been suitable and had a

reasonable portion of it been allotted to each body; since in that case there would have been only traces of bones, many of the bodies having been buried more than a century and a half. These experiences of the past teach us to avoid the two pernicious extremes of burial—(1) hermetical sealing of the body, as in lead coffins and cemented vaults; (2) overcrowding of bodies together without a sufficiency of earth for each, as in the pit burial of paupers, a practice which is not wholly extinct. The happy medium, burial in a perishable coffin and in a sufficiency of suitable earth, has been found a perfect mode of disposing of the body in the past; it ought to be our guide for the future.

It is refreshing to turn from these sights to our extramural cemeteries of the present day, which have been purchased and laid out with due regard to suitability of site, of soil, and other requirements of the times, and the surroundings of each cemetery. We may judge the capacity of the earth for receiving the bodies of the dead in the future by a glance at the past. Throughout the length and breadth of the United Kingdom there are hundreds of village churchyards in which the dead have been buried for centuries, yet there is still room for more, and they are still used. In the near vicinity of Liverpool are the churchyards of Walton, Childwall, and Sefton. The first received the dead from Liverpool as well as those from its own vicinity till the seventeenth century, and, though enlarged, there is still room in the ancient part. Sefton Churchyard has been in use since the twelfth century, and Childwall since the fourteenth. In them have been buried the bodies of victims of the plague, the cholera, small-pox, and fevers of all kinds. Were there any truth in the supposed dangers from the bacilli arising from these bodies, these burial-grounds ought long ago to have been noted as foci of disease, whereas their localities are all models of health. For a long time the burials were comparatively few, and up till the middle of last century were probably for the most part of the simplest character. Indeed, in the times when the 'black plague,' *Sudor Anglicanus*, or 'sweating sickness,' as it was variously called, was epidemic, coffins were dispensed with altogether, and so also in epidemics of small-pox and cholera. We thus see, side by side as it were, burial as it should be and as it should not be. The evils of disposing of the dead as shown by the description we have given were caused by the short-sighted policy of the former generations, who persisted in these urban burials, though they saw the town extending all round them, ignoring the excellent example of the Jews of old, who buried their dead outside the city; and consequent upon this want of foresight acre after acre of ground was taken, was filled to, and even beyond repletion, being then closed because it was absolutely necessary. In addition to the churchyards we have named, there were between thirty and forty other church- and chapel-yards, and three parish cemeteries, in all of which were every one of the abuses we have indicated: vault interment, overcrowded graves (for there was no foot of earth between each coffin in those days), ground unsuitable for burial purposes, pit burial—in short, every insanitary condition.

Now, although, as we have already observed, the mere fact of the dead being buried even in large numbers in any particular spot, assuming that they are all properly buried, is not prejudicial to the health of those living near, there can be no doubt that the intramural burials were a great aggravation of Asiatic cholera, of which Liverpool had visitations in 1834, 1849, and 1866. In the first of these years, the deaths from cholera were 1,523 in a population of 280,000; in 1849 they numbered 5,281, the population being then 376,000. Four years later came the Order in Council which closed absolutely most of

the town burial-grounds, and placed great restrictions upon all the others; this was followed by the formation of large extramural cemeteries in various suburbs. And when the cholera again appeared in 1866, the town having then a population of 478,000, the deaths numbered 1,782. It must be remembered that at that date the powers now possessed by local authorities of removing to hospital compulsorily those infected with this disease, and of conveying to a mortuary the bodies of those who had died from it, did not then exist. The good effects of forbidding intramural burial in reducing the mortality from cholera must therefore be admitted.

In all our modern cemeteries the suitability of the soil and site is guaranteed by a Government medical inspector as well as by local officials, who are now well aware of the importance of such details. The proper drainage is secured, and the contamination of water rendered impossible. The rights of householders are protected by the Act which forbids any cemetery being made within one hundred yards of any dwelling-house without consent; but it is very remarkable that most cemeteries, however isolated they at first may be, are gradually made to form the nucleus of a new centre of population. Residences must be built for the officials, and these are generally within the cemetery itself, or some within and some without. A sculptor's and statuary yard is a more or less necessary appendage to a cemetery, and one is generally to be found near at hand. Other requirements follow, and it is next to impossible for any cemetery to be absolutely extramural for long after its opening, though, fortunately, it can never become so intramural as those of old. These facts ought to induce Burial Boards and those in charge of cemeteries to conduct them as perfectly, sanitarily speaking, as is possible.

For our cemeteries will be of great value to the cities, towns, and villages near which they lie, if they are made suitable recreation grounds for the residents, a conglomeration of flower gardens and flower beds, delighting the eye of the jaded dweller in town, and presenting even in winter a green oasis in the desert of bricks and mortar. It is really surprising how trees, shrubs, and flowers will grow even in those cemeteries of the past, which were less carefully selected than those of more recent date; these leave little in this respect to be desired. For what were formerly dreary plains or vast tracts of barren soil are now cemeteries full of verdure, with broad level walks and every requisite of a well-kept recreation ground. It only remains to note what improvements are required to make them, as Lord Stowell said they should be, not only the cemeteries of this generation but of all to come.

1. Catacombs, vaults, bricked graves, should be absolutely forbidden. So far from there being anything unreasonable in this, reason herself demands it. All past experience shows that such a mockery of burial is unseemly to the dead, an outrage on Nature, and a very probable source of danger to the living. The dead outnumber the living to an extent hitherto unknown, and if such encroachments on cemetery space continue much longer, they will have to be forbidden by law. How much better that Burial Boards should voluntarily pave the way by putting a deterrent price upon vaults and by cheapening that of graves!

2. Every encouragement should be given to purchasers of graves by selling them at reasonably low prices for cash, singly or in plots according to the size of the family; or families, should two or more become joint purchasers. We are not going to suggest that they should become monopolists, far from it. Let us suppose a grave-plot of six graves ([1] [2] [8] [4] [5] [6]) taken by two families. Now, when the first death occurs, grave No. 1 or 6 can be used. If the soil be suitable and the coffin perishable, the sinking of the mound at the top will show that the body has been restored to the

we must reiterate our conviction that, if burial is to continue as the more general mode of disposal of the dead, it must be conducted in each and every case on the principles which we have indicated.

The following summary of a 'Memorandum on the Sanitary Requirements of Cemeteries,' issued by the Local Government Board in 1888, embodies the views of the official medical advisers of the English Government as to avoidance of dangers to health by cemeteries.

The dangers to the public health to which places of burial may give rise are of two kinds—viz. the contamination, first, of *air* by the gaseous and volatile, and, secondly, of *drinking-water* by the liquid and soluble products of decomposition.

1. *Contamination of Air*.—The gases evolved from putrefying bodies may make their way to the surface through pores or fissures in the ground, or may pass into open graves dug in their neighbourhood. Or they may diffuse themselves laterally through the ground air, and may be given off from putrid drainage water, whether baled out of graves and thrown upon the surface, or draining into open channels and water-courses. Thus nuisance and danger to health may be occasioned, not only to grave-diggers and persons attending funerals, but also to the inhabitants of houses in the neighbourhood of the burial-ground. To obviate these risks it is necessary that the number of decomposing bodies in a given portion of the ground should not at any time be so great that the gaseous products cannot be utilised into harmless substances in the interstices of the soil, or taken up by vegetation; that a sufficient depth of earth intervene between corpses and the surface; and that the soil be of a suitable nature and properly drained, the drainage water being innocuously disposed of. To avoid aerial danger, it is necessary that the place of burial should be in an open situation and at a sufficient distance from dwellings, in order that any effluvia arising from it may be diluted by diffusion, or dispersed by the winds, so as not to find their way in an injurious state of concentration to places where they will be liable to be inhaled.

2. *Pollution by Water*.—The percolation of foul liquids from graveyards into wells may occasion injury to health. The liability of wells to pollution depends partly upon their proximity to the graveyard and partly upon the configuration and geological structure of the ground. In order to obviate risk from this cause, it is necessary that a cemetery should have a suitable soil and be properly drained, and that it should be at a sufficient distance from subterranean sources of water-supply, and in such a position with respect to them that the percolation of foul matters from one to the other may be impossible.

Having regard, then, to—(1) suitable soil, and proper elevation of site; (2) a suitable position; (3) sufficient space; and (4) proper regulation and management, the Board advises that—

1. The soil of a cemetery should be of an open porous nature, with numerous close interstices; easily worked, yet not loose; free from water or hard rock to a depth of at least eight feet; and sufficiently elevated above the drainage level of the locality. Loam and sand are the best; clay and loose stones the worst soils.

2. It may be taken that a distance of 200 yards is amply sufficient to prevent any injury to health from a well-kept cemetery, so far as regards noxious matters transmitted through the air. The Burials Act of 1855 prescribes 100 yards as the minimum distance between new burial-places and human habitations; the Cemeteries Clauses Act, 1847, 200 yards. In France the prescribed distance is 100 metres (109 yards).

It does not, however, appear that the amount of danger to health to be feared from proximity to a well-kept cemetery is large. Since intramural interment has been abolished, recorded cases of injury to health, or even of nuisance arising from graveyard emanations, whether conveyed by air or water, are extremely rare. Sir Charles A. Cameron shows that the amount of organic matter deposited yearly in a well-kept cemetery is less than the amount removed from its surface in the form of vegetation, and considerably less than that spread as manure over a cultivated field. There is, therefore, no reason why a cemetery should necessarily be a nuisance, or become a source of danger to the health of those living near it. Nevertheless, it is desirable that a belt of ground should be preserved between the graves and the nearest land on which a house may be built, to obviate the risk of contamination of ground air and subsoil water. The burials should not take place within fifteen to twenty feet of the cemetery boundary. This is especially necessary when the houses are constructed with cellars.

The drainage of a cemetery should not be allowed to enter a stream from which water is drawn for domestic use. It does not appear that the risk to which wells are exposed from the proximity of a properly managed cemetery is in ordinary cases great. A leaky cesspool is a far greater source of danger than a grave. The solid and liquid excretions voided by a human being in the course of a single year amount to several times the weight of the body. The precautions to be taken to avoid pollution of wells and springs are—(1) sufficiency of intervening space between the cemetery and the water source; (2) proper drainage, especially for subsoil water; (3) proper cemetery management.

The English Acts and regulations prescribe no limit of distance for water-supplies within which a cemetery is not to be established. There is no power to prevent anyone from sinking a well on his own property, as near to a cemetery as he pleases.

Sites for cemeteries should be open and somewhat elevated; not shut in by hills or close belts of high trees; and enclosed with open palings. Trees, though useful, should not be allowed to become an impediment to the air.

8. *Sufficiency of Space.*—The regulations of the Home Office prescribe that no unwallled grave shall be re-opened within fourteen years after the burial of a person above twelve years of age, or within eight years after the burial of a child under twelve years of age, unless to bury another member of the same family, in which case a layer of earth, not less than one foot thick, shall be left undisturbed above the previously buried coffin; but if on re-opening any grave the soil be found to be offensive, such soil shall not be disturbed, and in no case shall human remains be moved from the grave. The size of the grave-space is to be 9 feet by 4 feet = 4 square yards, for an adult; and for a child under twelve, 2 square yards—viz. either $4\frac{1}{2}$ by 4 feet, or 6 by 8 feet. They allow for the hole dug for an adult to be 7 by 2 feet. In any case it is important that each grave should be at least a foot distant from the nearest grave on every side.

The minimum allowance of space in a cemetery should be about a quarter of an acre per 1,000 inhabitants. This allows the graves to be re-used every fourteen years.

THE MEDICAL OFFICER OF HEALTH

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ENGLAND AND WALES

In discussing the duties of and matters relating to medical officers of health it is necessary, in respect of some, to distinguish them in accordance with the several parts of the United Kingdom, whilst others are common to medical officers of health in all parts of it.

In the following pages it is assumed that the remarks are to be applied generally unless it is otherwise specified.

If properly wielded, the influence of the large body of those officers now scattered throughout the country is destined to fulfil an important part in the future destiny of the nation, inasmuch as the ever-increasing density of population will necessitate continued care and foresight to avert the deterioration of the public health so likely to accrue from that cause, whilst much will depend upon the thoroughness with which those who hold the office comprehend and administer the multifarious duties attaching to it.

The appointment of medical officer of health, as now existing, is of recent origin, but the justification of the creation has already been fully established by the results which have thus far been attained in a comparatively short space of time; the consequent benefits to the public health having fully warranted the innovation which was made by the Legislature.

The Towns Improvement Clauses Act, 1847, which extended only to such towns or districts in England or Ireland as should be comprised in any Act of Parliament subsequently passed which should declare it, or any part of it, to be incorporated therewith, provided for the appointment of a salaried 'officer of health' by the 'Commissioners' under the special Act, if they thought fit. The person appointed had to be of competent skill and experience, and the appointment had to be approved as prescribed by the special Act, or by one of Her Majesty's principal Secretaries of State. His duties were to 'ascertain the existence of diseases within the limits of the special Act, especially epidemics and contagious diseases, and to point out any nuisances or other local causes likely to cause and continue such disease, or otherwise injure the health of the inhabitants, and to point out the best means for checking or preventing the spread of such diseases within the limits aforesaid, and also the best means for the ventilation of churches, chapels, schools, registered lodging-houses, and other public buildings within the limits aforesaid, and from time to time as required by the Commissioners to report to them upon the matters aforesaid, and to perform any other duties of a like nature which may be required of him.'

The Commissioners, with the above-described approval, might discontinue the office or remove any such officer of health.

The Public Health Act, 1848, which could be adopted in England and Wales, under certain conditions, in any city, town, borough, parish, or place having a known or defined boundary, gave power to the local board of health of any district constituted under it to appoint a legally qualified medical practitioner, or a member of the medical profession, 'officer of health' for their district, who was to perform such duties as the then existing General

Board of Health should direct. That body issued a code of regulations prescribing those duties in 1851. The same person might be officer of health for two or more districts.

Under that Act, the Local Government Act, 1858, and some local Acts, officers of health were appointed in some cities, towns, and smaller places.

Dr. Duncan, of Liverpool, was the first medical officer of health, having been appointed in 1847 under, and immediately after the passing of, the Liverpool Sanitary Act, 1846. Sir John (then Mr.) Simon was the second, and was appointed in 1848, when the City of London obtained the power to appoint a medical officer, by means of the City Sewers Act of that year. In his instructive and valuable work entitled 'English Sanitary Institutions,' he has given a graphic account of his initiation of the routine of the office, which has now been extended throughout the length and breadth of the country.

It was not until the Public Health Act, 1872, was passed that the duty of appointing medical officers of health was imposed upon the local authorities in all parts of England and Wales outside the metropolis. Then for the first time the public health in any thoroughly country part came under regular medical supervision. Previously to that, however, section 14 of the Nuisances Removal Act, 1860, enabled the guardians of any union, or parish not within a union, to employ at any time one of their medical officers to make inquiry and report upon the sanitary state of the whole or of any part of their union or parish, and to pay him for so doing; but it does not appear that advantage was generally taken of this power.

The Act of 1872 divided the whole of England and Wales, exclusive of the metropolis, into 'Urban Sanitary Districts' and 'Rural Sanitary Districts' as they are now known, and placed them under the jurisdiction of 'Urban Sanitary Authorities' and 'Rural Sanitary Authorities,' who were to appoint medical officers of health, being legally qualified medical practitioners, in every one of their districts. The appointments first made after the passing of the Act were to be for a period not exceeding five years.

The Act also instituted 'Port Sanitary Authorities,' by whom medical officers of health could be appointed.

The Local Government Board were to have the same powers as they have in the case of district medical officers of unions, with regard to the appointment, duties, salary, and tenure of office of those medical officers of health whose salaries were partly paid out of moneys voted by Parliament, and in November 1872 they issued regulations with respect to the qualification, appointment, duties, salary, and tenure of office of such officers, which were very similar to those at present in force, and which now apply only to medical officers of health who were appointed on or before March 25, 1880, and have not been reappointed since, and whose salaries were partly paid as described above, but now by county councils.

It was enacted that the same person might, with the sanction of that Board, be appointed the medical officer of health for two or more sanitary districts, by the joint or several appointment of the sanitary authorities of the districts, and, with a like sanction, a district medical officer of a union might be appointed a medical officer of health.

The Act gave medical officers of health power to exercise any of the powers with which inspectors of nuisances were invested by any of the then existing Sanitary Acts.

Most of those Acts, together with the Act of 1872, except so far as some of them related to the metropolis, Scotland, or Ireland, were repealed, consolidated, and in some respects amended, for the rest of England and Wales

by the Public Health Act, 1875, under which medical officers of health are now appointed by all port sanitary authorities and by urban and rural sanitary authorities outside the limits of the metropolis. They are appointed in urban sanitary districts under section 189, in rural sanitary districts under section 190, and by port sanitary authorities under section 287 of that Act.

Urban sanitary authorities are authorised to appoint only one medical officer of health in their districts, though they may appoint any assistants that may be necessary for the efficient execution of the Act, but rural sanitary authorities are allowed to appoint more than one medical officer of health if they think fit.

Under section 191 of the Act of 1875, a medical officer of health may, with the sanction of the Local Government Board, be appointed for two or more districts, and any district medical officer of a union may, with a like sanction, and subject to such conditions as the Board may prescribe, be appointed a medical officer of health; in case of illness or incapacity of a medical officer of health, a local authority may appoint and pay a deputy medical officer, subject to the consent of the Board.

Under section 286 of the same Act, the Local Government Board may, by order, unite two or more districts for the purpose of appointing one medical officer of health for both or all of them, but no urban district containing a population of 25,000 and upwards, or (in the case of a borough), having a separate court of quarter sessions, can be included in any union of districts so formed without the consent of the local authority of such district or borough; and if the local authority of any district proposed to be included in the union give notice to the Board that they object to the proposal, their district cannot be included in the union without a provisional order of the Board.

The Board may assign to the district medical officer of any union comprising or coincident with any constituent district, such duties in rendering local assistance to the medical officer of health appointed for the united districts as they may think fit; such district medical officer may, with their sanction, receive additional remuneration for the performance of the duties so assigned to him.

A parochial committee formed by a rural sanitary authority cannot appoint a medical officer of health, but the Local Government Board have expressed it as their opinion that they may, in the exercise of their duties, consider the action taken by this officer on complaints of any nuisances, inform him of any nuisances requiring his attention, and report to the rural sanitary authority the manner in which he has discharged his duties.

Now power is given, by the Local Government Act, 1888, to county councils to appoint and pay medical officers of health, and they may make arrangements with any urban and rural sanitary authorities within their counties to employ such officers in place of appointing others under the Public Health Act, 1875.

The City of London Sewers Act, 1848, made permanent by the City of London Sewers Act, 1851, gave the Commissioners of Sewers power to appoint medical officers of health for the City of London.

In the rest of the metropolis those officers were appointed by the vestries and district boards, acting under the powers given them by the Metropolis Management Act, 1855. The 182nd section of that Act required that they should be appointed, and that they should be legally qualified medical practitioners. Such persons were to be called 'medical officers of health;' and it was lawful for vestries or district boards to pay to every such officer

such salary as they thought fit, and also to remove any such officer at their pleasure.

On and after January 1, 1892, all appointments of medical officers of health in London became regulated by sections 106 and 108 of the Public Health (London) Act, 1891; whilst temporary arrangements for the performance of the duties of such officers are provided for by the 109th section of that Act.

It is important to note that all future appointments in the metropolis are now placed upon the same footing as those in all other parts of England, that the Local Government Board have the same control over the qualifications, appointment, duties, salary, and tenure of office, of the officers appointed as they have in other parts of the country, and that one-half of the salaries must be paid by the London County Council.

Every sanitary authority in London is bound to appoint one or more medical officers of health for their district. The same person may, with the sanction of the Local Government Board, be appointed medical officer of health for two or more districts. Every such officer appointed, or re-appointed after the commencement of this Act, must (except during the two months next after the time of his appointment, or except in cases allowed by the Local Government Board) reside in his district, or within one mile of the boundary of it, and if, while not so residing, he assumes to act or receives any remuneration as such officer, he will cease to hold the office.

Unless the Local Government Board exercise their discretionary power in this behalf somewhat freely, either a hardship will be inflicted upon some capable officers, or else those most fitted for the post will not always be selected.

A medical officer of health in London, as in extra-metropolitan districts, may exercise any of the powers with which a sanitary inspector is invested.

Those appointed under this Act will be removable by the sanitary authorities, with the consent of the Local Government Board, or by that Board, and not otherwise; but the Board must take into consideration every representation made by a sanitary authority for the removal of any medical officer, whether based on the general interests of the district, on the conduct of such officer, or on any other ground.

Moreover, any such officer is not to be appointed for a limited period only. Surely this long-denied measure of justice should be extended to his less fortunate *confrères* in other parts of England at the earliest possible opportunity, the present precarious tenure of office being most inimical and harassing to every conscientious holder of the office.

A medical officer of health appointed under this Act must be legally qualified for the practice of medicine, surgery, and midwifery, and in all London districts he must be possessed of one or more of the same qualifications as are named in section 18 (2) of the Local Government Act, 1888, which is presently set forth in full. In the case of the holder of any office already in existence at the commencement of the Act these provisions are qualified by the 139th section as follows:—'Where a portion of the salary of such officer is paid by the London County Council, the Local Government Board has the same powers as they have in the case of a district medical officer of a poor law union with regard to his qualification, appointment, duties, salary and tenure of office.'

In any other case the Board may prescribe his qualifications and duties. Subject to these powers of the Board, the sanitary authority may make such payments as they think fit on account of his remuneration and expenses,

and he will be removable by the authority at their pleasure. The requirements of the Act with respect to their qualification does not apply to medical officers appointed before January 1, 1892; and the Act will not prevent any person who at its commencement is both a district medical officer of a union and a medical officer of health from continuing to hold both those appointments in like manner as if it had not been passed.

The medical officer of health of the Port of London has been appointed under the Public Health Act, 1875; but future appointments will probably be regulated by the Public Health (London) Act, 1891.

A person could not be appointed medical officer of health under the Public Health Act, 1875, unless he was a legally qualified medical practitioner, and now section 18 of the Local Government Act, 1888, enacts that—'Except where the Local Government Board, for reasons brought to their notice, may see fit in particular cases specially to allow, no person shall hereafter be appointed the medical officer of health of any county or county district, or combination of county districts, or the deputy of any such officer, unless he be legally qualified for the practice of medicine, surgery, and midwifery.

'(2) No person shall after January 1, 1892, be appointed the medical officer of health of any county or of any such district or combination of districts as contained, according to the last published census for the time being, a population of 50,000 or more inhabitants, unless he is qualified as above mentioned, and also either is registered in the Medical Register as the holder of a diploma in sanitary science, public health, or State medicine under section 21 of the Medical Act, 1886, or has been during three consecutive years preceding the year 1892 a medical officer of a district or combination of districts, with a population according to the last published census of not less than 20,000, or has before the passing of this Act been for not less than three years a medical officer or inspector of the Local Government Board.'

The following resolutions in regard to diplomas in State medicine were passed by the General Medical Council on June 1, 1889, November 30, 1889, and November 25, 1890:—

(a) This Council, having regard to the terms of section 18 of the Local Government Act, 1888, and observing that under that section special privilege is to be accorded to the holders of the diplomas granted under section 21 of the Medical Act, 1886, and therein described as diplomas in sanitary science, public health, or State medicine, thinks it essential to declare, with regard to its own future action under section 21 of the Medical Act, 1886, that it will not consider diplomas to 'deserve recognition in the Medical Register' unless they have been granted under such conditions of education and examination as to ensure (in the judgment of the Council) the possession of a distinctively high proficiency, scientific and practical, in all the branches of study which concern the public health; and that the Council, in forming its judgment on the conditions of education and examination, will expect the following rules to have been observed:—

(b) A period of not less than twelve months shall elapse between the attainment of a first registrable qualification in medicine, surgery, and midwifery, and the examination for a diploma in sanitary science, public health, or State medicine.

(c) Every candidate shall have produced evidence of having attended, after obtaining a registrable qualification, during a period of six months, practical instruction in a laboratory approved of by the body granting the qualification.

(d) Every candidate shall have produced evidence that, during a period of six months after obtaining a registrable qualification, he either has practically studied the duties of outdoor sanitary work under the medical officer of health of a county or large urban district, or else has himself held appointment as medical officer of health under conditions not requiring the possession of a special sanitary diploma.

(e) The examination shall have been conducted by examiners specially qualified, and shall comprise laboratory work, as well as written and oral examination.

(f) The rules as to study shall not apply to—

(a) Medical practitioners registered, or entitled to be registered, on or before January 1, 1890.

(B) Registered medical practitioners who have for a period of three years held the position of medical officer of health to any county, or to any urban district of more than 20,000 inhabitants, or to any entire rural sanitary district.

The new office of Medical Officer of Health of an Administrative County has been created by section 17 of the Local Government Act, 1888, in the following terms: ' (1) The council of any county may, if they see fit, appoint and pay a medical officer of health, or medical officers of health, who shall not hold any other appointment or engage in private practice without express written consent of the council. (2) The county council and any district council may from time to time make and carry into effect arrangements for rendering the services of such officer or officers regularly available in the district of the district council, on such terms as to the contribution by the district council to the salary of the medical officer, or otherwise, as may be agreed, and the medical officer shall have within such district all the powers and duties of a medical officer appointed by a district council. (3) So long as such an arrangement is in force, the obligation of the district council under the Public Health Act, 1875, to appoint a medical officer of health shall be deemed to be satisfied without the appointment of a separate medical officer.'

This appears to be a disadvantageous arrangement and one likely to defeat the object a county council should have in view in appointing a medical officer. One of the principal reasons for the existence of such an officer is that the council may have the advantage of his advice on any public health questions involved in their consideration of the manner in which the Public Health Acts and other matters affecting the public health are administered in the various sanitary districts within their administrative county; whereas if their officer were in the employ of any of the local authorities of those same districts it is scarcely probable that he would act as an impartial censor of their, or of his own, doings or shortcomings; he would, at any rate, be open to the suspicion of regarding them in a too favourable light, and of reviewing his own reports with undue leniency.

The Act does not refer to the tenure of office of these officers, but it is to be hoped that it will be placed upon a surer footing than that of medical officers appointed under the Act of 1875.

The position and duties of these new officers, of whom a few have already been appointed, are not defined by the Act, neither are the county councils nor the Local Government Board empowered by it to make bye-laws or regulations respecting them; in fact, beyond mentioning that such officers may be appointed, it is silent upon those matters, except when the local authority of any district within the county make arrangements with the council for rendering his services regularly available in their district, when he will have, within the district of the local authority, all the powers and duties of a medical officer of health appointed in the usual way. The qualifications he is required to possess are set forth in the eighteenth section of the Act, which has already been given at length in this article.

When not himself filling those local appointments he will have to exercise a wise discretion in his relations with those officers who hold them; he must not attempt to supplant them in the fulfilment of their statutory powers within their districts.

His duties are, to some extent, shadowed forth in a concise report made to the Staffordshire County Council, the first of the kind which appeared, by

Dr. George Reid, their medical officer of health, appointed under the Act of 1888. He understands that, with the view of assisting the council in forming an opinion regarding the matters contained in the annual and special reports which have to be sent to them by the medical officers of health of the various local authorities within their county, they have appointed him their medical officer of health, and that it is his duty to analyse and report upon those documents to the council, making such suggestions as he thinks desirable; but that, unless specially authorised to do so, he has no power to inspect personally any district, or to take any direct action in sanitary matters. He then comments upon the salient points in the reports which he had received, and makes suggestions for the guidance of the council in the action he considers called for on matters arising out of those reports, and he draws the attention of the council to various questions relating to public health.

County councils will find the need of being able to avail themselves of the advice of highly trained medical officers possessing special experience in public health work, if only to guide them in their action under sub-section 2 of the 19th section of the Act of 1888, which enacts that: 'If it appears to the county council from any such report that the Public Health Act, 1875, has not been properly put in force within the district to which the report relates, or that any other matter affecting the public health of the district requires to be remedied, the council may cause a representation to be made to the Local Government Board on the matter.'

It would appear impossible for the medical officer to properly advise the council on such matters without himself visiting and examining the localities concerned, and inquiring into all the circumstances; but the Act gives him no statutory right to visit the district of a local authority, for whom he does not act as medical officer, against their will, neither does it give him any power of entry to premises or structures such as belongs to a medical officer of health appointed under the Act of 1875; but he has right of admission to any premises for the purposes of section 45 of the Housing of the Working Classes Act, 1890, respecting any dwelling-house in a state so dangerous or injurious to health as to be unfit for human habitation, or respecting any 'obstructive building' in the sanitary districts comprised within the metropolis exclusive of the City of London, in the local board district of Woolwich, and in all rural sanitary districts.

Inasmuch as he will be brought into contact with, and to a certain extent will have to criticise the work of the local officers, it seems highly desirable that the medical officer of health of a county council should, whenever practicable, be chosen from amongst those officers who have had large and varied experience of local appointments.

He may be called upon to advise the council on any questions concerning health involved in the construction or management of any buildings belonging to them, in the framing or enforcing of any bye-laws they may make under the 16th section of the Local Government Act of 1888, in opposing, under the 15th section, any bills in Parliament, such, for instance, as may be promoted by local authorities for works of sewerage, sewage disposal, water-supply, &c., or upon any action the council may deem it necessary to take under the 14th section for enforcing any of the provisions of the Rivers Pollution Prevention Act, 1876, or under the Sale of Food and Drugs and Margarine Acts.

Beyond the foregoing his duties appear to be vague, and will probably vary in accordance with the particular ideas concerning them held by different county councils.

The Public Health Committee of the Lancashire County Council have reported to that authority what they deem may be considered to constitute the duties of the medical officer of a county council in the following terms, which appear to give a succinct epitome of what may possibly be expected of these officers :—

(a) To receive on behalf of the council the copies of the periodical reports from the local medical officers of health, and to tabulate and report upon the same for the information and guidance of the council.

(b) To inform the council whether the various Acts affecting the public health are or are not being properly put into force within any district; whether any other matters affecting the public health of a district, such as water-supply, drainage, scavenging, insanitary dwellings, &c., require being remedied, so as to enable the council to decide whether a representation should be made to the Local Government Board on the matter under section 19 of the Local Government Act, 1888.

(c) To assist and advise the local authorities and their medical officers and sanitary inspectors as to epidemics, &c., and generally to advise them in the performance of their duties when required by them, free of charge.

(d) To assist and supervise the inspectors under the Sale of Food and Drugs Act.

(e) To inquire and report as to the desirability of making bye-laws for the prevention and suppression of nuisances not already punishable in a summary manner.

(f) To perform the duties and be subject to the liabilities which, by the Artisans and Labourers' Dwellings Improvement Act, 1875,¹ a medical officer is required to perform and be subject to.

(g) To assist the council in dealing with the pollution of rivers.

(h) To advise the council on any bills introduced into Parliament with reference to—

- (1) The public health;
- (2) The extension of towns;
- (3) The water-supply of towns;
- (4) The housing of the working classes.

(i) To advise as to the sanitary condition of all buildings belonging to the council.

(j) To devote his whole time to the duties of his office, and to hold himself at all times ready to advise the council and its committees.

(k) To be a county centre for all vital and hygienic statistics and other information bearing upon public health, including meteorological data from all available sources. The committee also point out that many other important duties devolve on the council, in relation to which the advice and assistance of an experienced medical officer of health will be invaluable, such as the making of bye-laws, the execution of the Acts relating to contagious diseases of animals, and many other matters, all of which are likely to be materially increased at an early date by the transfer to county councils of additional powers.

'Having regard to the absolute necessity of these duties being at once properly carried out, and to the practical impossibility of the Committee undertaking them itself,' they recommended that a medical officer of health should be appointed for the administrative county of Lancaster.

Already the sanitary committee of the council of the West Riding of Yorkshire have directed that their medical officer shall, when desired by the local authorities, advise or assist them or their officers in the performance of their duties under the public health laws.

The county council medical officer may, by being enabled to take a broader survey of the circumstances of epidemic disease than is possible for local officers, be instrumental in elucidating the origin and spread of epidemics in his county.

In London the county council now have all the powers and duties formerly attaching to the Metropolitan Board of Works, so their medical officer will be concerned in the framing and enforcing of any bye-laws, so far as questions of health may arise, with respect to drainage, the emptying,

¹ Now the Housing of the Working Classes Act, 1890.

cleansing, closing, and filling up of cesspools, the removal and disposal of refuse, and new buildings, which they may make under the statutory powers for that purpose which they possess ; and he may be concerned with bye-laws for regulating seamen's lodgings, if the council make any under the power given them by the Merchant Shipping (Fishing Boats) Act, 1888.

In the metropolis, exclusive of the City of London, he will be charged with the duties attaching to medical officers of health under Parts I. and II. of the Housing of the Working Classes Act, 1890 ; and if bye-laws for the regulation of lodging-houses, under Part III. of the Act, are made by the county council, he might be called upon to advise in framing and administering them ; and when authorised by that authority he might have to inspect any lodging-houses established by them.

His attention will be required in respect of matters arising under the Dairies, Cow-sheds, and Milk-shops Order of 1885, and regulations made thereunder ; or out of any Orders of the Local Government Board, or bye-laws of the county council, for the regulation of dairies which may be made under section 28 of the Public Health (London) Act, 1891.

He will have to advise on questions in connection with the licensing of slaughter-houses, knackers' yards, cow-sheds, and places for the keeping of cows, the establishment anew, and regulation of offensive trades, and on any nuisances which may be created by sanitary authorities in dealing with house and street refuse.

He will be concerned with the weekly returns of infectious diseases made to the council, under the Public Health (London) Act, 1891, by the Metropolitan Asylums Board.

The Local Government Board have by General Orders, dated March 1891, prescribed regulations for the appointment, tenure of office, salary, and duties of medical officers of health of urban and rural sanitary districts appointed or reappointed after March 25, 1891, any portion of whose salary is paid by county councils ; and also regulations for the duties of other medical officers of health appointed or reappointed under the Act of 1875, after the above-named date.

The duties are dealt with in the following pages, but the other matters will not be discussed.

Beyond the legal qualifications there are others of a personal nature which are essential in the successful medical officer of health.

Since he is not so much an executive officer as he is the adviser of the local authority appointing him, on all matters pertaining to his office, he must be prepared from time to time to accept with equanimity the rejection of any advice he may have tendered to them, however sound it may have been. He must not be too sensitive in the event of his advice or actions being misunderstood or even misconstrued, if he is conscious of having been actuated by proper motives.

He must not advise lightly, hastily, or inconsiderately on any matter, but only after due deliberation, and must put himself in a position to be able to stand by and, if necessary, to defend his advice, since it may be the initiative of legal proceedings. Hasty or ill-considered advice, thoughtlessly given, would disparage him and diminish his influence with the public, as well as with the local authority.

Having advised conscientiously and to the best of his abilities, he has discharged his duty, and if his advice is not followed he must bear in mind that the decision of the authority may have been subject to considerations and influences which had no weight with him. If, on the other hand, he finds that his advice is systematically and persistently ignored by the authority,

and that the public health in his district is likely to be prejudicially affected in consequence, he must repeat it, again and again if need be, firmly but dispassionately. His advice on all matters of importance should be tendered to the local authority in writing, with a view to future reference.

He should, under many circumstances, endeavour to induce the authority themselves to propose any works which may be needful for the benefit of his district, rather than urge the proposals too much as emanating from himself. He will often find some member of the authority willing to take a matter up as a scheme of his own, and what may be legitimate pertinacity in a member of the authority might be considered unbecoming obstinacy in the medical officer of health. By paying attention to this point he will not infrequently gain his end more surely than if he had paraded the proposal as his own. It is not conducive to success for him to be continually thrusting himself prominently forward in his official capacity. His power for good will not be lessened if he sometimes judiciously obscures his individuality.

He should regard all questions he has to deal with from the view-point of the local authority, the owners and occupiers of premises, and the public, as well as from that of the medical officer of health intent upon the health-improvement of his district.

He must be possessed of good judgment, discretion, and tact, for he will have various and varied persons and matters to grapple with; from this it follows that the larger, wider, and more varied his experience in his special branch of the profession may be, the better able will he be to perform with efficiency the duties pertaining to it.

While careful to avoid encroaching in any way upon the province of the clerk, the surveyor, or the engineer of the authority, he should, nevertheless, have an intimate acquaintance with all the statutes and bye-laws relating in any degree to his office, and should be conversant with the principles of modern sewerage, drainage, disposal of sewage, water-supply, warming and ventilation of buildings, and house construction; and while not taking upon himself the duties of the inspector of nuisances, he should be intimately and practically acquainted with them.

He should have a knowledge of any trades, articles of trade, or manufacturing processes that are liable to be controlled by the local authority.

He must be well acquainted with the etiology, so far as it may be known, and the characteristics of those diseases, at any rate, which are capable of influencing the public health, the most numerous of which will be found amongst the zymotic diseases, and must know the principles of hospital construction, especially in relation to hospitals for the isolation of infectious diseases.

He should possess a general knowledge of bacteriology, but he will probably find that his multifarious duties will not allow him sufficient leisure to become proficient in the practice of it, and that he had better leave that to those who have the time and opportunity to become expert in it; such specialism and division of labour is a necessary outcome of the vast multiplication of the scientific knowledge of the age.

He must be trained in the methods of observation requisite for tracing the origin and course of epidemic and other diseases.

He must be capable of expressing himself in his reports with terseness and perspicuity.

He must know how to compile statistics, how to deduce sound conclusions therefrom, and how to avoid fallacies in dealing with them.

He should, at any rate, know how to properly interpret the results of analyses of water, air, and articles of human consumption, and should under-

stand meteorology, climatology, and cognate subjects. He should be acquainted with the general facts relating to the geology of his district.

He should possess an even temper, as he will find it will frequently be tried. If of an irritable or excitable temperament, he must train himself to control it; it is of the utmost importance that not one of his official actions or expressions should savour in the remotest degree of resentment or vindictiveness.

He must be patient and persevering, since his efforts should be directed to the accomplishment of his objects by persuasion rather than by compulsion. He must endeavour to educate rather than to force public opinion, seeing that little good is to be done by attempting to go much in advance of that in regard of matters pertaining to the public health.

He must not be impulsive, but must be methodical, exact, and punctual in all his dealings, and must conduct the business of his department in a manner that will not cause friction with any other department connected with the local government of his district. He must show courtesy and respect to every member of the local authority.

Whatever his political bent may be, he must strictly avoid making any public parade of it; it is absolutely essential that he should banish any suspicion of political bias from all his official proceedings; even if the authority is so unwise as to allow the local government of their district to be swayed by political considerations, as too frequently happens, he must not permit himself to be influenced by their baneful example, since he is the officer of the whole public and not alone of any one section of it.

He must not allow himself to be drawn into any local newspaper controversy in regard of any of his official actions. Such a proceeding marks a very raw and inexperienced office-bearer. If he has acted wrongly he will not thus clear himself, but if rightly, his sense of the dignity of his office should preclude him from taking such a course, as tending to bring it into contempt, and himself into ridicule.

He must not go beyond his special province; he will seldom gain any credit, but often very much the reverse, if he sets himself to do anything beyond that which comes strictly within his statutory duties. If, however, he observes anything amiss which does not come within their scope, he will do well to give an intimation of his observation in the appropriate quarter.

Above all, he must practise the strictest honesty, integrity, straightforwardness, and impartiality in all his actions, and must rigorously resist the slightest attempt on the part of anyone to do anything towards him which could by any possibility be construed as an effort to influence his conduct as a public officer. He must regard himself as the custodian of the fair fame of public offices in general, and of his own in particular.

It is desirable that he should occupy such a social position as will fully command the confidence and respect of all classes throughout his district.

He must cultivate cordial relations with the whole of the medical profession in his district, as without their confidence, respect, and co-operation, his work will become much more arduous and be far less successful, his power for good will be greatly crippled. His behaviour towards them should be regulated by what his conscience tells him would be the treatment he should expect from a medical officer of health if he were himself in practice. He must be careful not to wound their legitimate susceptibilities and sensitiveness: especially must he be on his guard in these respects if he is also a private medical practitioner. The passing of the Infectious Disease (Notification) Act, 1889, accentuates if possible the importance of mutual confidence

and esteem being upheld between the profession and the medical officer of health. Unfortunately this obligation is not always comprehended, and, in consequence, there have been instances in which the medical officer of health has erred grievously in that behalf, at some times through ignorance of his powers and duties, but at others through personal unfitness for the office.

He must not regard himself as in any way the censor of his medical brethren, but must endeavour to win their trust and confidence, so that they may freely seek his advice and assistance in regard of any matters within his province occurring in their practice.

If in the discharge of his duties he is brought into contact with any irregular practitioner, his dealings with him must not be regulated by ordinary professional rules, but must be guided by regard for the public weal, which must be the paramount consideration.

If he be appointed by a county council he must endeavour to keep in harmony with the medical officers of the various sanitary districts comprised within his district, and must not imagine that by virtue of his office he is placed over them.

Although the Public Health Acts do not place the inspector of nuisances under his control, yet in the General Orders of the Local Government Board of 1891, prescribing regulations as to inspectors of nuisances and sanitary inspectors, some of their duties are given in the following terms :—‘ Art. 19. (1) He shall perform, either under the special directions of the sanitary authority, or (so far as authorised by the sanitary authority) under the directions of the medical officer of health, or in cases where no such directions are required, without such directions, all the duties specially imposed upon an inspector of nuisances by the Public Health Act, 1875, or by any other statute or statutes, or by the Orders of the Local Government Board, so far as the same apply to his office.’

Clause (7) requires him to inspect shops and places used for the sale of butchers’ meat and other articles of food, and to seize and to have dealt with by a justice any article which appears to him to be unfit for the food of man : ‘ Provided that in any case of doubt arising under this clause he shall report the matter to the medical officer of health, with the view of obtaining his advice thereon.’ ‘ (9) He shall give immediate notice to the medical officer of health of the occurrence within the district of any contagious, infectious, or epidemic disease ; and whenever it appears to him that the intervention of such officer is necessary, in consequence of the existence of any nuisance injurious to health, or of any overcrowding in a house, he shall forthwith inform the medical officer of health thereof.’ ‘ (10) He shall, subject to the directions of the sanitary authority, attend to the instructions of the medical officer of health with respect to any measures which can be lawfully taken by an inspector of nuisances under the Public Health Act, 1875, or under any other statute or statutes for preventing the spread of any contagious, infectious, or epidemic disease of a dangerous character.’ ‘ (12) He shall at all reasonable times, when applied to by the medical officer of health, produce to him his books, or any of them, and render to him such information as he may be able to furnish with respect to any matter to which the duties of inspector of nuisances relate.’

And in the Orders of the Local Government Board relating to medical officers of health, one of their prescribed duties is : ‘ Subject to the instructions of the sanitary authority, he shall direct or superintend the work of the inspector of nuisances in the way and to the extent that the sanitary authority shall approve. . . .’

In that limited degree the inspector of nuisances, whether in urban or rural districts, may be called upon to act under the medical officer of health.

In the metropolis the relations between these officers varied and were dependent to a great extent upon the arrangements made by the various local authorities, but they are now made similar to those existing in other parts of the country by the Sanitary Officers (London) Order, 1891.

If the inspector finds that the medical officer of health is competent, and if he receives reasonably fair and considerate treatment at his hands, he will look to him for direction and guidance. This is what generally happens in practice, and they work loyally together for the furtherance of the public weal. If these relations do not subsist between those officers, it will be found, as a rule, that one or the other of them is at fault. Unless they do exist it is impossible for the work of either to be properly done. The inspector should be as the right hand of the medical officer of health, for the state of the district must largely depend upon the thoroughness with which he does his work, since it is impossible and undesirable for the medical officer to undertake every minute detail of inspection; there is plenty of other important work to fully occupy his time and attention. He should, however, at all times be able and willing to give the inspector any assistance or advice in the execution of his duties of which he may be in need.

Though not in general called upon to fulfil the part of the inspector, he must nevertheless be capable of doing so if occasion requires, and must be so well acquainted with the duties of that officer as to be able to instruct or correct him if the necessity for so doing should ever arise.

It is enacted in section 191 of the Public Health Act, 1875, that 'a medical officer of health may exercise any of the powers with which an inspector of nuisances is invested by this Act;' therefore, although the medical officer is not mentioned in sections 36, 49, and 266, in which the inspector of nuisances is named, he may nevertheless act under them, bearing in mind that section 49 relates to urban sanitary districts only.

In his relations with the public, the medical officer of health must be considerate and unobtrusive. He must endeavour to carry out his, occasionally unwelcome, duties in such an equitable manner as shall not provoke resentment, and must so exercise his powers that his visits may be welcomed rather than shunned; he must have regard for the rights and interests of the owners as well as of the occupiers of premises, so that his advice and assistance may be readily sought by both alike.

The limitation of the appointment of medical officers of health in England and Wales, outside the metropolis, first made after the passing of the Public Health Act, 1872, to a period not exceeding five years, was not re-enacted in the Act of 1875, but that Act gives the Local Government Board control over the tenure of office of medical officers of health whose salaries were partly repaid out of monies voted by Parliament, and now by county councils; they have, however, no such control over the appointment of others.

The metropolitan appointments have hitherto been of a more or less permanent character, having generally been held during the pleasure of the local authorities, and now they are not to be made for a limited period only; but it is far otherwise with most of those which have been made under the Public Health Act, 1875. On referring to the return as to the appointments of medical officers of health in England and Wales, ordered by the House of Commons to be printed on August 10, 1888, it is seen that of those made under that Act by single urban sanitary authorities, about 67 per cent. were for periods of one year or under, whilst about 77 per cent. of those

made by single rural sanitary authorities were for a like period. Most of the rest were for short terms, not exceeding five years, a few were for indefinite periods, fewer were held during the pleasure of the sanitary authorities, and still fewer at from three to six months' notice, whilst only seventeen out of 816 urban, and seven out of 486 rural appointments made under the Act were classed as permanent.

A few other appointments which had been made under earlier or under local Acts were returned as permanent.

The appointments made by two or more sanitary authorities in combination fared but little better; out of the total of thirty-seven, five were returned as made for one year, three for two years, fourteen for three years, one for four years, nine for five years, one for five years and ten months, one for seven years, two for ten years, whilst only one was returned as permanent.

The fifty-four appointments made by port sanitary authorities included five which were permanent, three held during the pleasure of the authority, one at three months' notice, and three for indefinite periods, whilst the remainder were held for short terms varying from six months to five years.

This uncertainty of tenure of office is anything but satisfactory, and is most unfair to the holders of the appointments, who, if they perform their duties 'without favour or affection, prejudice or malice to any person whomsoever,' as it behoves them to do, will experience oft-recurring opportunities of suffering for their conscientiousness. At the least, the sense of such a contingency continually hanging over them will not tend to lighten their burden of office.

It is to the interest of the public health, as well as of the officers themselves, that these appointments should be placed upon a surer footing.

Out of the total number of appointments made by urban, rural, and port sanitary authorities, only sixty-five of the officers were returned as being debarred from private practice, by far the greater number having been made amongst general practitioners, very many of whom were also poor-law medical officers.

Whilst freely admitting that some of the most accomplished medical officers of health are to be found amongst those who also follow their profession as private practitioners, yet it cannot be doubted that the multiplication of appointments to insignificant districts is the reverse of conducive to the successful administration of the laws relating to public health.

The constitution of districts to which medical officers of health are appointed varies from the smallest urban districts, comprising nothing more than large villages, to the urban districts of the largest cities and towns; from the small portions of the rural districts comprised within poor-law medical districts, to whole rural sanitary districts; and to combinations of rural, or more generally of urban and rural, sanitary districts, some of which are of large extent. In the case of port sanitary authorities, the medical officer of health of some adjoining authority is usually appointed by them, the port sanitary authority of London being the only one which has retained the full services of such an officer. There are also the districts of medical officers of health of administrative counties.

There is an entire absence throughout the country of uniformity in character and size of the districts, in the relative amount of salary attaching to the appointments, and in the fitness for the special work exhibited by the persons appointed.

The salary is often quite inadequate for the work which ought to be done. Medical officers of health appointed under the Public Health Act, 1875, have

no claim for any superannuation allowance, but the vestries and district boards in the metropolis may grant such allowances, under the Superannuation Allowances Act, 1866, to medical officers of health who may become incapable of discharging their duties efficiently, by reason of permanent infirmity of mind or body, or of old age after sixty years of age, and they may grant them gratuities, under certain circumstances, in case of retirement before they are entitled to a superannuation allowance.

The London County Council had the same power, but have procured its repeal, intending to substitute for it a system of superannuation, the officers contributing to the fund created for the purpose.

The medical officer of health should keep a correct account of all cash disbursements which he is entitled to have repaid to him, and he should bear in mind that such account may possibly have to be produced to an auditor.

The duties of medical officers of health are thus enumerated in the General Orders of the Local Government Board, dated March 28, 1891 :

Duties

Art. 18. The following shall be the duties of the medical officer of health :—

(1) He shall inform himself as far as practicable respecting all influences affecting or threatening to affect injuriously the public health within the district.

(2) He shall inquire into and ascertain by such means as are at his disposal the causes, origin, and distribution of diseases within the district, and ascertain to what extent the same have depended on conditions capable of removal or mitigation.

(3) He shall by inspection of the district, both systematically at certain periods, and at intervals, as occasion may require, keep himself informed of the conditions injurious to health existing therein.

(4) He shall be prepared to advise the Sanitary Authority on all matters affecting the health of the district, and on all sanitary points involved in the action of the Sanitary Authority; and, in cases requiring it, he shall certify, for the guidance of the Sanitary Authority or of the justices, as to any matter in respect of which the certificate of a medical officer of health or a medical practitioner is required as the basis or in aid of sanitary action.

(5) He shall advise the Sanitary Authority on any question relating to health involved in the framing and subsequent working of such bye-laws and regulations as they may have power to make, and as to the adoption by the Sanitary Authority of the Infectious Disease (Prevention) Act, 1890, or of any section or sections of such Act.

(6) On receiving information of the outbreak of any contagious, infectious, or epidemic disease of a dangerous character within the district, he shall visit without delay the spot where the outbreak has occurred, and inquire into the causes and circumstances of such outbreak, and in case he is not satisfied that all due precautions are being taken, he shall advise the persons competent to act as to the measures which may appear to him to be required to prevent the extension of the disease, and take such measures for the prevention of disease as he is legally authorised to take under any statute in force in the district or by any resolution of the Sanitary Authority.

(7) Subject to the instructions of the Sanitary Authority, he shall direct or superintend the work of the inspector of nuisances in the way and to the extent that the Sanitary Authority shall approve, and on receiving information from the inspector of nuisances that his intervention is required in consequence of the existence of any nuisance injurious to health, or of any overcrowding in a house, he shall, as early as practicable, take such steps as he is legally authorised to take under any statute in force in the district, or by any resolution of the Sanitary Authority, as the circumstances of the case may justify and require.

(8) In any case in which it may appear to him to be necessary or advisable, or in which he shall be so directed by the Sanitary Authority, he shall himself inspect and examine any animal, carcase, meat, poultry, game, flesh, fish, fruit, vegetables, corn, bread, flour, or milk, and any other article to which the provisions of the Public Health Act, 1875, in this behalf apply, exposed for sale, or deposited for the purpose of sale or of preparation for sale, and intended for the food of man, which is deemed to be diseased, or unsound, or unwholesome, or unfit for the food of man; and if he finds that such animal or article is diseased, or unsound, or unwholesome, or unfit for the food of man, he shall give such

directions as may be necessary for causing the same to be dealt with by a justice according to the provisions of the statutes applicable to the case.

(9) He shall perform all the duties imposed upon him by any bye-laws and regulations of the Sanitary Authority, duly confirmed where confirmation is legally required, in respect of any matter affecting the public health, and touching which they are authorised to frame bye-laws and regulations.

(10) He shall inquire into any offensive process of trade carried on within the district, and report on the appropriate means for the prevention of any nuisance or injury to health therefrom.

(11) He shall attend at the office of the Sanitary Authority, or at some other appointed place, at such stated times as they may direct.

(12) He shall from time to time report in writing to the Sanitary Authority his proceedings, and the measures which may require to be adopted for the improvement or protection of the public health in the district. He shall in like manner report with respect to the sickness and mortality within the district, so far as he has been enabled to ascertain the same.

(13) He shall keep a book, or books, to be provided by the Sanitary Authority, in which he shall make an entry of his visits, and notes of his observations and instructions thereon, and also the date and nature of applications made to him, the date and result of the action taken thereon and of any action taken on previous reports; and shall produce such book or books, whenever required, to the Sanitary Authority.

(14) He shall also make an annual report to the Sanitary Authority, up to the end of December in each year, comprising a summary of the action taken, or which he has advised the Sanitary Authority to take, during the year for preventing the spread of disease, and an account of the sanitary state of his district generally at the end of the year. The report shall also contain an account of the inquiries which he has made as to conditions injurious to health existing in the district, and of the proceedings in which he has taken part or advised under any statute, so far as such proceedings relate to those conditions; and also an account of the supervision exercised by him, or on his advice, for sanitary purposes over places and houses that the Sanitary Authority have power to regulate, with the nature and results of any proceedings which may have been so required and taken in respect of the same during the year. The report shall also record the action taken by him, or on his advice, during the year, in regard to offensive trades, to dairies, cow-sheds, and milk-shops, and to factories and workshops. The report shall also contain tabular statements (on forms to be supplied by us, or to the like effect) of the sickness and mortality within the district, classified according to diseases, ages, and localities:

Provided that, if the medical officer of health shall cease to hold office before December 31 in any year, he shall make the like report for so much of the year as shall have expired when he ceases to hold office.

(15) He shall give immediate information to us of any outbreak of dangerous epidemic disease within the district, and shall transmit to us a copy of each annual report and of any special report. He shall make a special report to us of the grounds of any advice which he may give to the Sanitary Authority with a view to their requiring the closure of any school or schools, in pursuance of the Code of Regulations approved by the Education Department, and for the time being in force.

(16) At the same time that he gives information to us of an outbreak of infectious disease, or transmits to us a copy of his annual report or of any special report, he shall give the like information or transmit a copy of such report to the County Council of the county within which his district may be situated.

(17) In matters not specifically provided for in this order, he shall observe and execute any instructions issued by us, and the lawful orders and directions of the Sanitary Authority applicable to his office.

(18) Whenever we shall make regulations for all or any of the purposes specified in section 134 of the Public Health Act, 1875, and shall declare the regulations so made to be in force within any area comprising the whole or any part of the district, he shall observe such regulations so far as the same relate to or concern his office.

In regard to every medical officer of health no part of whose salary is intended to be payable to an Urban (or Rural) Sanitary Authority by a County Council or by the Town Council of a borough in pursuance of the Local Government Act, 1888, we do hereby order:—

Art. 20. The following shall be the duties of the medical officer of health in respect of the district for which he is appointed:—

(1) He shall, within seven days after his appointment, report the same in writing to us.

(2) He shall perform all the duties prescribed by Article 18 of this Order for a medical officer of health in respect of whose salary a payment is intended to be made by a County Council as aforesaid.

Urban sanitary authorities may make regulations, under section 189 of the Act of 1875, with respect to the duties and conduct of their medical officers of health, but rural sanitary authorities are not empowered to do so.

Vestries and district boards in the metropolis may make bye-laws for regulating those duties, under section 202 of the Metropolis Management Act, 1855.

The Local Government Board issued a General Order in 1889, prescribing regulations as to medical officers of health in the metropolis appointed on or after April 1, 1889, and that was made to apply to any medical officer of health appointed by the Woolwich local authority by another Order. These are superseded by the Sanitary Officers (London) Order, 1891, which now applies to every medical officer of sanitary districts in the administrative county of London appointed, or re-appointed, on or after January 1, 1892. The duties prescribed in it are, for the most part, analogous to those in the Orders of March, 1891.

The Local Government Board have also issued a General Order, dated July 19, 1888, regulating the same matters with respect to medical officers of health appointed, or re-appointed, on or after August 1, 1888, by port sanitary authorities, in which the duties are given in the following terms:—

(1) He shall inform himself, as far as practicable, respecting all influences affecting or threatening to affect injuriously the health of crews and other persons on shipboard within the district.

(2) He shall inquire into and ascertain, by such means as are at his disposal, the causes, origin, and distribution of diseases in the ships and other vessels within the district, and ascertain to what extent the same have depended on conditions capable of removal or mitigation.

(3) He shall, by inspection of the shipping in the district, keep himself informed of the conditions injurious to health existing therein.

(4) He shall be prepared to advise the Port Sanitary Authority on all matters affecting the health of the crews and other persons on shipboard in the district, and on all sanitary points involved in the action of the Port Sanitary Authority; and in cases requiring it he shall certify, for the guidance of the Port Sanitary Authority or of the justices, as to any matter in respect of which the certificate of a medical officer of health or a medical practitioner is required as the basis or in aid of sanitary action.

(5) He shall advise the Port Sanitary Authority on any question relating to health involved in the framing and subsequent working of such bye-laws and regulations as they may have power to make.

(6) On receiving information of the arrival within the district of any ship or other vessel having any infectious or epidemic disease of a dangerous character on board, or of the outbreak of any such disease on board any ship or other vessel within the district, he shall visit the vessel without delay, and inquire into the causes and circumstances of such outbreak, and advise the persons competent to act as to the measures which may appear to him to be required to prevent the extension of the disease, and, so far as he may be lawfully authorised, assist in the execution of the same.

(7) On receiving information from the inspector of nuisances that his intervention is required in consequence of the existence of any nuisance injurious to health, or of any overcrowding in a ship or other vessel, he shall, as early as practicable, take such steps authorised by the Public Health Act, 1875, in that behalf as the circumstances of the case may justify and require.

(8) He shall perform all the duties imposed upon him by any bye-laws and regulations of the Port Sanitary Authority, duly confirmed, in respect of any matter affecting the public health, and touching which they are authorised to frame bye-laws and regulations.

(9) He shall attend at the office of the Port Sanitary Authority, or at some other appointed place, at such stated times as they may direct.

(10) He shall from time to time report in writing to the Port Sanitary Authority his

proceedings, and the measures which may require to be adopted for the improvement or protection of the health of crews or other persons on ship-board in the district. He shall in like manner report with respect to the sickness and mortality of persons on ship-board within the district, so far as he has been enabled to ascertain the same.

(11) He shall keep a book or books, to be provided by the Port Sanitary Authority, in which he shall make an entry of his visits, and notes of his observations and instructions thereon, and also the date and nature of applications made to him, the date and result of the action taken thereon, and of every action taken on previous reports; and shall produce such book or books, whenever required, to the Port Sanitary Authority.

(12) He shall also prepare an annual report, to be made to the end of December in each year, comprising tabular statements (so far as he shall have been able to obtain the necessary information) of the sickness and mortality of persons on ship-board within the district, classified according to diseases, ages, and vessels; and a summary of the action taken during the year for preventing the spread of disease. The report shall also contain an account of the proceedings in which he has taken part or advised under the Public Health Act, 1875, so far as such proceedings relate to conditions dangerous or injurious to health, and also an account of the supervision exercised by him or on his advice for sanitary purposes, over places and vessels that the Port Sanitary Authority has power to regulate, with the nature and results of any proceedings which may have been so required and taken in respect of the same during the year.

(13) He shall give immediate information to the Local Government Board of any outbreak of infectious or epidemic disease of a dangerous character on ship-board within the district, and shall transmit to the Board a copy of each annual and of any special report.

(14) Where any vessel within his district has had dangerous infectious disease on board, he shall give notice thereof to the medical officer of health of any port within the United Kingdom whither such vessel is about to sail.

(15) He shall observe and execute, so far as they may be applicable to his office, the rules and regulations of the Local Government Board in force for the time being, and any instructions of the said Board, and the lawful orders and directions of the Port Sanitary Authority.

The medical officer of health should keep an exact record of all his proceedings, and should make full and accurate notes of all inspections and observations at the time they are made; he should invariably date them correctly. This is especially important in all instances in which any legal proceedings are likely to arise, and it must be borne in mind that it is impossible to foretell when any exact record of a date or occurrence may become of importance.

All his notes should be entered in books kept for the purpose; he should avoid the habit of making them on loose pieces of paper, which are often not forthcoming when most wanted.

If his district comprises several parishes, as happens in rural sanitary districts, he will find it convenient for purposes of reference to keep a register of visits paid to each parish or separate place, together with an index of any entries in his note-books made on those occasions.

He will find that complete notes of his proceedings will be a great assistance in the compilation of the annual reports which he has to make. Instructions regarding the character and compilation of those reports are given in the 'Memorandum as to Annual Reports of Medical Officers of Health,' issued by the medical officer of the Local Government Board, and, since it is impossible to epitomise it without detracting from its clearness, reference must be made to it for information on the subject.

The medical officer of health should endeavour to advance all knowledge relating to preventive medicine, and with that end in view he should give close study and attention to any disease specially prevalent in his district; he should keep a precise record of all the circumstances referring to cases of such disease coming under his observation, and should embody in his annual reports any facts or observations of importance which may bear upon those questions. In so doing he will materially assist in the accumulation, as time

progresses, of valuable information bearing upon the obscure etiology of any diseases which may hitherto have baffled all attempts at elucidation, and the value and importance of his contributions to the literature of public health will be appreciably enhanced.

The obligation to make annual reports and to send copies of them to the Local Government Board and to the council of the county within which their districts are situated is imposed upon all medical officers of health of urban, rural, and port sanitary districts, and upon all metropolitan officers who may have been appointed on or after April 1, 1889, whether or no any portion of their salaries is paid by the county councils.

Section 106 (5) of the Public Health (London) Act, 1891, requires that the annual report of all metropolitan medical officers of health shall be appended to the annual report of the sanitary authorities.

It is important to note that sub-section (1) of section 19 of the Local Government Act, 1888, enacts that: 'Every medical officer of health for a district in any county shall send to the county council a copy of every periodical report of which a copy is for the time being required by the regulations of the Local Government Board to be sent to the Board, and if a medical officer fails to send such copy the county council may refuse to pay any contribution, which otherwise the council would in pursuance of this Act pay, towards the salary of such medical officer.'

On the other hand, if the Local Government Board certify to the county council, under paragraph (c) of sub-section (2) of section 24 of that Act, that any medical officer of health, one-half of whose salary is repaid to the local authority by the county council, has failed to send to that Board such report and returns as are for the time being required by the regulations respecting the duties of such officer made by order of the Board, a sum equal to such half of his salary shall be forfeited to the Crown, and the county council shall pay it into Her Majesty's Exchequer, instead of to the local authority.

It is needful, therefore, that the medical officer of health should not fail to fulfil his duty in these respects.

He should bestow much care upon the compilation of his annual reports, inasmuch as they may be made the medium for the dissemination of valuable instruction, and may be instrumental in the education of the public in a manner that may much conduce to the advancement of public health questions. With these ends in view it is desirable that the reports should be printed and circulated in his district.

He should carefully preserve copies not only of these, but of all his reports and correspondence.

It is quite indispensable, as well for the purposes of his annual reports as for his information as to the general state of health in his district, that he should be regularly furnished with returns of births and deaths from the district registrars of births and deaths; in point of fact, where compulsory notification of infectious diseases is not in force, those returns are often the first and only information of any infectious diseases occurring in his district. Sanitary authorities can, under section 28 of the Births and Deaths Registration Act, 1874, require the district registrars to furnish returns of the deaths they register in their districts, and may pay them for so doing. An arrangement for this purpose should invariably be made with the registrars; the returns should be made on proper forms, and be sent directly to the medical officer of health. They should be made weekly in most cases, but in very small districts it is sufficient if they are sent in monthly, or even quarterly. In all cases, however, deaths from infectious diseases, and any

unusual number from diarrhoea, should be returned to him at the time they are registered.

It is desirable that the medical officer of health should attend, whenever practicable, all the meetings of the sanitary authority, or of its committees, at which any business connected with his department may be transacted. By so doing he will keep in touch with the authority, and may be instrumental in infusing into it that interest in and regard for public health work which are sometimes lacking in those bodies. Supineness on the part of a local authority may not infrequently be traced to negligence in that behalf on the part of the medical officer.

In the event of many parochial committees being formed in any rural sanitary district he will probably find it impossible to be present at all their meetings.

In rural districts he should endeavour to get the sanitary authority to delegate their powers and duties to a committee formed under section 201 of the Act of 1875. Public health work is rarely properly done where a whole board of guardians act as the sanitary authority, except, perhaps, where the board is only a very small one.

In any case the meetings of the authority should always be held at a fixed hour, and preferably before the meetings of the guardians. If held at an indefinite time at the close of those meetings, which are not infrequently unduly protracted, the members of the board are tired or are impatient to get away, so that the work of the sanitary authority is done in a hurried and slovenly manner, whilst the time of the medical officer is needlessly and vexatiously wasted.

In urban sanitary districts the medical officer of health may have an office provided and maintained for him by the sanitary authority.

In his official correspondence with the Local Government Board he should address the Secretary. The Board request correspondents: (1) To quote the *number*, *reference letter*, and *date* of each letter to which they reply; (2) to let communications on different subjects, or relating to different unions or parishes, form separate letters; and (3) to use paper of *foolscap* size.

The *liabilities* of the medical officer of health should be clearly understood. He cannot be a member of any local authority by whom he is appointed. If he is appointed by a municipal corporation, any partner he may have is disqualified for being a councillor or alderman of that body. Section 198 of the Public Health Act, 1875, enacts that the medical officer of health, in common with other officers appointed under that Act by a local authority, shall not in any wise be concerned or interested in any bargain or contract made with that authority for any of the purposes of the Act. If any such officer is so concerned or interested, or, under colour of his office, exacts or accepts any fee or reward whatsoever other than his proper salary and allowances, he shall be incapable of afterwards holding any office under the Act, and shall forfeit and pay the sum of 50*l.*, which may be recovered by any person, with full costs of suit, by action of debt.

Before proceedings for the recovery of any penalty under this section can now be taken the consent in writing of the Attorney-General must be obtained, as provided for by the Public Health (Officers) Act, 1884; moreover, the section has been amended by section 2 of the Public Health (Members and Officers) Act, 1885, which enacts that it shall not be unlawful for any officer appointed under the Act of 1875, or a local Act, to be concerned or interested in any contract with the local authority, to be made in a manner prescribed in the section, for the sale, purchase, leasing, or hiring of any lands, rooms,

or offices, or to be concerned or interested in any contract with the local authority as a shareholder in any joint stock company, and that no officer shall be incapable of holding office or shall be liable to any penalty by reason only of his having been concerned or interested either before or after the passing of the Act in any such contract.

The enactment referring to the same subject in the metropolis is contained in section 64 of the Metropolis Management Act, 1855, which also provides that no person being a shareholder of any joint stock company shall be prevented from being employed as an officer by reason of any contract between such company and the board or vestry appointing him, or of any work executed by such company.

It has been decided, however, that a local authority is not precluded from giving an officer extra remuneration for any work done by him which is not included in his ordinary duties.

The medical officer of health is also subject to the provisions of the Public Bodies Corrupt Practices Act, 1889, which make it a misdemeanour, punishable by imprisonment with or without hard labour for any period not exceeding two years, by a fine not exceeding 500*l.*, or by both such imprisonment and such fine, together with other penalties, such as forfeiture of office and prohibition of holding any public office for seven years, to solicit or receive a reward in any shape or form for doing or forbearing to do any matter or transaction connected with the duties of his office, or to give or offer any such reward to any member, officer, or servant of any local authority within the United Kingdom; but a prosecution under that Act cannot be instituted without the consent of the Attorney-General.

The medical officer of health is protected by section 265 of the Public Health Act, 1875, which provides that no matter or thing done by any officer of a local authority *bona fide* for the purpose of executing the Act shall subject him personally to any action, liability, claim, or demand whatsoever; and that any expense incurred by him in so acting shall be paid for by the local authority out of the rates. A similar protection is given in the metropolis by section 124 of the Public Health (London) Act, 1891.

In the following pages the duties imposed upon the medical officer of health by the Public Health Act, 1875, and incorporated and other Acts, and by bye-laws made thereunder, are discussed, for the most part, in the order in which the subject-matter is found in the first-named Act, and unless otherwise stated they relate to the officers of both urban and rural sanitary districts. Subsequently any duties entailed upon him by other Acts of Parliament coming within the administration of local authorities are given.

It must be premised that the duties of the metropolitan officers are very similar to those of extra-metropolitan officers, their powers in some matters being derived from the same sources, but that they are chiefly regulated by the Public Health (London) Act, 1891, and by some Acts applying specially to the metropolis. It is not deemed necessary to make special references to those Acts in all instances.

The duties of officers in some places are, to a certain extent, affected by local Acts, but it would be beyond the scope of this article to enter into a discussion of those special cases, since they do not concern the medical officer of health in general.

Retail bakehouses, so far as their cleanliness and other sanitary conditions, and certain regulations for bakehouses established on or after June 1, 1883, are concerned, are placed under the control of sanitary authorities in urban and rural sanitary districts by section 17 of the Factory and Work-

shop Act, 1888, and in metropolitan districts by section 26 of the Public Health (London) Act, 1891.

The only bakehouses in urban and rural sanitary districts to which the above-named provisions apply are those within which the bread, biscuits, or confectionery which are baked are not sold wholesale, but by retail in some shop or place occupied together with the bakehouse, and in or in connection with which no steam, water, or other mechanical power is used for the purposes of the business; those in which any such power is employed, being 'factories,' are exempted.

In the metropolis all bakehouses which are 'workshops,' that is to say, 'any places in which are baked bread, biscuits, or confectionery, from the baking or selling of which a profit is derived,' and in which no mechanical power is employed, are included. Any belonging to the Crown are not exempted from the operation of the enactments, unless a Secretary of State declares them to be so during any public emergency.

The medical officer of health will have to see to the enforcement of these provisions, and for that purpose he has all such powers of entry, inspection, taking legal proceedings and otherwise, as an inspector under the Factory and Workshop Act, 1878. The powers he is likely to require to exercise are thus defined by section 68 of that Act: 'To enter, inspect, and examine at all reasonable times by day and night a . . . workshop. . . . To make such examination and inquiry as may be necessary to ascertain whether the enactments for the time being in force relating to public health and the enactments of this Act are complied with so far as respects the . . . workshop and the persons employed therein. . . . To exercise such other powers as may be necessary for carrying this Act into effect.'

There are other powers, dealing chiefly with employment and education, about which he need not concern himself.

In the metropolis he has the same power of entry into all bakehouses which are not 'factories,' to carry out the provisions relating to them, as he has into any premises for any purpose in relation to nuisances.

If he becomes aware of the employment of any 'child,' that is, a person under the age of fourteen, 'young person,' of the age of fourteen and under eighteen, or 'woman,' of eighteen and upwards, in any bakehouse he enters, he must forthwith give written notice thereof to the factory inspector of the district.

It appears as though taking legal proceedings in respect of these matters regarding retail bakehouses, in urban and rural sanitary districts, may devolve upon the medical officer, but it does not appear advisable that he should take any such proceedings without being authorised to do so, either generally or in any special instance, by the local authority; and if he is obliged to do so at any time he should obtain the assistance of the clerk of the authority.

The points to which he should direct his attention in the inspection of a retail bakehouse are:—Its state of cleanliness, and whether there is any accumulation of flour sweepings or other refuse within it; whether it is free from any effluvia arising from untrapped or improperly trapped drains, water-closets, earth-closets, privies, ashpits, urinals, or other nuisances; whether it is overcrowded while work is carried on, so as to be dangerous or injurious to the health of the persons employed in it; whether it is ventilated in such a manner as to render harmless, as far as practicable, any injurious impurities in the air generated in the course of baking; whether there are within it any conditions of drains, water-closets, earth-closets, privies, ash-pits, water-supply, nuisances, or other matters coming within the purview of the law relating to public health; whether the statutory requirements re-

specting the lime-washing, painting, or washing, as the case may require, of its interior, and respecting any adjoining sleeping-place on the same level, are complied with; and whether it is on sanitary grounds unfit to be used as a bakehouse.

In the inspection of any retail bakehouse first used on or after June 1, 1888, he should satisfy himself that there is no water-closet, earth-closet, privy, or ashpit, either within or in direct communication with it; that any cistern there may be for supplying water to such bakehouse is separate and distinct from any cistern supplying a water-closet; and that there is no opening into any drain for carrying off fecal or sewage matter within it.

He should also note whether any child, young person, or woman, is employed in any bakehouse which he inspects.

In addition to the provisions specially relating to retail bakehouses, he can exercise in respect to them the general powers concerning houses which he possesses, as explained in the next paragraph concerning other workshops and factories.

Factories and workshops, so far as the special enactments of the Factory and Workshop Acts affect them, do not concern the medical officer of health; but inasmuch as the definition of a 'house' in the Public Health Act, 1875, and the Public Health (London) Act, 1891, includes 'factories and other buildings in which persons are employed,' he may exercise in respect of them, in urban, rural, and metropolitan districts, all the powers relating to any house with which he is invested, except the provisions of section 91 (6) of the first-named, and section 2 (g) of the last-named Act, in the case of any factory subject to the provisions of the Factory and Workshop Act, 1878, relating to cleanliness, ventilation, and overcrowding; but this exception does not apply to any other factory, or to workshops.

Whenever he finds that the lime-washing, cleansing, or purifying of any workshop, or of any part of it, is necessary for the health of the persons employed in it, he must give a certificate to that effect to the sanitary authority. In the metropolis this provision applies to any workshop, other than a bakehouse, or factory which is not subject to the provisions of the Factory and Workshop Acts, and to any workplace.

If at any time he observes a child, young person, or woman being employed in any workshop, he must immediately give notice of the fact to the factory inspector of the district.

If a factory inspector finds any sanitary defect in a factory, workshop, or laundry, which can be dealt with under the law relating to public health, but not under the Factory and Workshop Acts, he has to give written notice thereof to the sanitary authority of the district in which the building is situated, and they have to take the necessary action. It will probably fall to the lot of the medical officer to inquire into the matter. A factory inspector may take him into any such place for the purposes of this provision, and may himself institute the proceedings if they are not commenced within a reasonable time.

The Factory and Workshop Act, 1891, gives both the medical officer of health and the inspector of nuisances in urban and rural sanitary districts, for the purposes of their duties with respect to enforcing sanitary provisions in workshops, all the powers of entry, inspection, taking legal proceedings or otherwise possessed by a factory inspector, which powers extend to workshops belonging to the Crown, unless a Secretary of State exempts them from the Acts in case of any public emergency.

In the metropolis these officers derive their power of entry under the Public Health (London) Act, 1891.

The duty of inspecting all workshops now devolves upon those officers.

In determining the number of persons who should be allowed to work in them, it has hitherto been the practice of the inspectors of the Home Office to require 250 cubic feet of space for each person, and 400 cubic feet when they work overtime; three ordinary gas-burners, or one gas iron-heater, where there is no provision for conveying away the products of combustion, should be considered to require the same space as one person.

The Secretary of State has issued an order, which came into effect on November 20, 1892, requiring the occupiers of factories and workshops engaged in the manufacture of articles of wearing apparel, cabinet and furniture making and upholstery work, the manufacture of electro-plate, and the manufacture of files, and every contractor employed by them, to keep lists of out-workers, which is to be open to inspection by the medical officer of health and inspector of nuisances or sanitary inspector.

The Local Government Board state that the officers should examine the lists from time to time, so that they may become aware of the places in which out-workers in those trades are employed, as they consider it is especially desirable that frequent inspection should be made of those places, so that prompt measures may be taken to deal with any sanitary evils existing in them.

The Housing of the Working Classes Act, 1890, has repealed nearly the whole of the numerous and complicated Acts which formerly related to artisans and labourers' dwellings and the housing of the working classes, and has consolidated and amended the laws relating to those most important subjects, which have such a direct bearing upon the health, well-being, both moral and physical, and happiness of the working classes.

Important responsibilities are cast upon the medical officer of health, and upon him chiefly will devolve the duty of putting the machinery of the Act into operation, which duty will now be more ready of execution than under the old order of things.

Part I. of the Act, dealing with 'unhealthy areas,' concerns medical officers of urban and metropolitan districts, and of the city and county of London, but not rural sanitary officers.

An 'official representation' for the purposes of this part of the Act means a representation made to the local authority by the medical officer of health of that authority, and in London made either by the medical officer of the county or by any medical officer of health.

It is the duty of a medical officer of health to make such representation whenever he sees cause to do so; and if two or more justices of the peace acting within his district, or twelve or more ratepayers, complain to him of the unhealthiness of any area within the district, he is obliged to forthwith inspect it, and to make an 'official representation' to the local authority stating the facts of the case, and whether, in his opinion, the area, or any part of it, is or is not an unhealthy area.

When a local authority have received an official representation from the medical officer of health that within any area within their district either any houses, courts, or alleys are unfit for human habitation; or the narrowness, closeness, and bad arrangement, or the bad condition of the streets and houses, or groups of houses within the area, or the want of light, air, ventilation, or proper conveniences, or any other sanitary defects, or one or more of such causes, are dangerous or injurious to the health of the inhabitants either of the buildings in the area or of the neighbouring buildings, and that the evils connected with such houses, courts, or alleys, and the sanitary defects in the area cannot be effectually remedied otherwise than by an improvement scheme for the rearrangement and reconstruction of the streets and

houses within the area, or of some of them, the local authority must take the representation into their consideration, and, if satisfied of its truth, and of the sufficiency of their resources, they have to proceed under the Act in the manner described in the article on the Law relating to the Public Health.

In the event of the local authority refusing to make an improvement scheme, the confirming authority may direct a local inquiry to be held, and a report to be made to them with respect to the correctness of the medical officer's official representation made to the local authority, and any matters connected with it on which they may desire to be informed.

Where a complaint of the unhealthiness of any area within his district is made to the medical officer of health by twelve or more ratepayers, as described above, and he fails to inspect such area, or to make an official representation with respect to it, or makes an official representation to the effect that in his opinion it is not an unhealthy area, the ratepayers may appeal to the confirming authority, who, on receiving satisfactory security for costs, must appoint a legally qualified medical practitioner to inspect the area, and to make a representation to them, stating the facts of the case, and whether, in his opinion, the area or any part of it is or is not an unhealthy area. The local authority have then to proceed in accordance with the finding of that representation.

Where a local inquiry is directed, the confirming authority have to send an officer to the area for the purpose of making an inquiry into the correctness of the medical officer's official representation to the local authority as to the area being unhealthy, and into other matters.

In the case of the illness or unavoidable absence of a medical officer of health a duly qualified medical practitioner may be appointed by the local authority for a period not exceeding six months, and subject to the approval of the confirming authority.

Part II. of the Act deals more particularly with 'unhealthy dwelling-houses,' as distinct from unhealthy areas. Under this it becomes the duty of the medical officer of health of *every* district to represent to his local authority any dwelling-house which appears to him to be in a state so dangerous or injurious to health as to be unfit for human habitation.

If in any district any four or more householders living in or near to any street complain in writing to the medical officer of health that any dwelling-house in or near that street is in a condition so dangerous or injurious to health as to be unfit for human habitation, he is obliged to inspect it forthwith, and to transmit to the local authority the complaint which he has received, together with his opinion upon it. If he is of opinion that the dwelling-house is in the condition described, he has to represent it to the local authority.

The absence of any such complaint, however, does not excuse him from inspecting any dwelling-house and making a representation upon it to the local authority.

If any local authority, not being within the administrative county of London or not being a rural sanitary authority, neglects to take action for three months after the receipt of the above described communications from the medical officer of health, the householders who signed the complaint may petition the Local Government Board for an inquiry, who, after that has been held, may order the local authority to proceed under this part of the Act.

It is incumbent upon every local authority to cause to be made from time to time inspection of their district, with a view to ascertain whether any dwelling-house within it is in a state so dangerous or injurious to health as to be unfit for human habitation.

The conduct of this inspection will devolve upon the medical officer of health, though he will probably receive assistance from the inspector of nuisances in the details of the work.

If he is of opinion that any dwelling-house, whether occupied or not, is unfit for human habitation, he will make a representation respecting its condition to the local authority, who must proceed as directed by the Act.

In the event of any such house being made fit for human habitation, however temporarily, he must admit it to be so, in spite of his knowing that the improvements cannot be of a lasting character.

If he, or four or more inhabitant householders, find that any building within his district, although not in itself unfit for human habitation, is so situate that by reason of its proximity to or contact with any other buildings it either—(a) stops ventilation, or otherwise makes or conduces to make such other buildings to be in a condition unfit for human habitation or dangerous or injurious to health; or (b) prevents proper measures from being carried into effect for remedying any nuisance injurious to health or other evils complained of in respect of such other buildings, he or the four or more inhabitant householders must represent to the local authority the particulars relating to the first-mentioned building, called 'an obstructive building,' stating that in his, or their, opinion it is expedient that it should be pulled down.

Such a representation must be dealt with by the local authority in the manner prescribed by the Act.

The medical officer will observe that an obstructive building may be of any class, that it need not necessarily be used for human habitation, and further, that it may be dealt with although it only conduces to the production of the unhealthy conditions complained of; it need not itself be the sole cause of those conditions, neither need the whole of it be implicated.

The evil conditions implied are such as the shutting out of light, sunshine, and air, conducing to darkness, dampness of site, and stagnation of air, either within or around the building, or the contraction of open space around buildings, preventing the construction of necessary out-offices, or conducing to general foulness of the atmosphere.

When a medical officer of health or any inhabitant householders make a representation or complaint, or give information to any vestry or district board in the administrative county of London, or to the Local Board of Woolwich, or to any rural sanitary authority, or to the medical officer of such authority, respecting any dwelling-house being in a state so dangerous or injurious to health as to be unfit for human habitation, or respecting an 'obstructive building,' or where a 'closing order' respecting any dwelling-house is made, the authority is required to send a copy to the county council, and if the county council, having received the prescribed information respecting the matter, are of the opinion that the district authority are in default in the performance of their duties under this part of the Act, they may, after giving due notice, replace that authority, and themselves take such action as they may deem necessary.

In that event their medical officer will have to advise them in the matter, and for the purposes here named he has the same right of admission to any premises as the officers of the 'district authorities' have for the purpose of the execution of their duties under the enactments relating to public health, and a justice may make the like order for enforcing such admission.

When a medical officer of any county makes a representation to the county council, and they forward it to any local authority in their county, except a borough as defined by the Municipal Corporations Act, 1882, it has,

for the purposes of this part of the Act, the like effect as a representation from the medical officer of health of the district.

In these respects the medical officers of county councils have acquired a certain control over medical officers of health of local authorities, and can to the specified extent supplant them in their offices. They will have to exercise these prerogatives with tact and discrimination, in order that friction between those officers and themselves may not arise.

Anything a medical officer has to do under Part I. or Part II. may be done by any person authorised to act temporarily as such officer.

He must make every representation in pursuance of the Act in writing.

When a medical officer of health makes an official representation to the London County Council, under the first part of the Act, relating to not more than ten houses, he will be directed by that body to represent the case to the local authority under the second part, and they will have to deal with it.

Where in the county of London a medical officer of health makes a representation under Part II. of the Act to any local authority, and they consider the matter to be of such general importance to the county of London that it ought to be dealt with under Part I.; or where an official representation under Part I. is made to the London County Council, and they resolve that the case is not of general importance to the county of London, and that it ought to be dealt with under Part II., the Secretary of State may direct a local inquiry to be held by an arbitrator, who will report to him, and he will then decide under which part of the Act the case is to be proceeded with. The medical officer or other proper officer will forthwith have to make the representation necessary for proceedings in accordance with such decision.

The London County Council may, with the consent of a Secretary of State, appoint one or more legally qualified practitioner or practitioners, for the purpose of carrying into effect any part of the Act; and those officers, as well as any medical officer of health appointed by that council, are to be deemed to be a medical officer of health of a local authority within the meaning of the Act.

Part III. of the Act: 'Working-classes Lodging-houses.' No special duties are cast upon the medical officer of health of any district in which it may have been adopted; but a lodging-house established under this part of the Act must at all times be open to the inspection of the local authority of the district in which it is situate, or of any officer from time to time authorised by such authority. It is possible that on special occasions it might be desirable for the medical officer to be so authorised to inspect any such lodging-house which may be in his district. He might also be called upon to advise the authority in framing any bye-laws for the regulation of such houses, or to assist in administering any which may be in force.

In order to determine whether any houses or areas within his district should be dealt with under the Act, the medical officer of health must ascertain by all the means at his disposal what is the actual state of the health of the inhabitants, so that he may be in a position to produce direct evidence upon that point.

It may be premised that any conditions which are even only dangerous to health may be dealt with.

In any area liable to be brought under this Act, probably a generally low condition of health and diseases tending to unduly shorten life will be rife, whilst various maladies will assume a low type; many of the denizens of the squalid dwellings will exhibit a sallow complexion and stunted growth, and will present evidence of injurious indulgence in stimulants, for their miserable conditions of life lead to excess in drink and other vices; there will be a high

death-rate, especially from zymotic and tubercular diseases, and from pulmonary affections and infantile diarrhoea; the mortality from those causes and the infant mortality will probably far exceed that of the more favoured parts of the district.

In forming an opinion upon these points the medical officer will be guided by the returns of births and deaths, with which he should always be regularly provided, and if notification of infectious diseases is compulsory in his district he will be greatly helped by it. With regard to general ailments, he may be aided by the returns of new cases of sickness amongst paupers, if he receives them; but he will probably endeavour to obtain information respecting them from any medical men practising in those particular parts of the district, or from the officers of any neighbouring dispensaries, hospitals, or other public institutions, and by direct inquiries made amongst the inhabitants.

A striking example of the excessive mortality of unhealthy areas is afforded by the annexed table, giving particulars respecting certain blocks of houses in Salford, about which official representations under Part I. of the Act of 1890 have been made by Mr. C. E. Paget, the medical officer of health, to whom the writer is indebted for the figures:—

No. of Block	Houses		Population	People per acre, including area of streets and courts	Average death-rates per 1,000 persons living for five years 1884-1888 inclusive		
	Inhabited	Un-inhabited			From all causes	From the six principal zymotic diseases (excluding diarrhoea)	From pulmonary diseases, including phthisis
1	182	17	615	360	57·9	5·9	25·4
2	68	16	240	377	40·8	5·8	19·2
3	77	5	110	468	78·2	14·5	21·8
4	21	21	97	233	41·2	4·1	14·4
5	19	2	68	319	52·9	8·8	14·7
In the same localities and with the same population in the years					1889	64·6	12·4
					1890	64·6	6·2
In the whole of the urban sanitary district of Salford estimated at the middle of 1888, 1889, and 1890					1888	23·0	3·0
					1889	23·0	3·7
					1890	25·1	3·2

In some unhealthy areas in Liverpool reported upon by Dr. J. Stopford Taylor, the average death-rate from all causes per 1,000 of the population for three years was 46·5, that of the whole city during the same period having been 28·7. Dr. Bate, in making an official representation respecting an unhealthy area in Bethnal Green, reported that the rate of mortality which prevailed in it was nearly double that of the parish generally, and that the mortality from zymotic and tubercular diseases even exceeded that proportion, as shown by the subjoined statement:—

	Bethnal Green, 1886-1888	Represented area, 1886-1888	Represented area, 1889
General mortality . . .	22·8 per 1,000	40·0 per 1,000	40·13 per 1,000
Zymotic diseases . . .	3·7 "	7·9 "	10·71 "
Tubercular diseases . . .	3·9 "	8·5 "	7·26 "

Many and varied are the conditions which may tend to bring single houses, or whole courts, alleys, or streets within the purview of the Act; no hard and fast lines can be laid down to control all cases. Houses, for instance, may be of such radically defective construction as to be past remedy; their walls may be of stud and mud,¹ of lath and plaster, of wood only, of old worn-out half-timber framing, of but four and a half inch brickwork, with no

¹ Or wattle and daub.

damp-proof course, the pointing probably perished, and with a general absence of window-sills, giving rise to a soddened condition from rising damp and driving rain, and so causing an all-pervading dampness, mouldiness, and fustiness. Other houses, although of more approved original construction, may through neglect, misuse, or from sheer lapse of time, have become so hopelessly dilapidated, with roofs leaking and ceilings falling, as no longer to be proof against wind and rain, their rickety stairs and floors endangering life and limb, or their general tumble-down condition making it an impossibility for them to be kept in a cleanly and healthy state. In some, the floors, perhaps below the ground level, may be paved with damp rotten bricks or tiles, or may have nothing to cover the bare earth, or if there be boarded floors they may be laid upon the earth, or there may be an entire absence of ventilation beneath them; perchance the basements or cellars may be reeking with moisture and abounding with fungoid life, and may be used as unhealthy underground dwellings. Others, again, may be so deficient in light and air, having no means of through ventilation, or being built back to back, as to be dangerous to health. Some may be found hopelessly overcrowded, with no chance of extra dwelling accommodation being provided on the spot, or so filth-sodden from top to bottom as to be past remedy by any ordinary process of cleansing. In the metropolis the absence of proper water fittings may be deemed to render a house unfit for human habitation. There may be insufficient closet accommodation, with no opportunity of having proper provision made. The paving or draining of the yards may be found perished or wanting, giving rise to general fouling of the subsoil; or the open space about the buildings may be inadequate for the maintenance of health.

What should be deemed a minimum space permissible around buildings may be gathered from the bye-laws on the subject in the model series respecting new streets and buildings (No. IV.) issued by the Local Government Board. The requirements of those for every new domestic building are: An open space in front, free from any erection above the level of the ground, except any portico, porch, step, or other like projection from the building, or any gate, fence, or wall, not exceeding seven feet in height, and which measured to the boundary of any lands or premises immediately opposite, or to the opposite side of any street which the building may front, shall, throughout the whole line of frontage of the building extend to a distance of at least twenty-four feet, measured at right angles to the external face of the building; and in the rear of the building an open space belonging exclusively to it, and of an aggregate extent of not less than 150 square feet, free from any erection above the level of the ground, except a water-closet, earth-closet, or privy, and an ashpit. This open space must extend laterally throughout the entire width of the building, and the distance across it from every part of the building to the boundary of any lands or premises immediately opposite or adjoining the site has to be in every case not less than ten feet, however low the building may be.

But now arises the question of a definite relation between the depth of the open space and the height of the building, which is a matter of much moment, and must always be taken into consideration. With regard to this, the bye-law goes on to state that when the height of the building is fifteen feet the above-named distance of open space must also be at least fifteen feet; not less than twenty feet when the building is twenty-five feet high; and at least twenty-five feet if the building is thirty-five feet high or over. It would, however, be desirable to have greater open space in connection with such very high buildings as are provided for in the bye-laws, extending up to 100 feet.

The height of the building has to be measured from the level of the ground of the open space to the level of half the vertical height of the roof, or to the top of the parapet, whichever may be the higher.

A better plan, and one which deals uniformly with all houses, whatever their height, is that which is in force in Liverpool. In accordance with it an open space ten feet in depth, and extending the whole width of the structure, must be left at the rear of every new house, and must not be built upon. Any structure may be erected upon the rest of the site so long as no part of it, with the exception of chimneys, dormers, &c., is allowed to extend above an imaginary line drawn from the posterior boundary of the premises towards the house at an angle of 45° with the horizon.

This regulation has been proposed for new dwelling-houses in London, and has been ably advocated and described by Dr. Longstaff, of the London County Council, as may be seen by referring to p. 99 of the fourth volume of 'Public Health.'

Courts or alleys may be found situated behind a front row of buildings, having at neither end an opening of full width extending from the ground upwards, the sole access to them being through narrow covered passages, leading to blind extremities.

Probably the courts are too narrow; and in judging of that the previously quoted bye-laws will again serve as a guide. Under those it would be impossible to construct any court less than twenty-four feet wide, or more than 100 feet long, for every new street not intended for use as a carriage road must be of at least that width, and must not exceed that length. Every new street of a greater length must be constructed for use as a carriage road, which has to be at least thirty-six feet wide. New streets of every description are required to have an entrance at one end at least of their full width and open from the ground upwards without any obstruction.

The courts or alleys may be found to be surrounded by buildings of great height, preventing the sun from ever shining in them, and shutting out light and air to such an extent that wells, as it were, of damp stagnant air are formed; there being no through currents, the drying and cleansing action of winds has not a chance of playing its beneficial part. Thus dampness of site is fostered, and impure products of respiration, combustion, and decomposition of organic refuse cannot be freely swept away, but accumulate, to add to, rather than to displace, the foulness of the air inside the dwellings.

The houses may be badly arranged, without any regard being had for healthiness of building or person. Blocks of back-to-back houses may fill the middle space of the courts; and if the houses at the sides are not constructed in that fashion, they may nevertheless have no doors or windows at their backs, there probably being no legal right for any to be opened out in them.

Very likely many of the houses are overcrowded, being sub-let to several families, whilst there may be no opportunity of providing more dwelling accommodation upon or near the site.

The open spaces, originally none too liberally bestowed, may have been subsequently encroached upon, or almost obliterated, by the erection of other buildings, so great is the greed of some owners, utterly regardless of the health or comfort of their miserable and probably helpless tenants. The density of the population may thus have been so increased, and the superficial area for each individual have become so diminished, as to be productive of disease, persons and houses being massed together without regard to the laws of health.

The closet accommodation may be inadequate, and yet, from want of space, there may be no chance of improving it.

Defective drainage, or uneven paving of the whole place, may lead to a prevailing state of filth or of dampness, whilst some sites may be so wet from natural causes as to be dangerous to health.

In some situations an impossibility of providing a proper supply of water may add to the unhealthiness of the places, and may become a factor in deciding the question of procedure.

Some of the evils may be found to be due to, or to be partially caused by, 'obstructive buildings,' and if the medical officer of health is of opinion that they should be pulled down, for the benefit of the health of the inmates of the neighbouring dwellings, even though they are not in themselves unfit for human habitation, he must make the necessary representation thereon to the local authority.

The unhealthiness of an area may be caused or aggravated by bad arrangements of the streets within its limits, owing to narrowness or excessive tortuosity. Thus in an unhealthy area, in Bethnal Green and Shoreditch, dealt with under the Act of 1890 by the London County Council, the streets varied in width from only eighteen to twenty-eight feet.

With high buildings extreme narrowness of streets becomes a still more serious blemish; so that the relation between the elevation of buildings and width of streets is an important consideration in the condemnation of any area.

In any improvement scheme under this Act all new streets should be of ample width, and there should be some relation between their width and the height of the proposed new buildings, in order that adequate open space may be secured for any class of buildings which may be erected.

It is amongst some such conditions as those which have been discussed that we must look to enable us to deal with any houses or areas under the Act; but it must not be imagined that all, or even the larger proportion of them, must be found before we can take action. No definite rules can be laid down to meet all cases; the medical officer must use his discretion in the matter. When he has made an official representation it will rest with the sanitary authority to decide whether the buildings in question are in a state to be made good, or whether they should take steps to have them demolished.

In metropolitan districts it will have to be decided whether any particular case is to be dealt with under Part I. or under Part II. of the Act. In coming to a decision as to what advice he should give on this point, the medical officer of health must be guided by considerations arising from the general attending circumstances.

If the case is not of general importance to the metropolis, the houses implicated being but few in number, their unhealthy influence being circumscribed and localised, affecting the inmates of the houses rather than the inhabitants of a more extended area; if the evils can be cured by the pulling down of any 'obstructive building'; if the benefits arising from the improvement of the houses, or from the demolition of them, or of any 'obstructive buildings,' or from the dedication as highways or open spaces of the ground previously occupied by the buildings, would apply to the inhabitants of the neighbouring houses, but would not be calculated to extend beyond the limited area affected; if the reconstruction of the houses more or less upon their former sites would effectually remedy the evils, if, in fact, the area comprising the buildings is too small to be dealt with as an unhealthy area, under Part I., and more particularly if the site is so small as to necessitate

but an inconsiderable provision of dwelling accommodation for rehousing the displaced persons of the working class, then he will advise proceeding under Part II. of the Act.

If, on the other hand, he finds the case is of general importance; that many houses have to be dealt with, or that the construction and arrangements of whole courts, alleys, or streets are intrinsically bad from original defects of design or from subsequent over-building, several owners perhaps being concerned in the property and no one of them having the power by himself to make the necessary alterations; that the unhealthy conditions of life thereby engendered are a standing menace to a more extended area; that the demolition of a few houses will not suffice, but that nothing short of a clean sweep and rearrangement and reconstruction of the whole or a part of the streets and houses in the area will satisfy the requirements of the case; and that a large portion of the district would reap a direct or indirect benefit from an improvement scheme, he will find that the provisions of Part I. will have to be resorted to, especially if the scheme is so large as to require an extensive rehousing of the displaced inhabitants.

Overcrowding, &c.—It will be the duty of the medical officer to promote the steps necessary for the abatement of any overcrowding in houses, or other nuisances in connection with dwellings, endangering health in his district.

In the metropolis he has to certify to the sanitary authority that any house is overcrowded before they can take proceedings for abating the overcrowding.

There are ample powers for preventing overcrowding; but it is a complex subject, and not infrequently a very difficult one to deal with effectually. Often there will be found an insufficiency of dwelling accommodation for the working classes, or, owing to dire poverty or high rents, the working man with a large family finds it quite beyond his means to procure sufficient accommodation. Under such circumstances the efforts of the medical officer to prevent overcrowding may be rendered nugatory, or, even if he succeeds in eradicating it in one place, the result may be simply to perpetuate the evil in another part. He must, therefore, use discretion in dealing with these cases, and have regard to all the attending circumstances of the districts in which they are found. He should endeavour to secure 800 cubic feet for each occupant of a bed-room.

The laws relating to public health do not take cognisance of destitution, or of questions of morality, except so far as they permit bye-laws to be made for the separation of the sexes in certain lodgings and in canal boats used as dwellings; nevertheless, when any such instances come within the medical officer's ken he will do well to bring them to notice in the appropriate quarters.

In any urban or rural sanitary district the medical officer may certify to the local authority, under the 46th section of the Public Health Act, 1875, that any house or part of a house is in such a filthy or unwholesome condition that the health of any person is endangered; the authority, if satisfied of the correctness of the certificate, must then get it whitewashed, cleansed, or purified, as the case may require.

Where bye-laws with respect to the closing of buildings or parts of buildings unfit for human habitation, and to prohibition of their use for that purpose, are in force, the medical officer may have to report to the sanitary authority on any such buildings erected after the date of confirmation of the bye-laws, and to certify that the buildings or part thereof are unfit for human habitation, so as to enable the authority to give the necessary notice and order for enforcing those bye-laws. The considerations which would

guide him in this matter are such as have already been discussed in connection with unhealthy dwellings.

When in the metropolis or in any urban sanitary district the medical officer becomes aware of any ruinous or dangerous buildings he will do well to report them without delay to the surveyor, who will then take such proceedings as may be required.

Cellar dwellings in urban and rural sanitary districts are under the control of the respective sanitary authorities. Sections 71 to 75 of the Public Health Act, 1875, relate to this subject; the medical officer of health is not named in them, but if he finds within his district any cellar dwelling occupied in contravention of them he should see that the offence is discontinued, or should report the matter to the local authority.

Underground rooms occupied as dwellings in the metropolis are subject to the control of the sanitary authorities, and are dealt with under sections 96 to 98 of the Public Health (London) Act, 1891. The medical officer may, amongst other officers, be appointed by the sanitary authorities to report to them all cases in which underground rooms are occupied contrary to the Act, and in that event he will be able to enter and inspect them at any hour by day, or between any hours specified in the warrant of a justice, if the occasion should arise for him to obtain one.

Common lodging-houses are placed under the control of both urban and rural sanitary authorities by sections 76 to 89 inclusive of the Act of 1875. The systematic inspection of them will not fall to the lot of the medical officer of health, but he should make himself practically acquainted with all that may be within his district, and should be not only able but willing at all times to assist the inspector of them with his advice respecting any questions that may arise in connection with them.

A house cannot be registered as a common lodging-house until it has been inspected and approved for the purpose by some officer of the local authority; and probably the medical officer will have to inspect any house in his district proposed to be registered as such, and to express to the authority his approval or disapproval of it for the purpose, as the case may require. What should be the nature of the inspection and what conditions should influence him in giving or withholding his approval of any house are tersely given in the preface of the Local Government Board to their model bye-laws for common lodging-houses (Series No. III.). In that they say:— 'The numbers for which the house and each sleeping room may be registered will depend, partly upon the dimensions of the rooms and their facilities for ventilation, and partly upon the amount of accommodation of other kinds. In rooms of ordinary construction to be used for sleeping, where there are the usual means of ventilation by windows and chimneys, about 800 cubic feet will be a proper standard of space to secure to each person; but in many rooms it will be right to appoint a larger space, and this can only be determined on inspection of the particular room.'

The medical officer of health must be prepared to advise the local authority on any points relating to health involved in the framing and execution of any bye-laws for the regulation of common lodging-houses which they may make under the powers of the Public Health Act. It is to him that the keepers of those houses have to give notice when any person is ill of fever or any infectious disease within them. He is to have free access at all times to the whole or any part of a common lodging-house.

Houses let in lodgings, other than common lodging-houses, in the metropolis and in any urban or rural sanitary district, may be regulated by bye-laws, in respect of which the medical officer of health may have to advise the

local authority, and under which he will have power of entry into such houses. It is not easy to define what class of houses should be included under this heading; better class lodging-houses, of course, cannot be included.

The Local Government Board have attempted to solve the question by inserting in their series of model bye-laws (No. XIII.) an exemption clause, relating to both unfurnished and furnished houses, which assumes that all houses below a certain rateable value will, if let in lodgings or occupied by members of more than one family, be within the scope of the bye-laws; and which confers exemption in the case of houses of higher rateable value, if the rent of each lodger exceeds a certain minimum.

It is intended that every local authority should determine for themselves what limits of rateable value and rents the circumstances of their district would require to be fixed upon; but this is not a satisfactory solution of the difficulty, and it still remains for one to be devised. In the St. Giles's District, London, the regulations as to lodging-houses require that the clerk of the authority shall keep a register of all lodging-houses in the district which the authority shall have directed to be entered upon it. Dr. Lovett, the medical officer of health, states that the regulations are applied only to the poorer tenements, of which some 553 are registered, and that rateable value has nothing to do with the question. He picks out the streets and houses and asks the sanitary committee to put them under the regulations, which were made in 1887, after the authority had failed in an attempt to enforce some they had previously framed on the basis of the model bye-laws above alluded to. These regulations have been carried out without friction and with satisfaction to the authority.

Tents, vans, sheds, and similar structures used for human habitation, are now under the control of metropolitan, urban, and rural sanitary authorities. Bye-laws may be made for promoting their cleanliness and habitable condition, for preventing the spread of infectious disease by the persons inhabiting them, and generally for the prevention of nuisances in connection with them. The medical officer of health may be called upon to advise the local authority on points relating to the framing and execution of such bye-laws. He must bear in mind that he will have no concern with any tent, van, shed, or structure, erected or used by any portion of Her Majesty's military or naval forces.

He may also have to advise the authority of an urban or rural sanitary district, on any matters affecting health, involved in the compilation and execution of any bye-laws they may make, under the Public Health Act, 1875, for securing the decent lodging and accommodation of hop-pickers, or under the Public Health (Fruit Pickers' Lodgings) Act, 1882, for securing the decent lodging and accommodation of persons engaged in the picking of fruit and vegetables.

The Local Government Board have issued a model series of bye-laws for the former, and have sanctioned bye-laws for the latter purpose, but neither in them nor in the Acts under which they are framed is the medical officer of health, or any other officer, authorised to enter any structure for the purpose of inspection, or of enforcing such bye-laws; whatever power of entry to tents, sheds, &c., used for human habitation, he possesses, is derived, in urban and rural sanitary districts, from the Housing of the Working Classes Act, 1885, but that does not refer to bye-laws made under the above-named statutes.

Seamen's lodging-houses may be controlled by bye-laws made under the Merchant Shipping (Fishing Boats) Act, 1883, by the London County Council, and, outside London, by any sanitary authority within whose district any seaport town is situated. Those bye-laws may provide, amongst other

things, for the inspection and sanitary conditions of the houses; and may confer power of entry into them, upon the medical officer of health, who might be called upon to assist the authority with reference to any questions of health in connection with them.

Canal boats used as dwellings are subject to registration, and to regulations made by the Local Government Board, under the provisions of the Canal Boats Acts, 1877 and 1884. The medical officer of health may, if duly authorised by a registration or sanitary authority, or by a justice of the peace, on producing (if demanded) a copy of his authorisation, signed by the clerk or a member of the authority, or some other sufficient evidence of his being so authorised, enter by day, that is between six o'clock in the morning and nine o'clock at night, any canal boat in order to ascertain whether there is on board any contravention of the Acts or any person suffering from an infectious disorder, if he has reasonable cause to suppose such to be the case; and he may, if need be, detain the boat for the purpose, but for no longer time than is necessary.

Exemption from inhabited house duty of houses used solely for providing separate dwellings at small specified rents, and a reduction of the duty in some instances, are provided for by the Customs and Inland Revenue Acts, 1890 and 1891, but there is required for that purpose the certificate of a medical officer of health, to the effect that any such house is so constructed as to afford suitable accommodation for each of the families or persons inhabiting it, and that due provision is made for their sanitary requirements.

On the request of the person who would be liable to pay the duty, if it were not remitted, the medical officer must examine the house for the purpose of ascertaining whether such a certificate can properly be given, and if he finds it is constructed so as to afford such accommodation and sanitary requirements, he must certify to that effect. He cannot charge any fee for the examination or certificate; but if much extra work is thrust upon him in carrying out this duty, the sanitary authority may, if they think fit, grant him some extra remuneration; and if they consider that his ordinary duties would be interfered with thereby, they may appoint some other properly qualified medical practitioner for the purpose.

A new and invidious duty has thus been thrown upon the medical officer, which he must exercise judiciously, bearing in mind that there is no appeal from his decision. On the one hand, he must not be too exacting in his requirements in dealing with old property, but, on the other hand, he must not be too lenient in granting certificates. The Local Government Board have stated that they do not consider a certificate under these Acts 'should be construed as meaning that the sanitary requirements are perfect in every detail according to the latest development of sanitary science.'

The *water-supply* of his district will naturally engage the attention of the medical officer of health, and he will frequently be called upon to advise the local authority upon various points in connection with it.

It behoves him to bestow much thought and attention upon a matter so vitally affecting the public health. Where works have been established by a local authority or by a private company, there will not be much difficulty in securing a general supply, if the source of the water is a safe one; but it is often far otherwise in rural or small urban districts, where the cost of initiating a proper supply, owing to natural obstacles in the way of reaching suitable water, may be prohibitory. In that case possibly the most he can do will be to endeavour to improve the surroundings of the wells yielding dangerous water, though he will often find that the result will be anything but reassuring. To recommend that a well which yields impure water

should be merely cleaned out in order to prevent the pollution, as is sometimes done, indicates an entire want of practical experience in the matter. We have known such advice to be given and followed, with the melancholy result that the well has yielded worse water after the cleansing operation than it did before. It is not what is in the well that contaminates the water, unless indeed there may be any dead animal in it, but it is the filth-sodden soil through which the water has to pass on its way into the well which renders it impure; and that condition remains unaffected by the cleansing of the well. In thickly inhabited parts, surface wells rarely yield reasonably safe water, and when the medical officer finds this to be the case, it will be his duty to urge the local authority to provide an extraneous supply of water whenever practicable.

Under the 62nd section of the Public Health Act, 1875, any urban or rural sanitary authority may, on the report of their surveyor, require houses to be supplied with water in certain cases. It must be observed that if a medical officer of health were to report that any house in his district was without a proper supply of water, the local authority could not take action under that section, since it mentions only the report of the surveyor, although the one officer would probably be as capable of giving a correct opinion upon the subject as the other. In rural sanitary districts, however, the medical officer of health may report to the local authority any occupied dwelling-house which has not an available and sufficient supply of wholesome water, in the manner to be presently described.

Local authorities can take legal proceedings for closing, or otherwise dealing with, any public or private well, tank, or cistern, or public pump yielding water likely to be used by man for drinking or domestic purposes, or for manufacturing drink for the use of man, which is so polluted as to be injurious to health. The representation required for the institution of those proceedings may be made to the authority by a medical officer of health in the metropolis under the 54th section of the Public Health (London) Act, 1891, and in urban and rural sanitary districts under the 70th section of the Public Health Act, 1875.

The Public Health (Water) Act, 1878, is in force in rural sanitary districts only, unless the Local Government Board have invested the authority in an urban sanitary district with any powers and duties of a rural sanitary authority under the Act, and if they are not so invested their medical officer of health can take no action under it.

The medical officer in a rural sanitary district may have to report to the local authority any occupied dwelling-house which has not within a reasonable distance an available supply of wholesome water sufficient for the consumption and use for domestic purposes of the inmates, and then the authority must act in accordance with the 3rd section of the Act. In the case of dwelling-houses erected or rebuilt after March 25, 1879, he will have to advise the authority as to whether there is provided an available and sufficient supply of wholesome water, so as to guide them in giving or withholding their certificate under the 6th section, which the owners of such houses must obtain before they can be legally occupied. He may have to ascertain the condition of the water-supply of the district for the guidance of the sanitary authority.

If he has reasonable ground for believing that any occupied dwelling-house within the district is without a sufficient supply of wholesome water, he must be admitted, between nine in the forenoon and six in the afternoon, into the premises for which the supply is required, or from which the water-supply may be derived, for the purpose of ascertaining whether or not the house has

such a supply within a reasonable distance. For the purposes of this admission sections 102 and 108 of the Public Health Act, 1875, apply in the same manner as if the admission were necessary for the purpose of examining as to the existence of any nuisance on the premises.

A certificate respecting the water-supply of every house built, or rebuilt, after the commencement of the Public Health (London) Act, 1891, is required also in all metropolitan districts; and there the medical officer of health may have to report any occupied house without a proper water-supply as being a nuisance and unfit for human habitation.

Analysis of water and air does not fall within the duties of the medical officer of health; this has been decided by the Local Government Board on various occasions. In 1874 they wrote to a sanitary authority: 'The Board understand the question of the guardians to be whether it forms part of the duty of the medical officer of health to make a chemical analysis of water, and, having given the matter their careful consideration, they are of opinion that he cannot be required to undertake such an examination as that referred to.'

Replying to an inquiry from a person in 1887, they stated that, 'while it is the duty of the medical officer of health to inform himself as to the character of the water-supplies in his district, and for this purpose to use, where needful, the ordinary chemical tests of the purity or otherwise of the water, he is not required to make a quantitative analysis of the water. If this is required, such analysis should, in the absence of any special arrangement between the sanitary authority and the medical officer of health, be procured from an analytical chemist.' Again, in the same year, the Board's secretary wrote to a sanitary authority: 'With reference to the proposal to require the medical officer of health to make chemical analyses of water, I am directed to state that, while it is the duty of the officer to inform himself as to the character of the water-supplies in the district, and for this purpose to use, where needful, the ordinary chemical tests of the purity or otherwise of the water, he is not required to make a quantitative analysis of the water. If this is required, such analysis should, in the absence of any special arrangement between the sanitary authority and the medical officer of health, be procured from an analytical chemist. The Board do not consider it expedient to require analysis of air from medical officers of health part of whose salaries are repaid by them out of the Parliamentary grant.'

But the less the medical officer relies upon such a broken reed as those ordinary chemical tests, whatever they may be, the better will it be for his own reputation and for the well-being of his district, inasmuch as they have often been the means of causing medical officers of health to make gross blunders in regard of the quality of water which they have professed to analyse or examine.

Generally speaking, the less the medical officer of health has to do with the analysis of water the better it will be for him, and he will find that if once the public imagine they have the right to have their water-supplies analysed by him, his whole time may be taken up in satisfying their curiosity. He must not trust too implicitly to the results of analysis, but should often be guided rather by consideration of the general conditions under which the source of the water is placed, and the possibility of its being subjected to the chance of dangerous contamination. Because a water may afford fairly good analytical results at one time is no proof that at others it may not be unsafe or injurious. He should make himself intimately acquainted with the natural composition of the water derived from any different geological formations there may be within his district; he will thus, and thus only, be in a position

to put a reliable construction upon the results of every analysis of water derived therefrom, inasmuch as very slight deviations from those natural standards may be sure indicators of danger; and he must be ever mindful that the analysis of water, taken by itself, may furnish valuable *positive*, but not always reliable *negative*, evidence as to danger and pollution.

Sewerage and Drainage.—There are no statutory duties imposed upon the medical officer of health under this heading, but he must be well acquainted with the general principles of modern sewerage and house drainage; he must understand the conditions which are liable to render them dangerous to health, and must be able to point out the directions in which the remedies for such defects are to be sought; whilst at the same time he must carefully avoid going beyond his own duties or trenching upon those of the surveyor or engineer in those matters. He must be competent to advise the local authority upon the requirements of their district in these respects. It is a question he will find often needing earnest consideration, for while in thickly populated towns there can be no question as to the necessity of providing sewerage, it is frequently a much more difficult matter to decide in smaller urban districts and villages, where local circumstances or difficulties in the way of readily purifying sewage may be such as to entail an expenditure which could not be borne in the event of such works being made. Take, for instance, the case of a small place where the only natural outlet would be into a river; if it were sewered, the sewage could not be conveyed into the river in an unpurified state, because that would be illegal, so the necessity of pumping it would arise, and thus not only would there be an additional initial expenditure for erecting the pumping machinery, but there would be a continuous outlay for working expenses. In another place the sewage might be conveyed by gravitation on to some suitable land, and be purified at no great cost, and there the work of sewerage might be quite feasible. It is plain that every place must be dealt with on its own merits; one rule cannot always be followed, and the medical officer will ponder well before coming to any decision as to what his advice should be.

He must be able to make a thorough examination of the drainage of any house, and to point out any defects there may be, and the proper remedies for them. Urban sanitary authorities, and rural sanitary authorities invested with urban powers, may regulate the drainage of new buildings by bye-laws, and he must be prepared to advise them on any questions affecting health which may arise in connection with the framing or working of such bye-laws, but in the model series (No. IV.) dealing with this matter, issued by the Local Government Board, power of entry for the purpose of inspection is given to the surveyor only, and not to the medical officer.

He must be capable of advising the local authority upon matters connected with the disposal and purification of sewage, and to that end he must be practically acquainted with the various processes of sewage purification which are in use, and he should have a knowledge of others which have been proposed from time to time.

The 36th section of the Act of 1875 gives to all urban and rural sanitary authorities the power to enforce provision of sufficient water-closet, earth-closet, or privy and ashpit accommodation, if it appears to them by the report of their surveyor or inspector of nuisances that any house within their district is without such accommodation. Although the medical officer of health is not named in this section, he may, by virtue of the 191st section of the Act, exercise the power of reporting under it. Similarly in the 41st section the surveyor and inspector of nuisances are alone named as being able to enter premises, when empowered by any local authority, for the purpose of examining any

drain, water-closet, earth-closet, privy, ashpit, or cesspool, which may be alleged by any person to be a nuisance; and it would appear that the medical officer cannot be so empowered, but he, amongst other persons, may make the written application to the local authority which is required before the powers of the section can be put into operation.

The removal of house refuse at regular and frequent intervals has an intimate relation with the prevention of disease and ill-health. It is incumbent upon the medical officer to see that refuse is not allowed to remain and decay in the vicinity of inhabited dwellings. In thickly populated parts he will probably find it necessary to advise the local authority, if they have not already done so, either themselves to undertake or to contract for the removal of house refuse from premises, and the cleansing of earth-closets, privies, ashpits, and cesspools. The adoption of these measures has been many times clearly proved to immensely improve the public health of places where preventable disease had previously been rife; diminished mortality and a generally bettered state of health having rapidly ensued. The medical officer should discountenance the toleration of large foul ashpits in all populous places, and should endeavour to have them replaced by small moveable receptacles; however often an ashpit may be emptied, it cannot fail to be in a foul condition ready to induce active decomposition in all putrescible matters put into it.

He may have to advise the local authority on any question relating to health involved in the framing and subsequent working of any bye-laws they may make, under the 44th section of the Act of 1875, respecting the cleansing of footways, the removal of house refuse, the cleansing of earth-closets, privies, ashpits, and cesspools, and, in urban districts, respecting the prevention of nuisances arising from snow, filth, dust, ashes, and rubbish, and for the prevention of the keeping of animals on any premises so as to be injurious to health.

In the metropolis the medical officer of any sanitary authority, or of the county council, may be concerned with similar bye-laws made under the 16th section of the London Public Health Act of 1891.

The pollution of rivers, streams, canals, ponds, lakes, and water-courses may demand the attention of the medical officer of health of any district, and he may be called upon to advise the local authority with reference to the discharge into them of unpurified sewage or of washings from gasworks, and also on questions arising under the 48th section of the Act of 1875, respecting any proceedings which may be requisite for enforcing the cleansing of offensive water-courses or ditches forming the boundary between different districts.

Although no statutory duty is imposed upon him by the Rivers Pollution Prevention Act, 1876, he may nevertheless have to advise the local authority in any district, or any county council, on any points which may arise with reference to enforcing that Act, and he must be acquainted with the character of the liquids proceeding from any factories or manufacturing processes within his district, so that he may be able to determine whether they would be in any way injurious if admitted into the sewers of the local authority, and to indicate the measures calculated to prevent any nuisance or pollution arising from them.

Nuisances are dealt with in urban and rural sanitary districts under the Public Health Act, 1875, and in the metropolis under the Public Health (London) Act, 1891. They are defined by the 91st and 92nd sections respectively of those Acts.

It is not within the scope of this article to discuss what the nature of a

nuisance must be in order to bring it within the jurisdiction of a local authority, but it is quite necessary that the medical officer of health should thoroughly understand this question.

Even if extensive structural works are not undertaken by a sanitary authority, very much may still be done towards bettering the health condition of any district by a vigorous onslaught upon all discoverable nuisances, and by an unremitting vigilance directed against their recurrence or fresh origin. In this regard much will depend upon the action of the medical officer.

It is the duty of every local authority to cause to be made from time to time inspection of their district for the detection and abatement of any nuisances which may exist within it. Although the medical officer of health may have to assist in that inspection, it is not in general his work to search his district for nuisances; that comes within the province of the inspector of nuisances. Nevertheless, that officer will often consult him regarding them, and he must be able and willing to render advice and assistance when asked to do so. He must assure himself that the inspector is thorough and impartial in executing the important duties with which he is intrusted.

He should make sure that nothing savouring of vexatious action is ever permitted, and that every legitimate effort is exhausted before the aid of the law is invoked for the abatement of any nuisance; for nothing is so calculated to hinder the progress of public health work as excess of zeal, officiousness, or indiscretion on the part of those charged with the enforcement of the laws in that behalf.

Information of nuisances may be given to the local authority by him. In some instances he reports on all cases requiring an official order for the abatement of nuisances; and in doing so he must be careful not to bring forward any matter which he has not personally inquired into; in other instances those cases are reported by the inspector of nuisances.

He has no statutory power to order the abatement of nuisances. In any case in which the inspector may have failed in his endeavours to get a nuisance abated, he will do his best to bring about that end; and a communication from him to the responsible person, couched in courteous terms, will frequently effect the desired result; but, if not, the matter will have to be reported to the local authority, to be dealt with by them. In that event all unnecessary publicity of names should be avoided.

He should make it his object to have the provisions of the laws of public health enforced within his district in such a manner as to avoid recourse to legal proceedings as far as possible, but these must not be shirked if he fails to attain his object in other ways.

He should see that the statutory powers are not exceeded, that any powers which go beyond them are not assumed, and that no notice for the abatement of any nuisance which cannot, if needful, be legally enforced is ever served.

Power of entry to all premises, and to ships not under the command of any officer bearing Her Majesty's commission or belonging to any foreign Government, for the purpose of carrying out any duties in connection with nuisances, is given to the medical officer of health in urban and rural sanitary districts by sections 102 and 110 of the Public Health Act, 1875.

For the purpose of examining as to the existence of any nuisance on any premises or of enforcing the provisions of any Act requiring fireplaces and furnaces to consume their own smoke, he may enter at any time between the hours of nine in the forenoon and six in the afternoon, or, in the case of a nuisance arising in respect of any business, then at any hour when such business is in progress or is usually carried on. Where a nuisance has been

ascertained to exist, or an order of abatement or prohibition has been made, he must be admitted to the premises from time to time between the above-named hours until the nuisance is abated, or the works ordered to be done are completed.

Where an order of abatement or prohibition has not been complied with, or has been infringed, he must be admitted from time to time at all reasonable hours, or at all hours during which business is in progress or is usually carried on, into the premises where the nuisance exists, in order to abate it. If he is refused admission to premises for any of these purposes he must give reasonable notice in writing to the person having custody of the premises of his intention to make a complaint to a justice, and on making the complaint on oath, such justice may, by order under his hand, require that person to admit him into the premises during the hours already named. If no person having custody of the premises can be found, the justice must, on oath made before him of that fact, give him a similar order authorising him to enter the premises during the same hours. Any such order made by a justice will continue in force until the nuisance has been abated or the work for which the entry was necessary has been done.

A similar power of entry to tents, vans, sheds, or similar structures used for human habitation, but not erected or used by any portion of the military or naval forces, is given to him by section 9 of the Housing of the Working Classes Act, 1885, when he is duly authorised by the sanitary authority, or by a justice of the peace, and on producing such authorisation if demanded, between six o'clock in the morning and nine o'clock in the evening.

The local authority should pass a resolution, to be entered on their minutes, giving him this authorisation, and he should possess a copy of it certified by their clerk, so that he may be able to produce it if demanded.

Like powers of entry are given to the medical officer of health in metropolitan districts by virtue of sections 10, 95, 110, and 115 of the Public Health (London) Act, 1891; but in all cases he must be able to produce, if required, some written document, properly authenticated on the part of the sanitary authority, showing his right to enter; and he can enter all premises, vessels, tents, vans, &c., with the exceptions above alluded to, between six o'clock in the morning and nine o'clock in the evening; whilst a warrant of a justice may authorise him to enter any house or part of a house alleged to be overcrowded, at any hour of the day or night specified in the warrant; and the warrant of a justice may authorise him to enter any premises, if need be by force, and with such assistants as he may require.

A medical officer of health in the metropolis or in any urban or rural sanitary district may have to take cognizance of nuisances arising without his district, in the event of the local authority dealing with such under section 14 of the Act of 1891, or section 108 of the Act of 1875.

In urban sanitary districts he will, as occasion may arise, have to advise the sanitary authority on the abatement, under the 47th section of the Act of 1875, of nuisances caused by the keeping of any swine or pigsty in any dwelling-house or so as to be a nuisance to any person, by the accumulation of waste or stagnant water within any dwelling-house, or by the overflow or soakage of the contents of any water-closet, privy, or cesspool; and it may be remarked, there is here no allusion to injury to health.

In metropolitan districts he may be concerned with the prohibition of the keeping of swine in unfit places, and any premises within forty yards of any street or public place are deemed to be unfit; or the keeping of any animal in unfit places, under the powers of the 17th and 18th sections of the Act of 1891.

Under the 49th section of the Act of 1875, in urban districts, and the 35th section of the Act of 1891, in metropolitan districts, the sanitary inspector can, if it appears to him to be necessary, give notice for the removal of any accumulation of manure or other filth, without obtaining an order from the sanitary authority. Although the medical officer of health is not named in those sections, he will nevertheless be able to give such notice, if the occasion should arise, by virtue of the power conferred upon him by the 191st section of the former and the 106th section of the last-named Act.

In urban, rural, and metropolitan districts he may be called upon to deal with smoke nuisances, as defined by the 91st section of the Act of 1875 and the 23rd and 24th sections of the Act of 1891. He must, therefore, make himself practically acquainted with the various means which have been devised for the prevention of those nuisances, in order that he may be prepared to give sound advice upon the subject.

When examining any premises for the purpose of ascertaining their fitness for habitation, he must use method in his investigation. Let him please himself as to whether he will commence the examination within or without, at the top or at the bottom of the house, but, having made up his mind, he must adhere to his plan in order that he may not overlook any point which should be observed, and he must enter his observations in his note-book at the time he makes them.

Suppose he elects to begin his examination with the outside and surroundings of the premises, he will direct his attention to the following matters, and more or less in the order in which they are here enumerated:—The character of the subsoil, whether porous or impervious, dry or damp; the elevation and aspect of the site, its surroundings, its relations to trees or neighbouring woods, and, if closely built over, any obstructive buildings and their effect upon the light and free circulation of the air; the paving of yards, whether sound or allowing of the accumulation of stagnant water or the soaking of filth; the drainage, if any, whether it discharges into a cess-pool or sewer, or otherwise; if into the former, the construction, means of ventilation, state, and frequency of emptying of the cesspool; if into a sewer, the quality and condition of the sewer, whether the system it is connected with is properly ventilated, cleansed, and flushed; whether there is any air-disconnection of the drain before such discharge, and if so, whether it is properly constructed and free from deposit, and the air-opening free from obstruction; whether the drain is constructed of suitable material, with sufficient fall; whether it is watertight, clean, free from deposit and properly ventilated; whether there are any means for efficiently flushing it; whether there are any loose iron traps on it, or any untrapped openings, except such as may be provided for its ventilation; whether any drain or soil-pipe ventilator ends near to any chimney-top, window, or other opening into the house; if there is any such ventilator, whether it ends under the roof-eaves or against any wall, or whether it is freely exposed, as it ought to be; the sufficiency or otherwise of closet accommodation; the disposal of excrement, if by water-closets, whether those situated out of doors are suitably constructed and ventilated, properly supplied with water with separate flushing cisterns, and whether clean and in good order; if the disposal is by any other method, the situation, condition, and frequency of emptying of the closets, and whether they would be liable to injure health; the disposal of house refuse, and if into an ashpit, its situation, condition, and times of emptying, and whether liable to injure health; if by a moveable receptacle, by whom and how frequently emptied, and the place of deposit of the refuse; if there is a basement in the house, whether there is a dry area around it, and if so whether it is drained

so as to prevent dampness, and whether drained into the house drain or otherwise ; the construction of the walls, their foundations and thickness, whether there is any rising damp or any damp-proof course ; if of brick, the condition of the pointing ; whether any cracks or crevices allowing rain to soak in ; whether there are any footings of the walls above ground, or plinths, causing dampness ; the condition of the windows, and whether they have proper sills, or whether there are none and their frames are set back from the face of the walls—a not uncommon cause of damp walls in old buildings ; the construction and state of the roof ; whether weather-proof, if much lead on it, whether any rain water that is stored off it is contaminated with that metal ; if any valley gutters or flats, whether liable to let rain through ; if any party walls are carried through the roof, whether they are properly coped and flashed so as to prevent rain soaking into them ; whether the eaves are properly spouted, and the rain water carried away so as to prevent dampness of walls and site, or whether the spouting overflows ; whether the stack-pipes discharge in the open air, or whether they serve as drain-ventilators, and if they do, whether they terminate under the eaves near to any window or other opening into the house ; if the rain water is collected, whether it is free from decaying leaves and bird-droppings from the roof, or any dead animals ; whether the receptacle is frequently cleansed ; if this receptacle is constructed of lead or of galvanized iron, whether the water takes any lead or zinc into solution ; whether any cistern or underground tank has an overflow, and if so, whether there is a proper air-disconnection on it in the event of its discharging into any house drain ; if there are stables, whether any drain from them has an air-disconnection before joining the house drain ; whether there are any animals or accumulations of manure or other refuse kept so as to be dangerous to health, and whether there are any other possible sources of nuisances upon the premises or upon any others in the vicinity.

Having made a general observation of all waste-pipes coming from the house, he will enter it, and examine them in detail : he will look to any cellars or basement rooms there may be to ascertain whether there is any dampness in them, and if there are any drain-openings in them, whether they are directly connected with the house drain or whether they are properly disconnected from it ; he will observe whether the site of the house has a layer of concrete beneath the floors and whether there is provision for circulation of air beneath any wooden floor ; whether any drain passes beneath it, and if so, whether there are any improper openings into it, or any leakage from it ; how its joints are made, whether it is bedded in concrete, and whether it has a proper air-disconnection and ventilation. On the ground floor he will observe whether any brick or stone floors are damp, and whether there is sufficient ventilation beneath any boarded floors, or any indication of dry-rot in them. He will observe whether there are any improper drain-openings on the kitchen or scullery floor, and will examine the waste-pipes from any sinks in those places, or in any pantry, to ascertain whether they have any inside trap, whether they are properly disconnected from the drain and discharge in the open air over channels leading to outside trapped gullies ; he will ascertain whether the latter are regularly cleaned out at frequent intervals. The waste-pipes from any lavatory basin, bath, urinal, or lead safe will claim his attention that he may see they do not join any soil-pipe or closet trap, and that they are properly disconnected from the drains ; he will also see that any slop sink is properly disconnected ; he will ascertain whether there is any system of excrement removal, other than a water-closet, within the house ; he will examine any water-closet there may be, observing its situation, whether in the middle of the house or whether against an outer wall, as

should invariably be the case, whether there is sufficient permanent ventilation to the outer air or any cross ventilation, whether the walls beneath the seat are properly plastered, and whether there are any cracks or crevices in the floor or ceiling which might allow foul odours to escape into other parts of the house; he will see whether the apparatus has any foul 'container' or 'D' trap, or whether it is of an approved form of construction, whether there is any weak joint between its trap and the soil-pipe, whether the waste-pipe from any lead safe beneath it or from the flushing cistern discharges into the trap or soil-pipe or into the open air, whether there is a sufficient flush of water, and whether the cistern is distinct from any which is used for the supply of drinking-water; he must see whether the soil-pipe is placed inside the house or outside, as it ought to be, what it is made of, whether there are any defects in it or in its joints, such as slip or putty joints, whether it is ventilated, and if so, whether the ventilating pipe is carried up the full size or not, with as few bends as possible, and whether it terminates in a fitting situation; whether the soil-pipe has an air-disconnecting trap. If more than one water-closet discharges into the same soil-pipe he will observe whether there is any possibility of either being untrapped by the flush of water from any other. If a water-closet discharges into the air over the head of an open soil-pipe he will take note whether there is any chance of foul odours from it entering the house through any windows or ventilating openings.

If he has reason to doubt the soundness of the system of drainage, he will best ascertain its condition by trying it with oil of peppermint and boiling water, or with some form or other of the smoke or other tests.

If he detects any bad odours in the house which he has not succeeded in accounting for, he will not neglect to ascertain whether any dead mice or other animals are concealed beneath floors or behind skirting boards, &c., and he will bear in mind that an odour may be conveyed by currents of air or through defects in walls, or through hollow walls or bell-wire tubes, or behind skirting boards, or beneath wooden floors, to any part of the house removed from the spot where it actually originates.

He will remember that a very common cause of stink in a house is the constant slight oozing of coal gas from leaks in pipes or fittings, from the joints in brackets, from the taps, or from ball-and-socket joints, or telescope joints of pendants, or through the loss of water by evaporation from the water-sealed joint of moveable gas pendants. He will not be satisfied if a leak is not detected on the application of a light, but will examine all possible sources of leakage by smelling close to them, since many a leak may be detected by the nose which is not revealed by the match. If the leakage is at all considerable he will avoid applying any light until the place where it occurs has been thoroughly aired, for fear of causing an explosion.

He will not neglect the possibility of any bad odour arising from a foul stove-pipe or chimney, either through defective brickwork or owing to their acting as air inlets into the house when insufficiently ventilated, as often happens when all outer doors and windows are closed, or from a chimney having been on fire; he will bear in mind the possibility of foul air from a drain ventilator ending near a chimney top being drawn into the house in that manner, or from a stack pipe through any window or ventilating opening.

He will note the number of rooms, the cubic capacity of all bed-rooms, the means provided for ventilating them, the situation and size of the windows, what parts will open, whether there are fireplaces in them, and if so, whether they or the chimneys are blocked up, whether there are 'registers' in the grates, and if so, whether they are kept closed as is usually the case; he will ascertain the number of people occupying each bed-room, the amount of

space taken up by the furniture in them, whether they are overcrowded, and whether the foul odour of overcrowding pervades any of them.

He will observe the state of cleanliness of the rooms and furniture, the condition of the walls, whether they show signs of damp or mouldiness from rising moisture or driving rain, or are out of repair, whether there is any chance of the air being contaminated by arsenical wall papers, bed hangings, or other fabrics.

He will see whether there are indications of dilapidations, and if so, whether the place appears to be in such a ruinous condition as to be dangerous.

Having examined the premises, he will probably next turn his attention to the water-supply; he will note its source, and if from a well or spring whether the surroundings are such as to be liable to pollute it, the approximate depth and construction of any well, whether it is liable to admit any surface soakage, and the geological formation out of which its water is derived; if the supply is taken from any water-course, whether such is liable to sewage pollution; if obtained from a waterworks mains he will ascertain whether the service is constant or intermittent, what metal the service pipe is made of, whether it is of sufficient size to give an ample supply, whether it is laid through or near to drains, cesspools, or any other possible source of contamination, or in any material capable of corroding it, whether there are any leaks in it, whether it is properly protected from the action of frost, and whether there is any direct service of water to closet pans. If the water is stored on the premises, he will ascertain the situation of the cistern and what it is made of; if it is of lead, whether the water has any action upon that metal, if of galvanised iron, whether it takes zinc into solution; if there is an overflow to the cistern, he will see whether it joins any water-closet trap, soil-pipe, or drain, or whether it is properly disconnected in the open air; if there is a standing waste-pipe in it, he will find out whether that is formed by a continuation of the soil-pipe acting as a ventilator; he will also see whether any water-closet is flushed from the cistern, or whether there is a separate flushing cistern for each closet.

This outline for the inspection of single premises may, on an extended scale, be applied to the examination of whole villages, towns, or other places; but then further influences bearing upon public health, such as the state of burial-grounds, elementary school accommodation, manufactories, unhealthy occupations, slaughter-houses, the arrangements for scavenging, the public water-supply, the food-supply, the pollution of water-courses, the general social condition of the people, the density of population, the sufficiency of house accommodation, &c., may have to be inquired into and taken into consideration.

Offensive trades, analogous to those specified in sections 112 and 114 of the Public Health Act, 1875, and in section 19 of the Public Health (London) Act, 1891, will come under the observation of the medical officer of health in metropolitan and urban sanitary districts, but not in rural sanitary districts, unless the authorities of those districts have been invested with urban powers for the purpose.

He must be acquainted with the processes carried on in connection with them, the causes of nuisances arising from them, and the appropriate means for preventing the same. He will be much assisted in reference to these matters by consulting Dr. Ballard's Report on Effluvia Nuisances, in the supplement to the Sixth Annual Report of the Local Government Board, subsequently issued in a separate form, and Dr. Hime's article on the subject in the first volume of this work.

He must be prepared to advise the local authority on the framing and

working of any bye-laws with respect to those trades which they may make. Under the model series (No. XVI.) issued by the Local Government Board he will have a right of entry for the purpose of inspection, at all reasonable times, into any places to which those bye-laws apply.

When he finds a nuisance arising from an offensive trade, he will, in the first instance, endeavour to get it conducted so as not to cause a nuisance; but if he does not succeed in this attempt he will give to the local authority a certificate stating the fact that it is a nuisance, or that it is injurious to the health of any of the inhabitants of the district, and then they will have to take the proceedings prescribed for procuring its abatement.

The medical officer of an urban sanitary district may have to certify that an offensive trade situated without his own district, even if it is within a metropolitan district, is causing a nuisance within his district, or that it is injurious to the health of any of the inhabitants of it, and then the local authority may take proceedings for abating the nuisance thus arising.

Similarly, a medical officer of a metropolitan district may have to certify respecting an offensive trade situated without it.

In both urban and rural sanitary districts the medical officer of health has a right of entry, under the 102nd section of the Act of 1875, in the case of a nuisance arising in respect of any business at any hour when such business is in progress or is usually carried on; and there is a similar power of entry in the metropolis.

It is worthy of remark that in the London Act of 1891 the words 'or dangerous to health' are applied to offensive trades, as in the case of nuisances.

In the County of London the medical officer of the County Council, and in the City of London the medical officer of the Commissioners of Sewers, will be concerned with the establishing anew of certain offensive trades, and with bye-laws for regulating offensive businesses and the structure of the premises on which they are carried on.

The Alkali, &c., Works Regulation Acts, 1881 and 1892, are executed by a chief inspector and other inspectors appointed by the Local Government Board; but if an urban, rural, or metropolitan medical officer of health finds that any work to which the Acts apply, whether situated within or without his district, is carried on in contravention of them, or that any alkali waste is deposited, either within or without his district, in contravention of the Act of 1881, and that a nuisance is thereby occasioned to any of the inhabitants of his district, he may make a written representation of the fact to the local authority of his district, and they may make a complaint to the Local Government Board, who will then have to make such inquiry into the matters complained of, and after the inquiry may direct such proceedings to be taken by an inspector under the Acts as they think just.

Unsound Meat, &c.—The medical officer of health of any urban, rural, or metropolitan district may, at all reasonable times, inspect and examine any animal, carcase, meat, poultry, game, flesh, fish, fruit, vegetables, corn, bread, flour, or milk exposed for sale, or deposited in any place for the purpose of sale, or of preparation for sale, and intended for the food of man; and if any such article appears to him to be diseased, or unsound, or unwholesome, or unfit for the food of man, he may seize and carry it away, or cause it to be seized and carried away, in order to have it dealt with by a justice, as directed by the Public Health Acts.

Animals, poultry, game, and fish need not necessarily be dead in order to bring them within the scope of these Acts. It must be noted that his powers in respect of these things cease as soon as they have been sold to any

purchaser, for he cannot then deal with them even if they prove unfit for human food.

The powers of the medical officer are strictly limited to the articles named above, and, except in the case of horseflesh, as will presently appear, he cannot enter any premises to examine or seize any other articles of food such as cheese or eggs, however rotten they may be; but in the metropolis, and where Part 3 of the Public Health Acts Amendment Act, 1890, is adopted, these powers are extended to all articles intended for the food of man.

He may have to advise the inspector of nuisances in any case of doubt arising under the inspection of those articles which that officer has to report to him, as required by the Orders of the Local Government Board of 1891, prescribing the duties of those officers in urban, rural, and metropolitan sanitary districts.

He has also, under section 181 of the Towns Improvement Clauses Act, 1847, incorporated with the Public Health Act, 1875, power to enter any butcher's shop, &c., or slaughter-place, in urban districts, for the purpose of examining any cattle or carcase and seizing any which he may find to be unfit for the food of man.

On complaint made on oath by him any justice may grant him a search warrant.

Limitation to specified articles of food is not found in the 15th section of the Markets and Fairs Clauses Act, 1847, incorporated with the 167th section of the Public Health Act, 1875, for the purpose of enabling any urban sanitary authority to establish or to regulate markets, which imposes the liability to a penalty upon every person who shall sell or expose for sale any unwholesome meat or provisions in any market which is subject to the provisions of that Act, and the inspector of provisions appointed by the 'undertakers' may seize such meat or provisions and carry them before a justice to be dealt with by him in accordance with the Act. Here there is an offence even if the unwholesome article of food has been already sold, and the term 'provisions' is very comprehensive. It is not at all likely that a medical officer of health will be appointed to be such an inspector, neither is it desirable that he should be, but he should be aware of this provision, that he may instigate the appointed person to take action under it if the occasion for so doing should arise at any time.

The Sale of Horseflesh, &c., Regulation Act, 1889, gives the medical officer of health at all reasonable times power to inspect and examine any meat which he has reason to believe to be horseflesh, exposed for sale, or deposited for the purpose of sale or of preparation for sale, and intended for human food, in any place other than a shop, stall, or place over or upon which there are at all times painted, posted, or placed, in legible characters of not less than four inches in length, and in a conspicuous position, and so as to be visible throughout the whole time, whether by night or day, during which such horseflesh is being offered or exposed for sale, words indicating that horseflesh is sold there. If such meat appears to him to be horseflesh he may seize and carry it away, or cause it to be seized and carried away, in order to have it dealt with by a justice in the manner provided by the Act. It will be observed that the method of procedure is precisely similar to that which has to be pursued in the case of diseased or unwholesome meat, &c., and there is the same power for the medical officer to obtain a search warrant from a justice on complaint made by him on oath.

It is not quite clear from the wording of the 3rd section whether or no a medical officer has power to proceed without being specially authorised by the local authority to do so. In a case heard in the Reading Police Court

the counsel for the defence raised the objection that the medical officer and the inspector of nuisances could not act without such instruction or authorisation, but the magistrates overruled the objection; however, as the point seems somewhat doubtful, it will probably be best for those officers to be formally authorised by the local authorities to carry out the Act. It is necessary for them to understand that 'horseflesh' includes the flesh of asses and mules, and means horseflesh, cooked or uncooked, alone or accompanied by or mixed with any other substance.

Slaughter-houses may be provided by urban sanitary authorities; and bye-laws may be made with respect to the management, &c., of any so provided and of private slaughter-houses. In rural sanitary districts these powers cannot be exercised unless urban powers for the purpose have been granted to the local authority by the Local Government Board.

The medical officer of health will not be the systematic inspector of those places, but he should assure himself that they are kept in a proper state, and under the model bye-laws respecting them issued by the Local Government Board (Series No. VI.) power of entry into them at all reasonable times is given to him.

When the local authority have under their consideration the propriety of granting or withholding the licence needed before a place can be newly established as a slaughter-house, the medical officer of health will probably be consulted as to the suitability or otherwise of the proposed site from the health point of view; and he will be aided in forming an opinion upon the question by the memorandum prefixed to the model bye-laws.

The Sale of Food and Drugs Act, 1875, and the Act of 1879 amending it, so far as they concern the medical officer of health, may be noticed in this place, since they also deal with articles of human food.

Any medical officer of health, under the direction and at the cost of the local authority appointing him, or charged with the execution of the Act, may procure any sample of food, that is to say, any article used for food or drink by man other than drugs or water, which is entirely exempted from the operation of the Act, or any sample of drugs, which include medicine for internal or external use, and if he suspect such sample to have been sold to him contrary to any provision of the Acts, he has to submit it to be analysed by the analyst of the part for which he acts, or if there be no such analyst to the analyst of another place; the analyst will have to give to him a certificate specifying the result of his analysis. If the analyst does not reside within two miles of the residence of the medical officer requiring the sample to be analysed, he may forward it to him through the Post Office as a *registered parcel*, under the 11th section of the Post Office Act, 1891, instead of as a *registered letter*, which the Act of 1875 required, but which the Postmaster-General will not now allow to be done.

If it appears from the analyst's certificate that any offence under the Acts has been committed he may take the prescribed proceedings against the vendor. It does not appear requisite that he should obtain special directions from the local authority before taking such proceedings, although he would naturally be guided in the matter by what he believes to be their desire.

When obtaining samples under the Act of 1875 he must strictly comply not only with the spirit but with the letter of the 14th section. After the purchase has been completed he must forthwith notify to the seller or his agent selling the article his intention to have the same analysed by the public analyst for the district. He must next offer to divide the article into three parts, and if he is required to do so, but not otherwise, he must so divide it,

and must mark and seal or fasten up in such manner as its nature will permit each part; he must then deliver one of the parts to the seller or his agent, one he must retain for future comparison and for production in court at the hearing of the case if proceedings are taken; the third part is for delivery to the analyst. If the seller or his agent does not accept the offer of the medical officer to divide the article purchased in his presence, then it must on no account be divided, but must be delivered whole to the analyst, who must himself divide it into two parts, one of which he must seal up and cause it to be delivered, either upon receipt of the sample, or when he supplies his certificate, to the medical officer, who must retain it for production in case proceedings are afterwards taken in the matter.

The medical officer may employ a deputy to make the purchase, but as soon as it has been completed that person must give the article purchased to him and he must complete the transaction as described above, and will take proceedings if the occasion should arise, in the same way as if he had actually made the purchase himself.

Under the 3rd section of the amending Act of 1879, samples of milk which is in course of delivery to the purchaser or consignee may be taken at the place of delivery by any medical officer of health, who is authorised in the same manner as under the Act of 1875, but in that case it is not necessary for him to observe the provisions of the 14th section of that Act.

It is most essential that all samples should be securely sealed in such a way that they cannot be afterwards tampered with. They are not infrequently sealed in such a slovenly manner that the contents of the package can be taken out and be replaced by another article without the seal being broken. If such were the case with any of the parts left with the vendors the reputation of the analyst might be unfairly damaged.

Corks should be cut off level with the top of the necks of bottles containing samples, and they should be firmly tied in with string, the knots being placed on the top of the corks: all packets containing samples should first be securely tied up with string; all knots should be completely sealed, and other parts of the string should also be sealed so as to prevent it being slipped off the packets without the seals being broken. The wax which is used should not be brittle, but should be of the best quality. A seal with a distinctive impression that cannot be readily imitated must be employed.

All samples must be carefully labelled; on the labels there should be printed the name of the district, and there should be places on them for a distinctive number, description of the article purchased, date and signature, all to be filled in by the purchaser at the time of purchase.

The name of the vendor of any sample should not appear upon the label, neither ought it to be divulged to the analyst. In the event of any sample being divided, the labels on all three parts must be alike.

An exact record of all the circumstances of the purchase of any sample should be made at the time of purchase in a book kept for the purpose.

The Margarine Act, 1887, imposes no special duty upon the medical officer of health, but when authorised to take samples under the Sale of Food and Drugs Act, 1875, he may procure samples of margarine for analysis if he has reason to believe that the provisions of the Act, requiring that when forwarded by any public conveyance it shall be duly consigned as margarine, are infringed, and examine and take samples from any package, and ascertain if necessary, by submitting them to be analysed, whether an offence against the Act has been committed.

He may also, without going through the form of purchase provided by the Act of 1875, but otherwise acting in all respects in accordance with its pro-

visions as to dealing with samples, take for the purpose of analysis samples of any butter, or substances purporting to be butter, which are exposed for sale, and are not marked 'margarine,' as provided by the Act: it is to be presumed that any such substance which is not so marked is exposed for sale as butter.

A medical officer of health cannot take any action under either of these three Acts unless he is duly authorised to do so. It is not desirable that he should be charged with the duty of procuring samples under them, and it is not likely that he will be, but it is quite necessary for him to know all the details of procedure, if only to be able to direct the inspector of nuisances aright if need be, inasmuch as a prosecution under those Acts will be liable to fail if the strict letter of the law is not followed when any samples are taken.

Infectious diseases are dealt with under several Acts which very closely concern the medical officer of health in the execution of his duties. The Regulations of the Local Government Board respecting those duties are set forth in their Orders of 1891, already quoted. Some of the principal provisions are to be found in the 120th and following sections of the Public Health Act, 1875, and, as regards the metropolis, in sections 55 to 74 of the Public Health (London) Act, 1891.

When the medical officer certifies to the local authority that the cleansing and disinfection of any house or part of a house, or of any articles therein likely to retain infection, would tend to prevent or check infectious disease, they have to take the prescribed steps to get such cleansing and disinfection done; but this will generally be effected without his having recourse to that formal procedure. When occasion arises he may certify in a like manner in respect of infected ships, under the 2nd section of the Public Health (Ships, &c.) Act, 1885, but he cannot take any such action in the case of any ship or vessel under the command or charge of any officer bearing Her Majesty's commission, or of any ship or vessel belonging to any foreign Government. Under the 4th section of the Canal Boats Act, 1877, he may have to certify to the local authority for the disinfection of infected canal boats, or for other steps requisite for them to take for the prevention of the spread of infectious disease therefrom. Whenever they have detained a canal boat for the purpose of being cleansed and disinfected, he may have to provide them with a certificate under the 18th clause of the Regulations of the Local Government Board under the Act, dated March 20, 1878, to the effect that the boat has been duly cleansed and disinfected, before they can allow it to proceed on its journey. If duly authorised by a registration or sanitary authority, or by a justice of the peace, he may, on producing (if demanded) either a copy of his authorisation signed by the clerk or a member of the local authority, or some other sufficient evidence of his being so authorised, enter by day, that is between 6 A.M. and 9 P.M., any canal boat on board of which he has reasonable cause to suppose either that there is any contravention of the Act or that there is any person suffering from an infectious disorder, and examine every part of it in order to ascertain whether such is the case; and he may, if need be, detain the boat for the purpose, but for no longer time than is necessary. He may require the master of the boat to produce to him the certificate of registry (if any) of the boat, and to permit him to examine and copy it; he may require him to furnish him with such assistance and means as he may need for the purpose of his entry and examination of and departure from the boat.

When authorised under the 9th section of the Housing of the Working Classes Act, 1885, he has the same power of entry to any tent, van, shed, or similar structure used for human habitation, when not erected or used by any

portion of Her Majesty's military or naval forces, for the purpose of ascertaining whether there is therein any contravention of any bye-law made by the local authority for preventing the spread of infectious disease by the persons inhabiting the same, or any person suffering from a dangerous infectious disorder.

This power extends to the metropolis; it is a very useful one, and should be generally exercised in all places where inhabited vans, &c., are congregated at fairs or on other occasions, inasmuch as there can be no doubt that they have often been the means by which infectious diseases have been introduced into places through the concealment of cases within them. Systematic inspection of such structures is a great safeguard against disease; the actual performance of it will devolve upon the inspector of nuisances, but it should be done under the supervision and with the assistance, if necessary, of the medical officer of health.

When he has received from the keeper of a common lodging-house in any urban or rural sanitary district notice under the 84th section of the Public Health Act, 1875, that there is in such house a person ill of any infectious disease, it will be his duty to satisfy himself whether or not it is necessary to remove such person to any hospital for infectious diseases there may be, and to see that any bye-laws made in that behalf are complied with. The bye-law relating to this matter in the model series (No. III.) issued by the Local Government Board requires, amongst other things, that the keeper of a common lodging-house shall obey any instructions of the medical officer of health respecting the removal by order of a sanitary authority of any lodger suffering from infectious disease to an isolation hospital, the discontinuance of receiving lodgers into infected rooms, and the disinfection of infected rooms and things; and that he shall give that officer written notice of having carried out the disinfection, and of the death, removal, or recovery of any lodger who has been ill with infectious disease.

If the local authority have made any bye-laws under the 90th section of the Public Health Act, 1875, with respect to houses let in lodgings, other than common lodging-houses, he may be concerned with any which provide for the giving of notices and the taking of precautions in case of any infectious disease. In the model series (No. XIII.) relating to this subject there are bye-laws requiring the landlords and lodgers to give immediate notice in writing to the medical officer of health when they become aware of the occurrence of any case of infectious disease in such houses; another makes it incumbent upon them to comply with any instructions they may receive from him respecting the removal of any sick person to a hospital when an order of a justice has been obtained for the purpose.

If in the district of the medical officer there are no means available for the disinfection of bedding, clothing, or other articles which have become infected, and which will not bear boiling in water, he will have to urge the local authority to provide a proper apparatus for that purpose, if he considers the needs of his district require it, inasmuch as the effectual disinfection of all infected articles is a highly important step in the eradication of infectious disease.

In this connection he will find much valuable information in the Report on Disinfection by Heat by Dr. Parsons, appended to the Report of the Medical Officer of the Local Government Board for the year 1884.

Moist heat is undoubtedly the most fitting agent to employ for the purpose; but as the result of some experiments with several forms of apparatus which Dr. Whitelegge and the writer have made, we formed the opinion that less importance should be attached to the use of steam at high pressures than is insisted upon by Dr. Parsons, and we arrived at the conclusion that

even bulky articles may be effectually disinfected by steam at very low pressure, and at most with an occasional assistance of a pressure not exceeding some five pounds to the square inch.

Inasmuch as the destruction of infection is aimed at as much in the interests of the public as of private individuals, and in many instances more so, it is fitting that it should be carried out by local authorities at the public expense, and the medical officer should endeavour to get this accomplished in his district.

In the event of the authority possessing no disinfecting apparatus, he will sometimes have to advise them to exercise their powers of having infected articles destroyed and paying compensation for them.

When he finds any person suffering from a dangerous infectious disorder who is without proper lodging or accommodation, or is lodged in a room occupied by more than one family, or is on board any ship or vessel, or is lodged in any common lodging-house, he may have to give a certificate to enable any justice to order the removal of such person to any suitable hospital or place for the reception of the sick which is within the district or within a convenient distance of it, with the consent of the superintending body of such hospital or place.

He might under some circumstances give a certificate that any house or part of a house in which a person has been suffering from any dangerous infectious disorder, and any articles therein, have been disinfected to his satisfaction, so as to allow of its being legally let for hire.

Regulations for the prevention of cholera made at various times by the Local Government Board impose several duties upon medical officers of health in the districts they relate to: for example, the General Order of that Board prohibiting the importation into England or Wales of any rags from Marseilles or Toulon, in 1884, required 'the express authority in writing of the medical officer of health' before any rags from the above-named places which had been landed and deposited in any place could be removed or be permitted to be removed therefrom, and it allowed him to give this authority either conditionally or unconditionally; if given subject to conditions as to the disinfection or otherwise of the rags, or as to their destination, the person having control over them had to have them removed and had to comply with his conditions; it further made it the duty of the medical officer of health to satisfy himself that the conditions were duly complied with.

Other similar Orders have empowered the sanitary authority to cause rags which had been landed in contravention of their provisions, unless immediately re-shipped or exported, to be destroyed, with such precautions as he recommended.

Some regulations made in 1892 dealt also with bedding and disused or filthy clothing.

Medical officers of port sanitary districts should see that apparatus capable of disinfecting bales of rags is provided and constantly kept in good working order, and should be prepared with some safe place for the deposit of suspected articles until they can be disinfected or destroyed.

The Cholera Regulations of that Board now in force are dated August 28, 1890, and September 6, 1892. Under them the term 'medical officer of health' includes any duly qualified medical practitioner, appointed by a sanitary authority, to act in the execution of the orders.

Whenever a sanitary authority receives notice from an officer of Customs that he has detained any ship known or suspected to be infected with cholera, their medical officer of health must forthwith visit and examine her for the

purpose of ascertaining whether she is infected with cholera. If he have reason to believe that any ship coming or being within the jurisdiction or district of the sanitary authority, whether examined by the officer of Customs or not, is infected with cholera, he must, or if she have come from a place infected with cholera, he may visit and examine her, for the purpose of ascertaining whether she is so infected, and the master of the ship is obliged to permit him to do so.

In either case, if the medical officer is of opinion that the ship is infected, he must forthwith give a certificate to that effect in duplicate, in a form prescribed by the Order, and must deliver one copy to the master, and retain the other or transmit it to the sanitary authority.

He must then, as soon as possible, examine every person on board, and if he finds anyone suffering from cholera, or any suspicious illness, he must certify accordingly; and any person who is not so certified by the medical officer will not be permitted to land unless he is satisfied as to his name, place of destination, and address at such place. He must then forthwith give the name and address of any such person to the clerk to the sanitary authority, who has to transmit it to the local authority of the place of destination.

Every person certified by the medical officer of health to be suffering from cholera must be removed, if his condition admits of it, to some hospital or other place of isolation appointed by the sanitary authority, and may not be removed therefrom until the medical officer has certified that he is free from the disease. If such person cannot be removed, the ship must remain subject, for the purposes of the Order, to the control of the medical officer of health, who must give his consent in writing before the infected person may be removed from or leave the ship.

Any person certified by the medical officer to be suffering from any illness which he suspects may prove to be cholera, may be detained for a period not exceeding two days, either on board the ship or in some place of isolation, in order that the nature of the illness may be ascertained. If the illness proves to be cholera, the medical officer will certify to that effect and the patient will be dealt with as described above.

Whenever a ship is certified to be infected, it devolves upon the medical officer of health to give directions and take such steps as may appear to him to be necessary for preventing the spread of infection, and the master of the ship must forthwith carry out his directions.

When any death from cholera takes place on board a ship while detained under the powers of the Order, the sanitary authority or the medical officer of health may direct the master either to take the dead body out to sea and commit it to the deep or to deliver it into the charge of the authority for interment.

The sanitary authority or the medical officer of health may direct the master to disinfect or destroy any infected articles, and he must cause the ship to be disinfected according to the directions of the medical officer.

The Cholera Regulations of 1892 further provide that when a ship is not infected with cholera, but has passengers on board who are in a filthy or otherwise unwholesome condition, or has come from a place infected with cholera, the medical officer of health may, if in his opinion it is desirable, with a view to checking the introduction or spread of cholera, give a certificate in duplicate to that effect in a prescribed form; then no person may leave the ship without satisfying him as to his name, place of destination, and address at such place, and he has to deal with that information as previously described.

When he has reason to believe that any ship coming or being within the jurisdiction of the sanitary authority is infected with cholera, or has come from a place infected with cholera, he may direct the bilge-water to be pumped out before she enters any dock or basin; and on the sanitary authority providing a proper supply of water for drinking and cooking purposes for persons on board, he may direct all casks or tanks on board containing water for their use to be emptied and cleansed, and the master must cause his directions to be carried into effect.

Where the Local Government Board, when any part of England appears to be threatened with or is affected by any formidable epidemic, endemic, or infectious disease, make regulations for the speedy interment of the dead, for house-to-house visitation, and for the provision of medical aid and accommodation, for the promotion of cleansing, ventilation, and disinfection, and for guarding against the spread of disease, it is the duty of the medical officer of health to observe them, so far as they relate to or concern his office. He has power of entry on any premises or vessel for the purpose of executing or superintending the execution of any regulations so made. The time during which that power may be exercised is not restricted to any specified hours.

Hospital accommodation for the isolation of persons suffering from infectious disease who cannot be effectually separated from the healthy in their own homes will demand the attention of the medical officer of health, and he will have to advise the local authority to provide sufficient for the needs of his district if such does not already exist.

The accommodation, to be effectual, must be in readiness before the advent of an epidemic. Too often the provision of it is left until such time as a disease has obtained an extensive hold upon a place, and then money is lavishly expended in erecting some temporary accommodation, under the influence of panic; but any accommodation so provided will seldom be well fitted for the future requirements of the district.

Infectious disease should not be left to be dealt with by such temporising measures; they cause needless expense and entail much extra labour and anxiety upon those concerned with their administration. They ought not to take the place of more permanent provision, but may often form a very useful adjunct.

The amount and character of the accommodation required will depend upon the locality, the character of the population, and other local circumstances, so that each case must be decided upon its own merits.

All isolation accommodation must be entirely free from any suspicion of being connected with pauperism, and should not be administered by the destitution authorities. It is not desirable that a hospital for infectious diseases should be situated within the precincts of a workhouse; but if it is it should be separated from the premises belonging to that institution by substantial walling, and, if in a rural district, it ought to belong to and be under the control of the guardians acting as the rural sanitary authority, and not as the poor law authority. It is true that in the metropolis the isolation hospitals are under the control of a poor-law authority, the Managers of the Metropolitan Asylums District, who have done much good work, but the Poor-Law Act, 1889, gave them power to admit into their hospitals any person who is not a pauper and is suffering from fever, small-pox, or diphtheria, the right to admit cases of the last-named disease being a new power.

Isolation hospitals should be made as comfortable and attractive as circumstances will permit, and with that object in view they should be designated by some euphonious title such as may not exert a deterrent influence upon those it is wished to induce to use them.

Inasmuch as the isolation of infectious disease is carried out in the interest of the public at large rather than of private individuals, it is desirable, and essential for the successful working of the system, that patients should not be charged with the cost of their maintenance in any isolation hospital, but that it should be defrayed at the public expense, unless separate accommodation is required.

In rural districts a cottage suitably situated and set apart for the purpose may suffice for one or more villages. If any movable structure is depended upon for several villages, arrangements for sites upon which to place it at any time should be made so that there may be no delay, with consequent spread of infection, when the occasion for using it arises. If tents are used—and they have proved of good service on more than one occasion—they should be of the best construction, their canvas should be water and fire proof, they must have double walls and roofs, be well ventilated, and have raised wooden floors which should be trenched all round; moreover, if they are to be used in cold weather there must be some provision for warming them, but paraffin stoves are unsatisfactory and dangerous. They are best warmed by hot-water pipes carried round them between the beds and tent walls, the boiler, of course, being placed in a separate outside structure; gas, if available, would be the best, though perhaps not the most economical fuel for heating it. Oil lamps are unsafe in tents—candles placed in secure lanterns are preferable, but if gas or electricity can be had, they are the safest means of lighting them. Gas jets should be protected by wire cages. Buckets filled with water should be always kept in readiness for use in case of fire, and if there are water-mains near, hydrants and fire-hose should be provided.

In boisterous weather tents require close attention and may give rise to much trouble and anxiety; the ropes must be constantly attended to, requiring, as they do, to be slackened or tightened as they become wet or dry.

If no provision for isolation whatever has been made, an empty cottage, a village school, or even a barn may answer the purpose on an emergency.

In urban districts of any size some permanent accommodation for isolation should always be in readiness, and if it is not in constant occupation the medical officer should assure himself that it is always kept dry and well aired, so as to be fit for use at any time. It should be so arranged as to allow of ready and convenient temporary extension in case of need. It should provide for the reception of cases of not less than two different diseases at one and the same time in separate buildings, or in a building divided into two or more parts with no internal communication whatever between them, and, of course, for the separation of males from females. It will be found economical to have at any rate an administrative block of buildings sufficiently large for the fullest probable present and prospective requirements of the district.

Much valuable information on this subject will be found in the Report on the Use and Influence of Hospitals for Infectious Diseases, by Dr. Thorne Thorne, appended to a Report of the Medical Officer of the Local Government Board in a supplement to the Tenth Annual Report of that Board, and also in a memorandum 'On the Provision of Isolation Hospital Accommodation by Local Authorities,' issued from the Medical Department of the Board, in which the proper standards of ward space are given as not less than 2,000 cubic feet of air space, with 144 square feet of floor space, and 12 linear feet of wall space to each bed. Dr. Thorne recommends that the relation of window surface to the cubic capacity in wards should not vary much beyond the limits of 1 square foot to from 60 to 80 cubic feet, a proportion of about 1 square foot to every 70 cubic feet being,

he considers, as a rule, the most advantageous. He states that in the Children's Hospital at Pendlebury, due regard being had to ventilation, the ward air cannot be maintained equably warm, and at the same time sweet, owing to a great extent to the excessive window surface, which there amounts to 1 square foot for every 85 cubic feet of space.

The memorandum indicates that proper means are to be provided for the adequate ventilation and warming of wards, and for securing them from closet emanations and the like; that earth-closets may be used as the means of excrement disposal, but that water-closets are to be regarded as preferable when efficient sewers are available; that there should be places for washing and disinfection, and for a mortuary; that there should be an interval of 40 feet everywhere interposed between every building used for the reception of infected persons or things and the boundary of the hospital site; that this boundary should have a close fence of sufficient height; and that the 40 feet of interval should not afterwards be encroached upon by any temporary building or other extension of the hospital; that in determining the locality where an infectious hospital should be placed, the wholesomeness of the site, the character of the approaches, together with the facilities for water-supply and for slop and refuse removal, are matters of primary importance; that sites for hospitals designed to receive small-pox cases require a very much larger space about them than sites for other infectious diseases hospitals; small-pox hospitals being apt to disseminate small-pox, their sites should, consequently, be placed outside of towns, and should be sought at places as far distant from any populated neighbourhood as considerations of accessibility permit.

The medical officer of health will not improbably be consulted as to the choice of sites for isolation hospitals, with special reference to their influence on the surrounding neighbourhoods. On this question he will find much valuable information in the report of Dr. Thorne, quoted above; in a Report on the Distribution of 'Fever' in the immediate Neighbourhood of the London Fever Hospital, by Mr. Shirley F. Murphy, embodied in that report; in three reports on the Influence of Fulham Small-pox Hospital on the Neighbourhood surrounding it, by Mr. W. H. Power; in the supplements of the Tenth, Fourteenth, and Fifteenth Annual Reports of the Local Government Board, containing reports of their medical officer; in reports by Mr. Power on Statistics of Metropolitan Small-pox Incidence, and on Small-pox in West Ham during 1884-85, appended to the Report of the Medical Officer of the Local Government Board for the year 1886; and in the Report of the Commissioners appointed to inquire respecting Small-pox and Fever Hospitals in the Metropolis, published in 1882.

Dr. Thorne says, with respect to infectious fevers other than small-pox, and especially with respect to scarlet fever, typhus, and enteric fever: 'I have been unable to ascertain, as the result of every inquiry, that any spread of infection has resulted in the vicinity of any of the hospitals visited, which, apart from an attack resulting from a visit to patients under treatment, could in any way be attributed to hospital influence. On the contrary, many instances have been met with in which the existence of cases of these fevers near the hospitals in question could not fail to have become known, and yet in these very instances it so happens that there has been a marked immunity from these diseases in the localities referred to.'

He states that instances which he gives, and which may be taken as characteristic of the experience gained during the course of his inquiry, 'tend to show that in well-administered hospitals having an open space of some 40 feet between the hospital wards and any neighbouring thoroughfares or

‘dwellings, no risk of the spread of infection from scarlet fever, typhus, and enteric fever need be apprehended.’ But inasmuch as in none of the instances he records ‘was the available experience spread over any long term of years, neither did the patients under treatment from any of the diseases named for any length of time exceed some forty in number,’ he quotes the following facts gathered from the more extended investigation of Mr. Shirley Murphy in his Report on the Influence of the London Fever Hospital:—

‘1. A not inconsiderable population in Islington has for many years past resided in houses at a distance varying from 36 feet to 71 feet of the London Fever Hospital buildings, from 49 feet to 80 feet of the wards themselves, and from 22 feet to 33 feet of the hospital gardens used by the patients; the gardens belonging respectively to the houses and to the hospital, being only separated by a boundary wall.

‘2. For a term of several years there were under treatment at one and the same time in the hospital wards from 100 to 200 typhus patients, along with a score or two of scarlet fever and enteric fever patients; during several months of this period, and by the aid of special temporary provision, accommodation was found for the simultaneous isolation of from 200 to 300 relapsing fever patients; and during a later period, the hospital being used for little but scarlet fever and enteric fever, it has contained at one and the same time from 80 to 100 scarlet fever, and some 30 to 40 enteric fever patients.

‘3. As regards the relation between the two things as measured by such inquiry as it was practicable to make, the following appear:—Not a single case either of typhus or relapsing fever could be heard of as having occurred amongst the population in question. As regards scarlet fever and enteric fever, there had been certain cases in the adjoining houses during the years covered by the inquiry. Making, however, such estimate as he could respecting the number of such cases to be expected in this population, according to the prevalence of scarlet fever and enteric fever about London generally, Mr. Murphy judges that the observed number and the estimated number are practically identical.’

Dr. Thorne further observes: ‘With regard to some other infectious fevers, such as diphtheria, measles, &c., no sufficient information was obtained to warrant any decided conclusion as to the spread of these infections being arrived at as the result of this inquiry, but there can be little doubt that these diseases may be judged of in the light of the experience gained with respect to those which have already been considered.’

As regards small-pox he observes: ‘Owing to the very limited information on this point which was elicited during the inquiry, I find it difficult to draw any further conclusion than that conditions of hospital construction and administration which have sufficed to prevent the spread of scarlet fever, typhus, and enteric fever are not proved to be adequate to prevent the spread of small-pox. It may be worth noting in this connection that in all the instances in which the latter disease was alleged to have been spread beyond the hospital limits, the other infectious fevers had been under treatment, and no such spread had taken place.’

Next in point of time came Mr. Power’s first Report on the Influence of the Fulham Small-pox Hospital. His inquiry was made in 1881, and was entered upon in consequence of allegations, made to the Local Government Board, that large small-pox hospitals are dangerous to the neighbourhoods in which they are situated. He prefaces the report with the remark that ‘the circumstances of the Fulham Hospital probably have a broad resemblance to those of other London small-pox hospitals in respect of the subject of these allegations; and the lessons to be learned from the present inquiry



will probably not be without their application, *mutatis mutandis*, to small-pox hospitals in other parts of the country.'

He conducted a long and intricate investigation during the time an outbreak of small-pox was actually in progress, and compared the results he arrived at with the behaviour of small-pox in the neighbourhood of the hospital during former epidemic periods. He found sufficient evidence of similar experiences on those occasions, and summarised his conclusions in the following terms:—

'1. There has been in each epidemic period an excessive incidence of small-pox on houses in the neighbourhood of the hospital as compared with more distant houses in Chelsea, Fulham, and Kensington.

'2. The percentage of houses invaded in the neighbourhood of the hospital has become gradually smaller as the distance of the houses from the hospital has increased.

'This gradation has been very exact and very constant.

'3. Houses upon the chief lines of human intercourse with the hospital have not suffered more than houses lying in other directions from the hospital.

'4. In point of time there has been a very marked relation between the varying use of the hospital and the manifestations of excessive small-pox in the neighbourhood.

'This relation has not shown itself while the use of the hospital has been for convalescents only.

'5. The appearance of excessive small-pox in houses around the hospital has never been delayed until the hospital has become full or nearly full. It has been always most remarkable at the time when admissions to the hospital were beginning to increase rapidly.

'In the succeeding months of active operations, though the use of the hospital may have gone on increasing, the excess of small-pox upon the neighbourhood has habitually become less marked.

'6. On comparison of different epidemics, an almost constant ratio is observed between the amount of the hospital operations and the degree of excess of small-pox in the neighbourhood.'

The administrative circumstances of the hospital, and the sufficiency of these to account for the observed results, next claim Mr. Power's attention. Every detail having been inquired into with much exactitude, he proceeds to other conclusions in his report, thus:—

'It becomes necessary, therefore, to formulate, as a further, and, to me, unexpected result of the present inquiry, the following additional propositions:—

'7. The machinery of the hospital administration, with inclusion of defects in that machinery, does not account for the peculiarity of small-pox incidence within the three parishes of Chelsea, Fulham, and Kensington since the establishment of the hospital.

'8. There must have been some condition or conditions operating to produce the observed distribution of small-pox around the hospital that have pertained to the hospital as such, and that have been in excess of the condition of small-pox extension as usually recognised.'

Mr. Power next proceeds to discuss the meteorological circumstances possibly affecting the wide aerial dissemination of small-pox around the hospital which he feels constrained to believe in, and arrives at the following additional conclusions:—

'9. During the present epidemic period, and most probably during former similar periods, there has arisen in the atmospheric circumstances of the

time peculiar facility for the dissemination in an undamaged state of any matter that may have been given off from the hospital.'

Commenting on this report, Dr. (now Sir George) Buchanan says: 'Looking to the great and prolonged care and thought that Mr. Power has given to his inspection, and to the appearances of uniform law in the results which he reluctantly announces, I have no choice but to accept the above conclusions, and to believe that the Fulham Hospital, with all its advantages of site and construction, and with the many excellencies of its administration, has, by dissemination of small-pox material through the atmosphere, given rise to an exceptional prevalence of small-pox in its neighbourhood.'

The Report of the Commission to inquire respecting Small-pox and Fever Hospitals in the Metropolis of 1881 contains matter of much interest and importance. In it the Commissioners state: 'That by some means or other the Asylums Hospitals in their present shape cause an increase of small-pox in their neighbourhoods appears to us clearly established by the experience of these five hospitals during the last ten years. . . . We cannot but conclude that the increase of small-pox near these hospitals which has been so uniformly subsequent to their being brought into full working, has also been consequent on it.'

In discussing whether hospitals are centres of infection *per se* they remark: 'It is indisputed that actual contact with a small-pox patient, or with a body which has been in actual contact with one, is not necessary for the propagation of the disease, or, in other words, that the infective matter of small-pox spreads at least a few yards through the atmosphere.'

'We have to inquire how far at the utmost the infection can spread, and under what conditions it can spread beyond the generally admitted distance; and unfortunately in the present state of ascertained facts it is not possible to give a conclusive answer to either of these questions. Respecting the distance to which infection may spread, the evidence will, indeed, show that the opinions of those who have most studied the subject vary from a maximum range of ten yards to that of one or two miles. And when we have asked the practical question—What breadth of ground should be left open round a hospital in order that its neighbours may be in a state of reasonable safety from its effects? we have received answers ranging from sixteen or twenty-four feet to half a mile. So also with regard to the conditions under which infection may spread most widely, of which conditions the number of cases collected together is by many regarded as of most importance. If we endeavour to ascertain what number of small-pox patients may be treated in the same hospital without appreciable effect on the health of the neighbourhood, we find that Sir W. Gull considers that twenty acute cases are a large number to have in a hospital. Mr. Bostock and Dr. Gayton think that fifty or sixty would be safe, or tolerably so. If the cases are not acute but mixed, Dr. Bridges, Mr. Bostock, Dr. Munk, and Sir James Rison Bennett incline to a maximum of 100. Dr. Tripe, who has carefully watched the working of Homerton Hospital, declares that no mischief has occurred there while the number of (mixed) cases was under eighty, and suggests a limit of 100, or at most 150.'

'The larger of these numbers is also suggested by the Society of Medical Officers of Health, while Sir W. Jenner strenuously protests even against the smaller. Even those authorities who are disposed to reject the idea of distant atmospheric dissemination are not generally disposed to exceed a maximum of 150.'

Referring to Mr. Power's report, the Commissioners say: 'It is very difficult to estimate the value of Mr. Power's analysis, and it would, therefore, be

useless here to reproduce it. We will only say that it is very careful, but that it must be accepted subject to all that we have said respecting the extent to which small-pox is capable of being propagated through unexpected and undiscernible channels.

'On the whole, the following appears a fair *résumé* of the case for atmospheric dissemination founded on the evidence brought before us, in giving which we adopt freely Mr. Power's language :—

'We find in each epidemic period an excessive incidence of small-pox in the neighbourhood of the hospital as compared with that at a distance.

'Comparing epidemic with epidemic, we find that the aggregate incidence varies with the amount of hospital operations.

'Analysing the incidence, we find that the proportion of houses invaded by small-pox decreases as they are more distant from the hospital with a regularity strongly suggestive of a natural law.

'And examining the incidence from fortnight to fortnight we find that the number of cases of small-pox arising in the neighbourhood varies generally with the number of acute cases under treatment in the hospital.

'In a special and carefully studied outbreak of disease we find a large number—an unusually large number, it is said—of independent cases which cannot, after the most minute inquiry, be connected with the personal communications of the hospital, or with any other source of infection by contact, and particularly that the houses on the lines of human intercourse have not suffered more than other parts of the same neighbourhood. Lastly, we have in atmospheric dissemination a vehicle of contagion of admitted potency within a certain range, and capable, if we suppose that potency to have been hitherto underrated, of producing exactly all these results.

'On the other hand, against a belief in widely extending atmospheric dissemination, it may be urged :

'That the number of facts supporting it is at present too small.

'That the chief of these facts have been observed in the case of only one hospital.

'That the evidence in disproof of sufficient personal communication in the neighbourhood of this hospital is necessarily very negative and incomplete.

'And that the immunity of persons living near small-pox hospitals, if guarded from all, even indirect, personal communication with their inmates, is quite inconsistent with the belief in infection by particles carried far through the air.

'The following are instances of this immunity :—

'The City of London Workhouse, which overlooks the Homerton Small-pox Hospital, and is distant from it, window to window, only 90 feet, had scarcely any cases in the epidemics of 1871 and 1877, when the disease was extremely prevalent in the surrounding streets, although at that time the inmates were not protected by re-vaccination. The same may be said of the Hackney Union Workhouse and Infirmary, which are about a quarter of a mile from the Homerton Small-pox Hospital, and therefore well within the area supposed to be affected by aerial contamination.

'At Highgate, the Central Sick Asylum Infirmary, with 400 or 500 inmates, is within 200 feet of the Small-pox Hospital, and there has been very little small-pox there, certainly not more than at other poor-law infirmaries remote from hospitals. At the Holborn Union Infirmary, Highgate, distant about a quarter of a mile from the hospital, such cases as occurred were traceable to the visitation of friends.

'At Homerton the Fever Hospital and the Small-pox Hospital are in close

proximity within the same enclosure, but very few instances have occurred in which fever patients or convalescents have contracted small-pox. In 1876 it became necessary to receive small-pox cases into the Fever Hospital, and for some time there were fever cases at the west end and small-pox at the east end, but there was not a single case of small-pox among the fever patients, and not a single case of fever among the small-pox patients.

'One point appears to have made so much impression on several important witnesses that it should be specially noticed. Mr. Power himself clearly perceives and fully admits that the graduated distribution of disease around the hospitals, which is so remarkably illustrated in his report, is as explicable by personal communication as by atmospheric dissemination. "*With such a hypothesis,*" he says, meaning that of dissemination, "*equally as with a hypothesis of conveyance by human movements, the gradation of hospital influence from centre to periphery would be in complete accordance.*" . . . " We think it right thus to report the chief grounds for accepting or rejecting the theory of distant atmospheric dissemination of small-pox. But we feel that so long as it is not proved that "personal communication" is adequate to the explanation of the whole spread of small-pox, and so long as distant "atmospheric dissemination" is not shown to be in the highest degree improbable, so long is it essential that in the construction and management of small-pox hospitals both sources of danger should be, with the utmost care, guarded against.

'And in this conclusion we are more than supported by the opinions of witnesses of the greatest authority, including Sir William Jenner, Sir William Gull, Sir James Risdon Bennett, and Mr. (now Sir John) Simon.'

With regard to hospitals for fevers other than small-pox, the Commissioners say: 'It is therefore extremely fortunate that all evidence goes to show that well-conducted fever hospitals involve no appreciable risk to the neighbourhood. This is the testimony as well of the medical superintendents of the Asylums and other fever hospitals as of the officers of health who are in charge of the districts in which these hospitals exist. Nor have we received any statements of an opposite tendency.'

They state that it is evidently of paramount importance that the areas of the small-pox wards, as well as their administration, should be rigorously separated from those of the fever hospitals, and, further, that their construction should be such as to reduce within the smallest limits the chance of spreading infection—such, for instance, as was suggested to them by Dr. Burdon Sanderson, which he considered would render it possible to combine efficient ventilation with the collection of the whole of the air contaminated by the proximity of the patients, in such a way as to expose it either to the disinfecting action of heat or to submit it to any other process of disinfection that might be judged useful.

In their 'Practical Recommendations' they state: 'We are of opinion that the provision of hospital accommodation for persons suffering from infectious disease in the metropolitan districts should be entirely disconnected from the administration of the poor law, and treated as part of the sanitary arrangements of the metropolis. . . . Within the hospital those who are desirous of being placed in separate wards should be allowed such accommodation on paying for it. In case of ordinary accommodation it appears to us a question whether payment should be claimed even from those who can make payment without difficulty.' They also recommend that the entrance appropriated to the sick should be entirely separate from that of the tradesmen, contractors, and others; that the hospital authorities should have the entire control of the ambulances, by which all other modes of conveyance should be as far as

possible superseded ; and that various other obvious precautions, mainly for the protection of the neighbourhood, should be strictly enforced.

These recommendations are equally adapted to all hospitals for infectious diseases wherever they may be situated.

Mr. Power records as the result of his later observation (1881 to 1884) of the influence of Fulham Small-pox Hospital on the neighbourhood surrounding it, in his report made in 1885, that notwithstanding improvements in administrative circumstances since his inquiry of 1881, consisting in the limitation of the number of patients to a maximum of thirty-five, the remodelling of the internal administration of the hospital for the purpose of reducing the number of communications of the hospital staff with the outside world, the reduction and efficient supervision of communications of the outside world with the hospital, and the vigilant supervision of the ambulance traffic of the hospital :—

‘ 1. There has occurred during the epidemic prevalence of small-pox in 1884 (in the same way as before recorded for 1881) an excessive incidence of the disease on houses in the neighbourhood of Fulham Hospital as compared with more distant houses in Chelsea, Fulham, and Kensington.

‘ 2. The percentage of houses invaded in the neighbourhood of the hospital has become gradually smaller as the distance of the houses from the hospital has increased.

‘ 8. Houses upon the chief lines of human intercourse with the hospital have not suffered more than houses lying in other directions from the hospital.

‘ 4. In point of time, the excessive incidence on the neighbourhood of the hospital has only been observed while the hospital has been in use for the treatment of small-pox.

‘ 5. The appearance of excessive small-pox in the houses around the hospital was not delayed until the cases under treatment there approached the maximum attained during the year. During succeeding fortnights of active operations, while use of the hospital increased, the excess of small-pox upon the neighbourhood became less marked.

‘ In each of the foregoing respects the relation of the hospital to the prevalence of small-pox in the houses of its neighbourhood has been, in 1881 and 1884, identical with that recorded in my former report as having existed in previous epidemic periods. But I have obtained a new experience from the 1884 epidemic. It is this :—

‘ 6. The excess of small-pox on the neighbourhood of the hospital was quite and specially remarkable at a time when the total admissions to hospital had not exceeded *nine*.

‘ 7. The communications of the hospital with the outer world give no indication of having determined the behaviour of small-pox in the neighbourhood of the hospital, and may be affirmed not to have had concern with the outburst of the disease at the beginning of June.

‘ 8. In so far as communications of the hospital with the outer world have at any time operated to produce the small-pox witnessed, either their efficiency to this end has been greatest when the number of them was least and the amount of small-pox infection liable to be conveyed by them was at a minimum, or else the infection-material distributed in the special area by these agencies during the first week of hospital operations must have possessed far greater potency than that spread by them in subsequent weeks.

‘ 9. There is evidence, alike from the experience of 1881 and of 1884, that small-pox has on occasions spread round the hospital to houses at all

points of the compass in such a way that its spread cannot be accounted for unless its contagium has been conveyed through the general atmosphere. On such occasions there has been coincident peculiarity of atmospheric conditions.

'10. Neither a hypothesis of mediate distribution of small-pox by means of hospital traffic, nor a simple hypothesis of direct atmospheric convection of hospital-derived infection suffices to explain the very pronounced ability of small-pox in May, and its relative disability in July, to extend itself in the neighbourhood of the hospital.

'11. There has probably existed at Fulham some condition or conditions in excess of those heretofore recognised as affecting small-pox that have operated to produce greater infectivity of small-pox cases in May than in later months of the summer of 1884.'

Dr. (now Sir George) Buchanan, commenting on this report in his Report for 1884, says :—

'Mr. Power has, I think, established very strong grounds for believing the spread of small-pox from Fulham Hospital in 1884 to have taken place in the same way as he regarded it as having occurred in 1881; and as respects the conditions for the occurrence of such a spread, he gives reason for expecting that other factors determining this ability of small-pox remain to be discovered, some of them being concerned in the very nature of small-pox itself. . . . There are not a few indications, subsequent to the Report of the Commission of 1881, tending to the conclusion that the Asylums Hospitals in their present shape continue to cause an increase of small-pox in their several neighbourhoods.'

Mr. Power's further observations (1884-85) on the same subject entirely corroborated his previous experiences, and enabled him to reaffirm the conclusions he had already laid down.

In his comments on these observations, in his Report for 1885, Sir George Buchanan thus expresses himself:—'The whole of the experiences now on record concerning the districts of London which are in special relations with small-pox hospitals combine, I think, to form a very strong corroboration of the view of the Commission of 1881, that in the metropolis all small-pox hospitals share the disastrous ability of Fulham Hospital to spread small-pox "by some means or other" (as the Commission has it) over the neighbourhoods around them. This ability is now, I hold, proved to extend to the distance of at least a mile, and to be independent of lines of human communication. It has now (Reports of 1884-85) been shown to be exerted when the number of acute cases in a hospital has been restricted to twenty or thirty, and it was on one occasion exerted when only five acute cases were in hospital together. It has not been extinguished, as Mr. Power's recent researches and the reports of health officers show, by the regulation of methods of transit, or by the removal of opportunities for personal communication with patients in hospital.'

Inasmuch as the medical officer of health may not improbably be called upon to advise the local authority respecting the subject-matter of these reports, it is essential that he should be master of them all.

As the result of the vast experience they embody his advice should be that, whereas in the case of a hospital for infectious diseases other than small-pox a moderate degree of site isolation will suffice, it is far otherwise with a small-pox hospital, which ought to be as far removed from populous parts as local circumstances and considerations of accessibility will permit, and ought not to be placed in the vicinity of any hospital for other diseases. Difficulty in securing a suitable site may be experienced, and considerations of that

kind may influence his advice, for under such circumstances he may feel warranted in sanctioning one which he would not otherwise be satisfied with ; but those objections are less likely to be raised in respect of any site which is remote from dwelling-houses or land which is likely to be built upon.

Where circumstances of locality permit, he may even feel it advisable to recommend the use of a floating river hospital for the isolation of small-pox patients, if great difficulty in acquiring a suitable land site is experienced.

Where an isolation hospital is situated within the range of a system of telephonic communication it should be connected therewith.

Medical attendance on the patients is in no way included in the ordinary duties of the medical officer of health, but the local authority may make special arrangements with him to undertake it, and in that case should grant him extra remuneration.

Although the control of the administration of such hospitals is not specifically alluded to in the Orders of the Local Government Board relating to him, it is fitting that the medical officer of health should assume that as one of his duties, and he will concern himself with the framing and strict enforcement of regulations to be observed by the patients and staff of any hospital with the view of preventing it from becoming a centre of infection by its incidents. He will see that the friends of patients are allowed to visit them only in case of dangerous illness or approaching death ; that proper precautions are taken against the conveyance of infection by persons visiting the patients or by tradesmen or others having business at the hospital, by the despatch of letters insufficiently disinfected, by the communication of officers, attendants, and nurses with the outside world, by the exposure of infected bedding or clothing, or by imperfect drainage arrangements.

He should see that the nursing staff is ample and thoroughly efficient ; that all due consideration is had for the health and comfort of the nurses in the performance of their arduous and responsible duties ; and that while sufficient rest and recreation are allowed to them, their departures from the precincts of the hospital should not be needlessly frequent.

He should insist upon all nurses and other persons employed in or about a small-pox hospital being efficiently protected by vaccination or re-vaccination.

The ambulance will demand the attention of the medical officer ; he will have to see that means are provided for the conveyance of persons suffering from infectious disease to any hospital for their reception which may be available.

It is undesirable that any public vehicle should be used for the purpose, even if it is thoroughly disinfected after each time of use.

The conveyance may with advantage be one of the specially constructed ambulances. These are, perhaps, best adapted for large towns. In smaller districts, where the use of them might give undue publicity to the existence of infectious disease in any house during the removal of a patient, it may be preferable to use a conveyance partaking more of the ordinary type, in order that it may attract less attention when being used. An old capacious brougham may be adapted by having the lining taken out and replaced by match-boarding.

There should always be room within the ambulance, whatever form it may assume, for a nurse or attendant to accompany the patient. It should be arranged with due consideration for the comfort and safety of the patient, and should be equipped with a supply of stimulants or other necessaries which may be required during the removal.

The medical officer should assure himself that there is no loitering at public-houses or elsewhere on the part of the driver of any ambulance. With that end in view, the driver may be required to wear a uniform, as in the case of those employed by the Metropolitan Asylums Board. Such an arrangement is necessary in large towns, but is undesirable in smaller places, since it gives too much publicity to the fact of the removal of patients from particular houses; under those circumstances, if the driver cannot be relied upon to comply with instructions, it is better for a trustworthy inspector in the employ of the local authority to accompany him.

An ambulance should be set apart for the removal of small-pox patients, and should not be used for the conveyance of persons suffering from any other disease.

All ambulances should be disinfected after each time of use; they should be kept in places belonging to the local authority, either on the hospital premises or elsewhere, as may be most convenient for the accommodation of the district.

If not in frequent use, the medical officer should assure himself, by having them periodically examined, that they are in good order and fit for use at any moment; if neglected, parts of the wheels may get shrunk and loosened, and thus become unsafe.

If the local authority do not keep a horse that will serve the purpose, they should make definite arrangements with some person to provide a man and horse at a fixed charge, whenever required, in order that there may be no delay when occasion arises for using the ambulances.

In all large towns there should be a complete ambulance system under the sole control of one authority, which in most places should be the sanitary authority, so planned that every part of the districts may be within easy reach of an ambulance station, after the manner of that which has been established by the Metropolitan Asylums Board, who now have power under the provisions of the Public Health (London) Act, 1891, to allow their carriages to be used for the conveyance of persons suffering from any dangerous infectious disorder to and from hospitals and places other than those provided by themselves, and to make a reasonable charge for that use.

With regard to the distances to which persons ill with infectious diseases may be removed, the Hospital Commissioners remark: 'The witnesses who have been concerned during the last few years with the treatment of small-pox on a large scale concur, on the whole, in the opinion that while in some cases the movement of the patient is impossible, and in others only safe for very short distances, yet that with such precautions as those adopted by the Asylums Board this movement is practicable in a greater number of cases and over a greater distance than has been usually taken for granted. But this experience does not apply to fever, whether scarlet or typhoid. In these diseases there is a considerable period during which long removals must not be risked, even in cases that are not very severe.'

Notification of the occurrence of infectious diseases by medical practitioners to the local authority or the medical officer of health previously to the passing of the Infectious Disease (Notification) Act, 1889, was of a very uncertain and perfunctory nature, except in the case of fifty-nine towns in England and Wales and four in Scotland, where clauses requiring the notification of certain of these diseases had been inserted in local Acts of Parliament, the first of which came into force in Huddersfield in 1876, but that Act required notification only in cases of dangerous infectious disease which were considered to be without proper lodging or accommodation for the patient.

There is also a slight exception with regard to pauper sickness, the Local Government Board having stated in 1879 that guardians of the poor should instruct their clerks to regularly inform medical officers of health of all new cases of sickness amongst paupers after each of their meetings, and request the poor-law medical officers to give them the earliest possible information of cases of dangerous infectious disease under their charge. In the same year the Board made it incumbent upon district and workhouse medical officers, and medical officers of district and of separate workhouse schools, to furnish them with returns of pauper sickness and deaths, and to notify to them the outbreak of dangerous infectious disease. These arrangements, to say the least, have not been carried out in all districts.

Then there is the obligation to notify to the medical officer of health any cases of fever or any infectious disease imposed upon the keepers of common lodging-houses by section 84 of the Public Health Act, 1875 ; and upon the landlords of and lodgers in houses let in lodgings, other than common lodging-houses, by any bye-laws framed under the 90th section of that Act ; and upon officers of Customs detaining any ship infected or suspected to be infected with cholera, by the Cholera Regulations of 1890 made by the Local Government Board ; also the obligation to notify any infectious disease to the sanitary authority cast upon the master or owner of a canal boat, as the case may be, by the Regulations of 1878, made by that Board under the Canal Boats Act, 1877 ; and possibly upon the occupier of any tent, van, shed, &c., used for human habitation, by any bye-laws for preventing the spread of infectious disease made under the Housing of the Working Classes Act, 1885.

In a few places the local authorities have made arrangements with the medical practitioners within their districts for voluntary notification of certain infectious diseases, and for which they have in some instances undertaken to pay small fees.

Generally, however, the information about the occurrence of infectious disease in his district which the medical officer of health has received has hitherto been but meagre, and he has often first become aware of it through various casual channels, not infrequently by returns of deaths from it made to him by the registrars of births and deaths, when too often the mischief, which he might have averted if he had received early notification, has been done.

The medical men practising in his district have given him the information when it suited their purpose, but have withheld it when it was not convenient for them to furnish him with it. They are not to be blamed for this, since they are acting well within their rights ; so long as it is not a statutory duty they cannot reasonably be expected to be compelled to give the information, especially now that an Act has been passed which, when adopted by any sanitary authority, requires them to notify certain infectious diseases. It may reasonably be inferred that any local authority which refuses to adopt that Act does not wish for any information respecting the occurrence of infectious diseases in their district, and the medical practitioners within it should act accordingly.

The experiment of compulsory notification in the sixty-three towns already alluded to has been carried on in some of them for a period of from twelve to sixteen years, and, except in connection with a very few eccentric members of the medical profession, has proved in the long run a great success. That the principle of compulsory notification of infectious diseases is generally accepted by sanitary authorities as a correct one is made evident by the fact that although the Infectious Disease (Notification) Act, 1889, was not passed until August 30 in that year, by the early part of 1890 it had been

adopted by over seven hundred sanitary authorities, and that including London, to which the Act extended without adoption, and the fifty-nine towns already possessing local provisions for the purpose, more than two-thirds of the population of England and Wales were then living under a system of compulsory notification of infectious diseases, whilst the number had increased to more than five-sixths at the end of 1892.

If the Act has not already been put in force in his district, the medical officer of health will probably feel it incumbent upon him to urge the local authority to adopt it.

It does not endow him with any new power or duty, nor does it require that he should himself visit every house in which a case of infectious disease is notified. Where it is in operation he must not take it for granted that he is informed of every case, but must supplement notification by independent inquiry.

The Act applies to small-pox, cholera, diphtheria, membranous croup, erysipelas, scarlatina or scarlet fever, and fevers known by any of the following names—typhus, typhoid, enteric, relapsing, continued, or puerperal, and to any other infectious disease to which the Act has been applied by the local authority of any district.

When it has been adopted by any sanitary authority, the medical officer of health should receive from the medical man attending a case of any of those diseases in any building used for human habitation, or in any ship, vessel, boat, tent, van, shed, or similar structure used for human habitation, and not belonging to Her Majesty the Queen, and not being a ship, vessel, or boat belonging to any foreign Government, as soon as he becomes aware of its nature, a certificate stating the name of the patient, the situation of the building, &c., and the infectious disease from which, in his opinion, the patient is suffering.

In the metropolis such certificate will also give the age and sex of the patient, and state whether the case occurs in the practitioner's private practice or in his practice as a medical officer of any public body or institution, and when it refers to the inmate of a hospital it will specify the place from which and the date at which the inmate was brought to the hospital.

He should also receive a notice of the case from the head of the family, or from any other person in charge of the patient, or in default of them, from the occupier of the building.

More often than not this dual notification is not given to him; but it is, nevertheless, a useful provision, inasmuch as it makes it incumbent upon such person to give him the information even if the patient is not being attended by a medical man, and so there can be no inducement to omit such attendance with the view of concealing the infectious disease; moreover, it makes the notification by the medical attendant a less invidious task.

Where in any sanitary district there are two or more medical officers of health, a certificate has to be given to such one of them as has charge of the area in which is the patient it refers to, or to such other of those officers as the local authority may from time to time direct.

A notice or certificate under this Act may be delivered to the medical officer of health, or may be left at or sent by post to his office or residence.

If he is himself in practice, he is entitled to the fee prescribed by the Act for a certificate in respect of any patient of his own.

When the medical officer of health of any metropolitan district receives a certificate under the Public Health (London) Act, 1891, relating to a patient within the metropolitan asylum district, he has within twelve hours of the receipt of it to forward a copy to the Metropolitan Asylum Managers, and

to the head teacher of the school attended by the patient (if a child), or by any child who is an inmate of the same house as the patient.

The medical officer of health must keep a correct register of all the medical certificates and other notices under the Act which he receives, and he should see that the correct amounts are paid quarterly for those certificates.

If he thinks there has been delay on the part of any medical practitioner in certifying a case of infectious disease, he must bear in mind the frequent difficulty and uncertainty of diagnosis in the early stages of those diseases; but if, on the other hand, he has reasonable grounds for believing that such delay is wilful, or that any practitioner fails to notify, he should send a courteous communication on the subject to him, and if that fails in its object he should endeavour to see him and induce him to comply with the requirements of the Act. Rash or hasty proceedings to enforce any penalties incurred under it will result in hindering the smooth and successful working of its useful provisions.

Some amount of opposition may be expected in the early time of its adoption, but this should be regarded with leniency, inasmuch as experience has proved that when the novelty of the system has worn off and its working has become better understood, resistance to it has ceased, and it has come to be regarded in a very favourable light by all parties concerned. Not a little will depend upon the tact and discretion of the medical officer of health himself.

The names and addresses of persons notified to be ill with infectious disease should be kept as private as possible. To include them in reports to sanitary authorities and to allow them to be published in local newspapers, as we occasionally see done, indicates inexperience or want of fitness for the office on the part of the medical officer of health.

For any person to attempt to conceal the existence of infectious disease in any house is not only dangerous to the community but is improper; nevertheless, when the responsible people are allowing everything possible to be done in the way of effectual isolation or the removal to hospital of the patient, and disinfection, it is only fair and reasonable that privacy should be observed by the officers of the local authority, especially in the case of business establishments, where any unnecessary publicity might inflict a serious injury; this is, perhaps, of more particular moment in the smaller places, where every occurrence so readily becomes noised abroad.

The medical officer of health has no right of entry into any house for the purpose of inquiring as to the existence of infectious disease therein, or simply on account of there being any person so suffering within it; neither has he the right to see or examine any person affected with it; infectious disease not being a 'nuisance' within the meaning of the Public Health Acts, he derives no power of entry under them on account of it.

Under the Canal Boats Act, 1877, and the Housing of the Working Classes Act, 1885, or the Public Health (London) Act, 1891, he may, when duly authorised, enter any canal boat, tent, van, shed, or similar structure used for human habitation, with the exceptions previously named, inhabited probably by wandering people who most likely would not call in medical aid, for the purpose of ascertaining whether there is therein any person ill of infectious disease. He may also, under the powers conferred upon him by the Local Government Board's Cholera Regulations of 1890, visit and examine any ship, vessel, or boat, and examine all persons on board of her, to ascertain whether she, or any person, is infected with cholera.

In all these instances, be it noted, there is no reference to a *house*; how-

ever, section 187 of the Act of 1875 gives him power of entry on any *premises*, as well as on any vessel, for the purpose of executing, or superintending the execution of, any regulations made by the Local Government Board under section 184 of the Act, which, amongst other things, may provide for house-to-house visitation, but there are no such regulations in existence.

It is fitting that the law of the land should not invest the medical officer of health with the right to visit any person ill with infectious disease in any house. If it did, the whole public health service would suffer, not only through the resistance of the public, but also through the opposition of the medical profession, who would rightly refuse to tolerate such interference.

The Local Government Board in their Orders of March 1891 direct that on receiving information of the outbreak of any infectious disease the medical officer of health shall visit the *spot*, but not the *patient*, without delay, and inquire into the *causes* and *circumstances*, but not into the *existence*, of such outbreak.

If any person who is ill with infectious disease is not under the care of a medical man, the medical officer of health may find it necessary to attempt to gain the permission of the people in charge to himself examine such person; but he should make a point of not seeing anyone who is being attended by a medical practitioner except with him, or with his permission, or at his request. The Act does not impose upon him the duty or right of visiting patients.

It is not within his province to verify the diagnosis of medical practitioners, but if he has reasonable grounds for doubting the existence or the nature of an alleged case of infectious disease he ought to ask the medical attendant to allow him to visit the patient with him; if that permission is refused, he will generally have to rest contented with the diagnosis which has been made, he having no more right to see the patient of a medical man than any one practitioner has to visit the patient of any other. The only justification for his breaking through this rule would be if he had very strong grounds for believing that any notification had not been made in good faith. A decision to this effect has been given by the Local Government Board; but such a suspicion is a grave matter, and ought not to be lightly entertained.

On receipt of the notification of a case of infectious disease, the inspector of nuisances will probably visit the premises in the first instance, and obtain full particulars, which should afterwards be entered in a book kept for the purpose, so that a complete record of cases of infectious diseases may be preserved.

The inspector will deliver any disinfectant which the local authority may supply; also any instructions giving plain directions for the measures necessary for preventing the spread of infection, and setting forth the penalties incurred by neglecting them, which the medical officer will probably have had printed for the purpose.

Where there is a public library, inquiries should be made whether any book from it is in the infected house; if there is, it should not be returned until means have been taken for preventing it from carrying infection, and instructions should be given that no book must be taken from the public library so long as the house remains infected.

The inspector will direct the attention of the medical officer to any matter he considers he should be informed of; and if he finds that the patient cannot be properly isolated at home he will inform him of the fact, when he will

endeavour to get the patient removed, with the consent of the medical attendant, to any hospital which may be provided.

The medical officer must not undertake the great responsibility of removing a patient in opposition to the opinion of the practitioner in attendance.

Where there is proper hospital accommodation the public soon learn to appreciate it, and are then not loth to avail themselves of the use of it when occasion arises; but sometimes unreasonable objections may be raised, and then, if the removal is urgently called for, the patient being without proper lodging or accommodation, or lodged in a room occupied by more than one family, or being on board any ship or vessel, or being lodged in any common lodging-house, the medical officer of health may have to give a certificate for the purpose of obtaining an order of a justice for the removal of the patient to a hospital.

All removals to hospital should be supervised by persons in the employ of the local authority, and should be conducted with due regard to the comfort and safety of the patient and the public, and to the avoidance of any unnecessary publicity.

As soon as possible after the removal of the patient, or on the termination of the illness, the room which has been occupied and all its contents should be disinfected to the satisfaction of the medical officer, and, if possible, under the direction of someone who is employed by the local authority.

At the time of his first visit the inspector should leave a circular letter or a post-card addressed to the medical officer, by which the friends of the patient can intimate to him when the medical attendant declares the case, when treated at home, to be at an end, and the house to be ready for disinfection.

The inspector will warn the occupier of the house against doing anything whereby infection is likely to be spread. He will see that children living in the house are excluded from attendance at any school, and if any inmates are following an occupation through which infection may be conveyed to others he will warn them of the danger and of any risk which may be incurred.

In times of serious outbreaks the medical officer will himself give much personal attention to the circumstances of the cases, and will himself visit the premises where they occur.

In the metropolis infected persons are prohibited from milking any animal, picking fruit, engaging in any occupation connected with food, or carrying on any trade or business in such a manner as to be likely to spread infectious disease.

By some local Acts powers are given to sanitary authorities to prevent persons living in infected houses from following their employment, to isolate the healthy inmates, to order the closure of houses of business where there is infectious disease, to grant compensation for any consequent loss of earnings, or to provide nurses for the sick.

The exercise of these various powers will greatly depend upon the advice given by the medical officer of health; they may be of great service in arresting the spread of infectious disease, and he should endeavour to see that they are used judiciously where they are in force, and that equitable compensation is given where it is allowed.

He should have notices printed to be addressed to the head teachers of elementary schools, in order that they may be promptly informed of the occurrence of infectious disease amongst any of their scholars, and be requested not to allow any children from the infected houses to attend their school for a named period. One of these should be sent as soon as it has been ascertained at what school they have been attending.

General Acts of Parliament do not give local authorities the power to close schools during times of epidemics; but some can do so under local Acts, and the Code of Regulations of the Education Department, which refers only to public elementary schools, and not to *dames'* and other private schools or to Sunday schools, has conferred that power upon them since 1882; article 88 (1898) stating that 'the managers must at once comply with any notice of the sanitary authority of the district in which the school is situated, or any two members thereof acting on the advice of the medical officer of health, requiring them for a specified time, with a view to preventing the spread of disease, either to close the school or to exclude any scholars from attendance; but after complying, they may appeal to the Department if they consider the notice to be unreasonable.'

It will be advantageous for the sanitary authority to authorise the medical officer of health to act upon his own responsibility in giving notice, when necessary, to exclude any scholar from attendance at school, in order to avoid the delay of waiting for a meeting, since prompt action is essential. He might, if desired, report all such cases at their next meeting.

Where notification of infectious disease is in force, with consequent early intimation of cases, much more may be done, and with less interruption in the system of elementary education, towards preventing the spread of disease through schools, without having recourse to the more serious step of closing them, than in those places where the medical officer receives only casual notice of disease, especially when a proper understanding exists between him and the school managers, as there ought always to be. They should work harmoniously together, and should co-operate with the view of protecting the public health; indeed, their interests in this behalf are identical, for disease cannot attack the scholars without affecting injuriously the interests of the school.

If the managers have the impression that the medical officer is inclined to suggest the closure of a school without just cause, they are apt to mistrust him, and to resent his interference at other times when, perhaps, it may be of vital importance to have their school closed. On the other hand, if they find that in the first instance he is desirous of giving a full and fair trial to the milder method of excluding children living in infected houses from attendance at school, they will the more readily listen to his counsels when, in his opinion, it is necessary to pursue the severer course.

Occasionally it may be found wise to exclude from school for a time all the children living in a particular street, or in some detached part of a village, or in a whole village when one school serves for more than one place, where there may be an excessive incidence of infectious disease, inasmuch as some of the children may be incubating it, or their clothes may have become infected from association with others in infected houses.

Though there is no power of closing Sunday schools, those having control over them should be asked to act in unison with the managers of the day schools, otherwise the prevention of disease achieved during the week may be undone on the Sunday.

The medical officer will find it useful to arrange to be informed by the school teachers, or the school attendance officers, respecting children who have been detained at home on account of alleged illness. He may then prosecute his inquiries in the localities indicated, and thus be able to discover some concealed or unrecognised cases of infectious disease.

The teachers and school attendance officer should be asked to inform him if they observe any children with obvious symptoms of infectious disease;

in fact, it should be an understood thing that they will at all times give him immediate information of infectious sickness amongst the scholars.

Reference may with advantage be made to a memorandum on this subject by the medical officer of the Local Government Board, dated 1890. In it he remarks: 'As regards duration of exclusion from school of particular children, the time to be specified will vary in different diseases and different cases, and in this matter the sanitary authority will doubtless be guided by the advice of their medical officer of health, who may properly be entrusted with some general duty of acting for the authority in this subject-matter.'

'Medical officers of health having to specify a time during which any scholars are to be excluded from attendance at any school, should have regard as far as practicable to the circumstances of the particular scholars suffering from infectious disease or living in infected households. Not only the nature of the infection and the length of illness, but the environments of the individual as affecting the retention of infection will deserve consideration. The period of exclusion, for example, will need to be different according to the conditions of a patient's lodgment, according to the sufficiency of the separation that can be effected between a patient and excluded scholars, and according to the opportunities of effectual disinfection that can be afforded to the household. Thus a hard-and-fast rule, such as has been laid down in some districts where scarlatina has been present, that no child shall go to school from an infected house for three months after the disease has begun in that house, is not to be commended. It is indeed possible that, under the circumstances of a particular household, a child convalescent from scarlatina or living in the same house with convalescents should not in the interests of other children be permitted to return to school until after so long a period as this; but the same ought not to be assumed of all households in the district that may be invaded by scarlatina. The better plan would be for the sanitary authority to secure, during a shorter period, the exclusion of individual sick persons and their housemates from school; and when that period is about to expire to cause fresh inquiry to be made as to the expediency of further exclusion, and, if found requisite in particular cases, to cause fresh notice to be given to the school managers.'

As to the *closing of schools*, he says: 'This, by more seriously interfering with the educational work of a district, is a much more grave step for a sanitary authority to take than to direct the exclusion of particular scholars. It is a measure that seldom ought to be enforced, except in presence of an actual epidemic, nor even then as a matter of routine, nor unless there be a clear prospect of preventing the propagation of disease such as could not be looked for from less comprehensive action.'

'The mere fact that in an epidemic many of the sufferers are school children does not necessarily show that the disease was caught at school; but the school may with probability be regarded as spreading infection if in a large majority of households attacked the first case be a child attending school; and with still greater probability if a number of children living at a distance from one another, and with no circumstances in common except that they attend the same school, should be simultaneously attacked, and if it can be ascertained that a child or teacher in an infectious state has actually been attending the school. . . .

'In deciding whether an outbreak of infectious disease among children of school age may be best combated by closing the school, or whether it will suffice to exclude the children of infected households, the two most important points to be considered are:

'The completeness and promptness of the information received by the

officers of the sanitary authority respecting the occurrence of infectious cases.

'The opportunities which exist for intercourse between the children of different households elsewhere than at school.'

The action of the local authority with respect to closing schools will depend greatly upon the information and advice they receive from the medical officer of health.

Whenever a school is closed he should certify to the managers that the closure is requisite on account of a local epidemic, so that in compliance with Article 88 (a) of the Education Code the school may not suffer any avoidable pecuniary loss.

When a school has been closed on account of the prevalence of infectious disease it ought to be disinfected to the satisfaction of the medical officer of health before it is again opened; and, as a precautionary measure, during the prevalence of an epidemic, the fumigation of the schoolroom every week by burning sulphur may be recommended, and may usually be done on Saturdays without interruption of school duties.

The medical officer of health is not concerned with the administration of the laws relating to *vaccination*, but in a 'Memorandum on Re-vaccination' issued by the medical officer of the Local Government Board in 1888, Sir George Buchanan writes: 'Medical and sanitary officers and the medical profession generally are, therefore, invited to urge upon parents and guardians the importance of having their children re-vaccinated at the age of twelve years or thereabouts, and to urge upon all persons beyond this age who have not yet been successfully re-vaccinated the duty of obtaining for themselves the additional protection which may be had by this means.'

Referring to the same subject, in a 'Memorandum on the Steps specially requisite to be taken in Places where Small-pox is Prevalent,' issued from the Local Government Board in 1888, the medical officer remarks: 'The sanitary authority of any district into which a case of small-pox may be brought, or in which it may occur, should immediately, on obtaining information of the occurrence, give notice to the board of guardians (the local authority for vaccination purposes) or the vaccination officer, in order that all requisite measures in regard to vaccination may be taken. The sanitary authority should also instruct their officers to assist in the administration of the Vaccination Acts by spreading a knowledge of the advantages of vaccination and re-vaccination, and by giving to the vaccination officer any information they may obtain as to children and others unprotected by vaccination.'

The 'General Memorandum on the Proceedings which are advisable in Places attacked or threatened by Epidemic Disease,' issued from the Medical Department of the Local Government Board by Dr. Thorne, C.B., in 1892, may be consulted with advantage for guidance in dealing with outbreaks of disease.

The medical officer of health may be called upon to advise the local authority with regard to the desirability of their taking proceedings in particular cases for the infliction of the statutory penalties for the exposure of infected persons or things, and in the event of such proceedings being taken he may have to give evidence in support of them.

Where gross carelessness in that behalf is exhibited he may find that a well-chosen case, successfully prosecuted, may prove a useful example, inducing greater care, with consequent benefit to the district.

In any case in which infectious disease is introduced into his district from some other part, and he ascertains where it has been contracted, he should, as a matter of courtesy, inform the medical officer of health hold-

ing office in the district in which the implicated place is situated of the fact.

The methods of investigating outbreaks of infectious disease should be thoroughly mastered by the medical officer of health.

In any inquiry he may have to make into the circumstances of an outbreak, he will be greatly assisted if he has made a complete and clear record of every case of illness in the book which, as previously described, ought to be kept for the purpose. He will not, of course, confine his attention solely to the matters under the various headings in that book, but will also make careful note of any other facts which may come to his knowledge, even if their bearing on the subject of inquiry may not at first be apparent, for he must recollect that what may in an early stage of the inquiry appear of trivial import may subsequently prove to concern it very closely.

It is to an exhaustive process of inductive reasoning that he must look to complete his chain of evidence in unravelling the not infrequently intricate circumstances of disease outbreaks, and he may often be still further helped by simultaneously bringing the numerical method of investigation to his aid. The four successive steps in inductive reasoning may be summarised as Observation, Hypothesis, Deduction, and Verification.

By the exercise of his senses, and it may be by experiment, the investigator must first make extensive and minute observations, in order to collate all the facts which can possibly have any bearing upon the subject of the inquiry he may have in hand. The greater the exactness and method displayed in this stage, the greater will be the chance of his ultimate success.

Having, it may be with much patience and perseverance, exhausted the resources at his command in gathering together this preliminary information, he will proceed to the second step of his inquiry, and will formulate an hypothesis. In so doing he will assume as true some one or more propositions which may appear to tally best with the facts he has ascertained.

The third step having been reached, he must now bring his deductive reasoning powers into play, and will formulate various propositions which will be true if his hypothesis be correct. He should make his deductions as numerous, and under as varied circumstances, as possible, verifying them as far as he is able.

In the fourth step he must not hastily assume that his hypothesis is correct, simply because it is in accord with a few of the facts, but must compare his deductions from it with the facts already in his possession, and if possible must make fresh observations or experiments in order to prove or reject it. If in this comparison he finds his deductions and observations disagree in several respects, in all probability he will have alighted upon a false hypothesis, and in that event must modify it in some particulars, or must cast about for one more accurate. If, however, it proves to be true in many of its results, and especially if by its means he is able to correctly predict what he would never otherwise have believed or discovered, he may feel almost assured of its truth.

To take an imaginary example, let us suppose that in inquiring into an outbreak of typhoid fever the medical officer of health has found from his preliminary observations that a large proportion of the persons affected reside in separate towns or villages, in which there are entirely separate systems of sewerage; that the drainage of the majority of the houses in which they reside is properly constructed and in good order; that they have been obtaining their milk from several dairymen, most of whom have procured their milk from different farms; that the incidence of the disease has not fallen upon the customers of only one or two of the dealers trading in the

implicated area, but that the customers of all have been affected in somewhat equal proportions whilst persons served by them in other parts have escaped ; that no other article of diet procured from a common source has been partaken of by the patients in a larger proportion than by other inhabitants who have not suffered ; that there is one public water-supply laid on to all the towns and villages in which the disease has broken out.

Seeing that the water-supply appears to be the only connecting link between the sufferers, he may now feel in a position to form the hypothesis that it is concerned in the cause of the fever outbreak.

In pursuance of the third step of his reasoning he will proceed to formulate deductions from this hypothesis somewhat as follows :—

That the persons affected should constitute a considerable percentage of the whole consumers of the suspected water-supply. That the disease should have fallen chiefly upon any members of the affected households who had been the largest consumers of the water. That any persons who happened to have drunk the water only after it had been boiled should not have suffered. That if any house has some other water-supply as well as the public supply any of the inmates who may have used the former exclusively should have escaped attack. That the outbreak should not extend to any house having an entirely different water-supply. That if any persons living in a house so circumstanced were attacked they should have had some communication with the patients or their houses. That if the public supply had become contaminated, the date of first attack in a large proportion of the houses would nearly coincide. That there must have been an opportunity for the water to have become contaminated with infective discharges from some antecedent case of typhoid fever, either at its source or during the course of distribution. Proceeding then to verify his deductions, he will make further inquiries which may result in establishing most of the propositions which he has formulated. For instance, he may thus ascertain that the disease has invaded a certain considerable percentage of the houses in the infected area to which the suspected water-supply is laid on, and that in those houses it proves to be a fact that the sufferers are amongst the largest water-drinkers, whilst others, more cautious than the rest, having drunk only boiled water, have escaped infection ; that in houses where there is a duplicate supply from wells, the consumers of that have also remained unaffected, some, at any rate, of the other inmates who have partaken of the public supply having fallen ill ; that the disease has passed by most of the houses with only private water-supplies, but sometimes that has not been the case, whilst in those exceptional instances he is able to prove that the sufferers, owing to relationship or friendship, have either visited, or been visited by, persons residing in some of the infected houses ; that nearly a whole village to which the water has not been laid on has escaped infection, although surrounded by places severely affected ; that, in fact, during the earlier part of the outbreak the disease did appear in many houses within a short space of time.

It may be that, as frequently happens, he fails to trace the infection to its starting point from some antecedent case of typhoid fever. However, having verified so large a proportion of his deductions, he may at first feel warranted in concluding that the hypothesis he has formed is correct. But on further investigation he may find that several other places to which the outbreak has not extended are served by the same water-supply. He must accordingly reconsider his hypothesis, and either modify it or abandon it for a fresh one.

Let him still assume that the water has been the cause of the outbreak, but that only a portion of the supply has been implicated. In that case he

must endeavour to discover the cause of the unequal disease-producing power of the water in the infected parts as compared with that in the rest of the water-area, and for that purpose he must formulate fresh deductions.

His propositions might be :—

That there are two sources of water, the one filling the mains of the infected area, which has become contaminated, the other, which has remained unaffected, being distributed to the rest of the water-area. That there are direct services between the water-mains and some water-closets in the infected area. That there is some defect in a branch main commanding exclusively that area, or in some house-service pipe connected with it.

He then prosecutes further inquiries and observations, by which he discovers that there is only one source of supply for the whole water-area, and that there is no direct communication between the water-mains and any water-closet, all of which are provided with separate flushing cisterns.

Lastly, he examines a plan of the whole system of water-mains, observes a certain branch commanding exclusively all the places in which the outbreak has occurred, and comes to the conclusion that there must be some defect in it which has allowed the water passing through it to become contaminated.

On having that branch laid bare he finds a broken pipe or leaking joint in ground sodden with filth from a defective sewer or cesspool, or possibly even within a sewer.

He now concludes that his modified hypothesis is correct, but may deem it advisable to obtain still more conclusive proof by experiment, and for that purpose will perhaps deposit some strong brine or other easily determinable innocuous substance at the site of the leak, to ascertain whether the character of the water is affected by insuction through it.

If he succeeds in ascertaining that shortly before the commencement of the outbreak the sewer or cesspool has received specifically infected discharges from some person suffering from the fever, his strong conviction as to the cause of the epidemic will be changed to a certainty.

If he cannot trace a disease outbreak to any ordinarily accepted channel of infection he will cast about for one less generally observed, such as the existence of some similar disease amongst animals and the possibility of its communicability to man. In this regard he must not neglect the possibility, which is too often overlooked, of insuction into water-mains taking place, not only during intermission of supply but even into pipes 'running full.' Sir George Buchanan has drawn attention to this in a Report on an Epidemic of Enteric Fever at Croydon in 1875, and an illustration of it, and of the danger arising from closet-pans being served directly from water-mains, is to be found in the late Mr. Spear's Report on an Epidemic of Enteric Fever in the Mountain Ash Urban Sanitary District, Glamorganshire, appended to the Report of the Medical Officer of the Local Government Board for the year 1887, in which he clearly showed an extensive outbreak of that fever to have been due to causes of this nature.

In the case of diseases which may be readily communicated from person to person through the air the medical officer may find it advantageous, under some circumstances, to deal with houses with first attacks only, in contradistinction to the total number of cases, inasmuch as the disease, having once gained entrance to a house, may spread locally amongst the inmates, independently of the original cause of the first attack.

It may happen that the opportunity of his tracing an outbreak through its full extent may be restricted owing to the limitation of his district, and in this is seen one of the advantages of the medical officer being appointed for a combination of the smaller sanitary districts ; in the event of such a contin-

gency the investigation may have to be undertaken by an inspector of the central medical department, or possibly in future the medical officers of county councils may prove of service in that behalf.

Reference may be made to Mr. Power's Report on Milk-Scarlatina in London in 1885, appended to the Report for that year of the Medical Officer of the Local Government Board. A perusal of that masterly investigation and unravelling of the intricate circumstances of a disease outbreak will serve to show the character of the methods and inferences required in such inquiries.

A *mortuary* for the reception of dead bodies before interment is a necessity in populous places, which the medical officer of health should endeavour to induce the local authority to provide, if not already in existence. It obviates the obligation of keeping dead bodies in small houses to the detriment of the inmates' health, and especially is it a want in the event of deaths from infectious disease.

When an authority decide to build a mortuary for their district, the medical officer will probably be consulted by them with respect to its site and construction, and regarding the compilation of any bye-laws for its management which they may wish to make. Useful suggestions as to these points are to be found in the memorandum of the Local Government Board attached to their model bye-laws (Series No. XV.) with respect to mortuaries.

When the local authority have provided a mortuary, the medical officer may have to give a certificate when required to enable a justice to order the removal thereto of the body of anyone who has died of an infectious disease which is retained in a room in which persons live or sleep, or of any dead body which is in such a state as to endanger the health of the inmates of the house or room in which it is retained, and to enable him to direct the same to be buried within a time to be named in such order.

Burial-grounds may come within the observation of the medical officer of health, and, although having no direct control over them, he may have to report to the local authority upon the state of any there may be within his district. If he finds any over-filled and liable to be detrimental to health he should bring the matter to their notice, and they would then call the attention of the controlling authority to it.

Where he finds any burial-ground is without proper space for burial, or by reason of its situation in relation to the water-supply, or on any other account is dangerous to the public health, or where for the protection of the public health it is expedient to discontinue the use of any burial-ground, no other suitable one being available, he may have to advise the local authority of any urban or rural sanitary district to provide a cemetery under the powers given them by the Public Health (Interments) Act, 1879, and to assist them in framing bye-laws with respect to the management of such cemetery.

The regulation of burial-grounds provided under the Burial Acts does not concern him, such being subject to regulations issued by the Home Office. A summary of a 'Memorandum on the Sanitary Requirements of Cemeteries,' issued by the Local Government Board, will be found on page 728.

New streets and buildings in urban sanitary districts, in the districts of rural sanitary authorities who have obtained the powers of an urban authority for the purpose, and in the metropolis, may be regulated by bye-laws. When the local authority contemplate framing any for that purpose the medical officer of health will probably have to advise them concerning any matters affecting health involved in them, and he will not infre-

quently be called upon to give his opinion respecting the application of any which are in force in so far as they apply to anything influencing the public health.

Under the model bye-laws respecting this subject issued by the Local Government Board (series No. IV.), power of entry for inspection is given to the surveyor only, and not to the medical officer of health.

Legal proceedings may be instituted and carried on by the medical officer of health when he is authorised by a resolution of the local authority, either generally or in respect of any special proceeding; and when so authorised he may appear for them before any court or in any proceedings they are authorised to undertake under the Public Health Acts. It is, however, not desirable that he should undertake this duty; it is better that it should be performed by the clerk of the authority, who is generally a member of the legal profession, or by the inspector of nuisances, assisted by the clerk.

Section 266 of the Public Health Act, 1875, provides that if notices, orders, &c., require authentication by the local authority, the signature of them by their clerk, surveyor, or inspector of nuisances shall be sufficient authentication. This would make it appear that the signature of the medical officer of health would not authenticate a form of notice requiring the abatement of a nuisance; but on again referring to the 191st section of that Act it will be seen that he may exercise any of the powers with which an inspector of nuisances is invested by the Act; so he has the power to sign such notices, though it is not desirable that he should do so as a rule; it is better that the inspector should.

When a person on whom a notice to abate a nuisance has been served makes default in complying with any of its requisitions within the time specified, or if the nuisance, although abated, is in the opinion of the local authority likely to recur, the medical officer, acting on their instructions, may make a complaint relating to the nuisance before a justice for the purpose of obtaining a summons requiring the person in default to appear before a court of summary jurisdiction; but it is better that such complaint should be made by some other officer of the local authority, and generally it is made by the inspector.

However, when a person has been so summoned, the medical officer will often have to give evidence at the hearing of the case. In doing so he should express himself clearly, concisely, and in simple language; avoiding, as far as he may be able, any technical terms which may not be generally understood. He must not allow his zeal for the improvement of his district or for the success of his cause to prompt him to overstrain any opinion he may have to express, or to exaggerate any fact he may have to depose to, in any way that might unfairly affect or prejudice the defendant's case. The Public Health Act, 1875, distinctly states that false evidence given on oath under any of the provisions of that Act is punishable as perjury; but it is not at all probable that any medical officer would lay himself open to the infliction of those penalties.

When notes have been made at the time of observation of any circumstance connected with the case, the medical officer may refresh his memory from them, but he cannot read them out as evidence. If he has made a copy of his notes, on account of their being indistinct, or for any other reason, he should preserve the original for production if required.

Dairies, cow-sheds, and milk-shops have been placed under the control of urban and rural sanitary authorities by the Contagious Diseases (Animals) Act, 1886; in the metropolis they remain subject to the control of the Corporation of London and of the London County Council, under the pro-

visions of the Public Health (London) Act, 1891. To those authorities is entrusted the duty of enforcing the provisions of the Dairies, Cow-sheds, and Milk-shops Order of 1885, and the Amending Order of 1886, and their medical officers will have to assist them in so doing.

For the purpose of enforcing the Orders and any others which may be made by the Local Government Board, and any regulations or bye-laws made thereunder by local authorities, those officers are to have the same right to be admitted to any premises as the sanitary authorities and their officers have for the purpose of examining as to the existence of any nuisance thereon.

If they are refused admission to any premises they have the same power as under the Public Health Acts to obtain a magistrate's order for admission, which can be enforced under the powers of those Acts.

They must not, however, enter any cow-shed or other place in which an animal affected with any disease is kept, and which is situated in a place declared by the local authority within the meaning of the Contagious Diseases (Animals) Act, 1878, to be infected with such disease, except with the permission of such authority.

The word 'animal' includes cattle, sheep, goats, and all other ruminating animals, swine, horses, asses, mules, and dogs; 'cattle' means bulls, cows, oxen, heifers, and calves.

The term 'disease' means cattle-plague or rinderpest, contagious pleuro-pneumonia of cattle or pleuro-pneumonia, foot-and-mouth disease, sheep-pox or sheep-scab, glanders, farcy, swine fever, anthrax, and rabies.

The occurrence of any other infectious disease, or of either of those diseases among any animals other than those named above, will not hinder those officers from entering a place in which any animal is kept.

It will probably be found convenient for them to obtain from the local authorities, under the Act of 1878, of their districts a general authorisation to enter any cow-shed or other place in which there is any diseased animal, when situated in an infected place.

Mention is not made in the Act of 1886 of any particular officers who are to be entrusted with the execution of its provisions; it gives all alike a statutory right of entry to any premises for the purpose of enforcing any Orders and Regulations.

The clerk of the local authority will be concerned with all legal questions arising out of the administration of the Act, and will have control of any proceedings at law that may be necessary.

Upon the inspector of nuisances will probably devolve the chief part of the work required for enforcing Orders and Regulations, and probably he will, under the direction of the clerk, conduct proceedings for prosecuting offences and recovering penalties which may be incurred.

The medical officer of health will have to advise the local authority on questions arising out of the enforcement of Orders of the Local Government Board, and upon the framing and enforcing of any Regulations they may make under such Orders. He will also advise the inspector upon any points concerning which he may be in doubt, and will exercise a general supervision over the enforcement of the Orders and Regulations.

In the event of the outbreak of fever or any infectious disorder which may possibly be due to the consumption of infected milk, he will have important duties to perform in the matter of making inquiry into the causes of epidemics, and of advising the local authority on the adoption of such measures as may appear to him to be necessary when he possesses evidence of human disease being spread through the medium of milk from any dairy,

cow-shed, or milk-shop. Under those circumstances he will, of course, exercise much tact and discretion in prosecuting such inquiries, for untold injury might be inflicted upon persons engaged in the trades through any want of caution on his part.

In the metropolis, and in districts where the 4th section of the Infectious Disease (Prevention) Act, 1890, is in force, he has additional powers of inspection of dairies which are described with the provisions of that Act.

Where there is a surveyor, the medical officer should seek his advice in any case in which important structural alterations may be contemplated.

If the work in any district should prove to be heavy, the officers chiefly concerned would fairly be entitled to some extra remuneration for the additional duties thus cast upon them.

The medical officer of health has no powers or duties in respect of contagious diseases amongst animals in dairies, or cow-sheds, or elsewhere, except so far as relates to milk from diseased cows. Such matters are still to be dealt with by the same local authorities as under the Act of 1878; but the Anthrax Order of 1892, made by the Board of Agriculture, requires that whenever an inspector under the Contagious Diseases (Animals) Acts receives notice of the occurrence of anthrax amongst animals he shall forthwith give information of the receipt of the notice to the medical officer of health of the district in which the diseased or suspected animals are located, though it does not impose upon this officer any duty or obligation in respect of that information.

In a circular letter addressed in 1886 to local authorities, the Local Government Board observe that they 'understand that the Agricultural Department of the Privy Council are of opinion that it is desirable, for the due carrying out of the Contagious Diseases (Animals) Acts, that the officers of sanitary authorities should give notice to the county authorities of any disease of animals found by them in any dairy or cow-shed; and the Board suggest that the sanitary authority should give directions to their officers to furnish this information to the county authority.'

It would be of further advantage if the local authorities acting under those Acts were to instruct their officers always to furnish medical officers of health with information of the occurrence of any disease of animals in their districts, so that they might be prepared to obtain, if not previously furnished with it, the needful permission to enter any cow-shed or other place in an infected place, and so be able to duly enforce the article of the Order of 1885 restricting the sale of the milk of diseased cows. That article cannot be enforced unless a cow is ill with either cattle-plague, pleuro-pneumonia, foot-and-mouth disease, anthrax, or rabies; it will not apply in the event of a cow suffering from any other disease, not even from tuberculosis, or any disease suspected to be causing diphtheria or scarlatina, such, for instance, as the Hendon cow-disease; the order does not prohibit the sale of the milk of cows thus affected.

It is incumbent upon the medical officer of health to exercise these powers in respect of dairies, cow-sheds, and milk-shops with due regard to the large interests concerned; at the same time he must see that the control of those in his district is something more than nominal, whilst in no respect needlessly harassing. He must insist, and insist firmly, on all reasonably practical precautions against infection being rigidly adhered to, but he must not attempt to convert old cow-sheds and dairies into ideal standards of perfection, or to make such demands as might well be satisfied by large concerns, but which would crush out the smaller men; he must attempt to do as much as he fairly can to prevent disease without causing any expendi-

ture beyond what the persons carrying on the trades may be reasonably called upon to incur.

There are various matters in respect of which offences, punishable by fines, may be committed by such persons ; it is, therefore, only fair to them that it should be defined as clearly as may be what those offences are, and that as little as possible should be left dependent upon the caprice of this or that officer.

For the accomplishment of this end it is necessary that clear, concise, and well-defined regulations should be made. When advising on the formulation of such regulations, the medical officer must have regard to the general principles to be kept in view, which are—that the regulations must adhere most rigidly to the letter of the law ; that they may in no respect go outside or beyond the specific matters in respect of which powers have been given to local authorities to make them ; that, like all bye-laws, they must only amplify and particularise such matters, but must not merely re-enact anything that has been specifically enacted in an Act of Parliament or in Orders made thereunder ; and that they must not be of too restrictive a character or repugnant to the laws of the land, but must be reasonable, certain, determinate, and of general application, as they will have locally the binding effect of a statute.

Although he may consider matters other than those individually named in the Acts and Orders to have a distinct bearing on disease prevention, yet he cannot comprise them in the regulations, because they would be *ultra vires*.

The Infectious Disease (Prevention) Act, 1890, invests the medical officer of health of any urban or rural sanitary district in which it has been adopted with some special powers, which also belong to all metropolitan medical officers, similar provisions being included in the Public Health (London) Act, 1891.

Section 4 gives him power to inspect any dairy, within or without his district, if authorised by an order of a justice having jurisdiction in the place where it is situated, and, if accompanied by a veterinary inspector or some other properly qualified veterinary surgeon, the cattle therein, when he has reason to believe that milk supplied therefrom has caused, or is likely to cause, infectious disease amongst any persons within his district. If he is of opinion that it has been caused by the milk, he must make a report to his local authority, which must be accompanied by any report he may have received from a veterinary inspector or veterinary surgeon, and they may, if necessary, make an order prohibiting the supply of milk in their district. When the medical officer or the local authority is satisfied that the milk-supply has been changed, or that the cause of the infection has been removed, the order must be withdrawn.

He must undertake this inspection himself, since a like power is not conferred upon the inspector of nuisances, and he may for the purpose enter any farm, farm-house, cow-shed, milk-store, milk-shop, or other place from which milk is supplied, or in which it is kept for purposes of sale.

Having thus the power to go into any other district for the purposes of this section, it is very desirable that he should take the earliest opportunity of informing the medical officer of any district he may enter of the circumstances of the case, so that they may act in concert with each other.

Although it is quite necessary that the interests of the dairyman should be reasonably safeguarded, yet it is to be wished that a less cumbersome method of securing that end should have been devised.

Section 5 enables the medical officer to give a certificate for the cleansing

and disinfection of any infected house, room, or article contained therein, which has then to be done to his satisfaction.

Under section 6, when generally empowered by the local authority to do so, he may, by notice in writing, require the owner of infected bedding, clothing, or other articles to deliver them over to an officer of the authority for removal for disinfection.

He might certify as to the proper disinfection of houses, &c., for the purposes of the 7th section.

Under section 8, his sanction in writing, or that of another registered medical practitioner, has to be obtained before the body of a person who has died from any infectious disease may be retained for more than forty-eight hours in any room used at the time as a dwelling-place, sleeping-place, or work-room.

He may give a certificate under the 9th section to prevent the removal, except for burial, of the body of any person dying of infectious disease in any hospital or place of temporary accommodation for the sick.

He may make application, under the 10th section, for a justice's order for removal to a mortuary and burial of the body of any person who has died from any infectious disease which remains unburied in a room used at the time as a dwelling-place, sleeping-place, or work-room, for more than forty-eight hours after death without his sanction, or that of another registered medical practitioner; or of the dead body of any person when retained in any house or building so as to endanger the health of the inmates of such house or building, or of any adjoining or neighbouring house or building.

He may make a representation, under the 12th section, to a justice for the purpose of getting an order from him for the detention in a hospital for infectious diseases of any person who is there ill with any infectious disease, and who on leaving would not be provided with lodging or accommodation in which proper precautions could be taken to prevent the spread of infection.

The adoption of *Part III. of the Public Health Acts Amendment Act, 1890*, affects the medical officer of health in urban and rural sanitary districts but slightly.

Possibly he might be authorised by a local authority to enter any premises for the purpose of examining them to ascertain whether any chemical refuse, waste steam, or any liquid of a higher temperature than 110° Fahrenheit, is turned into their sewers in contravention of the 17th section, and if entry is refused him he can then obtain a justice's order for admission in the prescribed manner.

Under the 21st section he may be concerned with the condition of sanitary conveniences used in common.

The 28th section empowers him to examine, and, if necessary, to seize any article of food whatever, without reference to the limited number enumerated in the 116th section of the Public Health Act, 1875.

He may be called upon to advise an urban sanitary authority upon questions connected with health involved in framing and enforcing bye-laws under the 23rd and 26th sections, and also a rural sanitary authority, so far as they are concerned with those sections.

SCOTLAND AND IRELAND

The remarks in the foregoing pages respecting the powers and duties of medical officers of health under the Acts enumerated below apply to those officers in Scotland and Ireland, namely:—The Factory and Workshop Acts,

the Housing of the Working Classes Acts, 1885 and 1890; the Merchant Shipping (Fishing Boats) Act, 1888; the Rivers Pollution Prevention Act, 1876; the Alkali, &c., Works Regulation Acts, 1881 and 1892; the Sale of Horseflesh, &c., Regulation Act, 1889; the Sale of Food and Drugs Act, 1875, and the Amending Act of 1879; the Margarine Act, 1887; the Infectious Disease (Notification) Act, 1889; the Contagious Diseases (Animals) Act, 1886; in Scotland only, the Customs and Inland Revenue Acts, 1890 and 1891; and in Ireland only, Part III. of the Infectious Disease (Prevention) Act, 1890, and the Public Health Acts Amendment Act, 1890, where adopted.

Scotland

Dr. Littlejohn, of Edinburgh, states that his appointment as officer of health was probably the earliest in Scotland. He was appointed in 1863, under the General Police and Improvement (Scotland) Act, 1862, which applied only to burghs, and which provided that an 'officer of health' might be appointed; but he might also be 'surveyor of paving and drainage and inspector of cleansing,' so evidently it was not necessary that he should be a medical man. He was described 'as a person of competent skill and experience, whose duty it shall be to ascertain the existence of disease within the limits appointed to him, especially of epidemic, endemic and contagious diseases, and to point out any local causes likely to occasion or continue such diseases or otherwise injure the health of the inhabitants; and to point out the best means of checking or preventing the spread of such diseases, and from time to time, as required by the commissioners, to report to them on the matters aforesaid and to perform any other duties of a like nature which may be required of him.'

Professor Gairdner was the first medical officer of health of Glasgow, and was appointed in 1863, under a local Act. Dr. Dunlop, who was an assistant under him, says that he did more for sanitary science in Scotland than any other man.

In 1867 the Public Health (Scotland) Act was passed. The 8th section empowered all local authorities named in the Act to appoint medical officers, and to make bye-laws for regulating their duties. Where it was thought necessary by the Board of Supervision for the purposes of the Act they were obliged to make such appointments. The officers had to supply reports to the Board when required.

The Local Government (Scotland) Act, 1889, has constituted county councils, having the whole powers and duties of the local authorities under the Public Health Acts of parishes so far as within the counties (excluding burghs and police burghs), and has divided the counties into districts with district committees, for the purposes, amongst other things, of the administration of the laws relating to public health.

Those committees, subject to the provisions of the Act, are now the local authorities under the Public Health Acts, and may exercise within their districts all the powers and duties transferred by the Act to, or conferred on, the county councils with respect to the administration of the laws relating to public health, except those relating to medical officers or sanitary inspectors for the counties. Medical officers of counties or districts may appeal from any proceedings or order of district committees to the county councils, who may on such appeal make an order under the Public Health Acts.

The power of appointing officers under the Public Health Acts is varied, so that it is now lawful to appoint them either for whole districts or any part thereof or parishes therein as shall be deemed expedient.

Section 52 requires the council of every county to appoint and pay a

medical officer or medical officers, who shall not hold any other appointment or engage in private practice or employment without express written consent of the council. The county council and any district committee, as the local authority under the Public Health Acts, may from time to time make and carry into effect arrangements for rendering the services of such officer or officers regularly available in the district of the district committee, on such terms as to the contribution by the district committee to the salary of any medical officer, or otherwise, as may be agreed, and the medical officer shall then have within such district all the powers and duties of a medical officer appointed by a district committee. So long as such an arrangement is in force the district committee need not appoint a separate medical officer.

Section 53 requires every medical officer to send to the county council of the county in which his district is situated a copy of every report of which a copy is for the time being required by the regulations of the Board of Supervision to be sent to that Board.

A county council may cause a representation to be made to the Board if they consider that the Public Health Acts have not been properly put in force within any district, or that any other matter affecting the public health of the district requires to be remedied.

Section 54 enacts that :— '(1) No person shall hereafter be appointed the medical officer of any county or district or parish, unless he is a registered medical practitioner. (2) No person shall after January 1, 1893, be appointed the medical officer under the Public Health Acts for a county or district or parish which contained, according to the last published census for the time being, a population of 80,000 or upwards, unless he is qualified as above mentioned, and also is registered on the medical register as the holder of a diploma in sanitary science, public health, or State medicine under section 21 of the Medical Act, 1886. . . . (4) Every medical officer and every sanitary inspector appointed under this Act or under the Public Health Acts shall be removable from office only with the sanction of the Board of Supervision.'

A medical officer of any county, or of any committee under the Act, cannot be elected as a county councillor; and no county councillor or his partner can be appointed as such medical officer; this disqualification applies to any person and his partner during six months next after ceasing to be a county councillor.

In 1890 the Board of Supervision made Regulations and Bye-Laws for medical officers appointed under the Public Health Act and the Local Government Act, which are here quoted. The Regulations, marked No. I., are *obligatory*; they apply to every medical officer of a district of a county, and take effect without any minute or resolution of the district committee. The Bye-Laws may be regarded as model sets of bye-laws authorised by section 8 of the Public Health (Scotland) Act, 1867. Those marked No. II. are applicable to medical officers appointed by county councils under the Local Government (Scotland) Act, 1889; and those marked No. III. are applicable to medical officers of local authorities generally. They are not obligatory, and do not come into force until they have been adopted by the county councils or the local authorities, as the case may be, and approved by the Board.

No. I.—Regulations under § 53 (1) of Local Government Act.

LOCAL GOVERNMENT (SCOTLAND) ACT, 1889, SECTION 53 (1).

*Medical Officers of Districts of Counties.**Regulations of the Board of Supervision.*

By virtue of the authority conferred by the foregoing enactment, the Board of Supervision hereby make the following Regulations, and require every medical officer under the Public Health Acts for a district in any county in Scotland to conform thereto.

I. Every such medical officer shall annually prepare a report with regard to his district for the year ending December 31, which report shall contain :

1. A general account of the sanitary state of the district, and the measures which in his opinion should be adopted for its improvement.
2. A statement of the general inquiries he has made during the year, and of any special inquiries as to sanitary matters.
3. A general statement of any matters as to which he has given advice or granted certificates, including any action as to offensive trades and the sanitary condition of factories and workshops.
4. An account of his inspections of the bakehouses in the district, and of any proceedings taken with regard to them.
5. An account of the supervision exercised by him over the hospitals belonging to the local authority, or to which the local authority are entitled to send patients.
6. A summary of the action taken to prevent the outbreak and spread of infectious disease.
7. A statement as to the causes, origin, and distribution of diseases within the district, and the extent to which the same have depended on or been influenced by conditions capable of removal or mitigation.
8. A tabular statement (in such form as the Board of Supervision may from time to time direct) of the sickness and mortality within the district.

II. He shall transmit a copy of the aforesaid report to the Board of Supervision and the county council not later than January 31 immediately following the year to which such report refers.

III. When in his opinion any disease of an infectious or contagious kind threatens to become dangerous or epidemic within the district, he shall forthwith report the same to the Board of Supervision and to the county council, stating the extent of the outbreak, its supposed origin, and the measures adopted for the prevention of the spread of the disease, and for the isolation and treatment of those affected.

IV. He shall report to the Board of Supervision and to the county council every case of small-pox in the district as soon as it comes to his knowledge.

No. II.—Bye-Laws under the Public Health and Local Government (Scotland) Acts.

RECOMMENDED BY THE BOARD OF SUPERVISION FOR REGULATING THE DUTIES OF MEDICAL OFFICERS OF COUNTIES.

1. The medical officer shall by inspection of the county, and by such other means as are at his disposal, keep himself informed respecting all influences affecting or threatening to affect injuriously the public health within the county.

2. The medical officer shall inquire into and ascertain, as far as practicable, the causes, origin and distribution of diseases within the county; and ascertain to what extent the same have resulted from or may depend on insanitary conditions capable of removal or mitigation.

3. The medical officer shall advise the county council with respect to all matters affecting the health and sanitary condition of the county, and the execution of the Public Health Acts therein.

4. The medical officer shall advise the county council with regard to the carrying out of the Sale of Food and Drugs Act, the Rivers Pollution Prevention Act, and any other Acts in regard to which his advice may be required.

5. The medical officer shall, when applied to by the sanitary inspector of the county

or by the district committee, or the medical officer, or sanitary inspector of any district in the county, give advice and render assistance in any matter connected with the public health.

6. On receiving information that any disease has become or threatens to become epidemic in the county or in any part thereof, the medical officer shall co-operate with the district committee and their medical officer in taking measures for its prevention or mitigation.

7. The medical officer shall make such reports and returns as may be called for by the county council or the Board of Supervision; and shall observe and execute all lawful orders and instructions of the county council or the Board of Supervision applicable to his office.

8. The medical officer shall report to the county council whenever it appears to him that the Public Health Acts have not been properly put in force within any district, or that any other matter affecting the public health of the district requires to be remedied, in order that the county council may be enabled to decide whether a representation should be made to the Board of Supervision on the matter in terms of § 53 (2) of the Local Government Act.

9. The medical officer shall report to the county council as to the advisability of making bye-laws for the prevention and suppression of nuisances not already punishable in a summary manner, in terms of § 57 of the Local Government Act.

10. The medical officer shall keep a journal, in a form to be approved by the county council, in which he shall enter his visits, inspections, and other proceedings, with notes of his observations, and of any instructions he may give. He shall submit his journal to every meeting of the county council, and shall produce it when required by the Board of Supervision or their inspecting officer.

11. The medical officer shall annually prepare a report for the year ending December 31, which report shall contain:

- (a) A general account of the sanitary state of the county, and the measures which in his opinion should be adopted for its improvement.
- (b) A statement of his inquiries and proceedings, and of the matters in regard to which he has given advice or taken action during the year.
- (c) A statement of the causes, origin, and distribution of diseases in the county, and the extent to which the same have depended on or been influenced by conditions capable of removal or mitigation.
- (d) A summary of the action taken to prevent the outbreak and spread of infectious disease, and an account of the hospitals or other means of isolation existing within the county.
- (e) A tabular statement of the sickness and mortality within the county, embodying the information contained in the reports which the medical officers of districts or parts of districts are required to send to the county council.

He shall transmit a copy of such report to the Board of Supervision and the county council not later than the 31st day of March of the year immediately following that to which the report refers.

No. III.—Amended Bye-Laws under § 8 of the Public Health (Scotland) Act, 1867.

RECOMMENDED BY THE BOARD OF SUPERVISION FOR REGULATING THE DUTIES OF MEDICAL OFFICERS (OTHER THAN MEDICAL OFFICERS OF COUNTIES).

1. The medical officer shall inform himself, as far as practicable, respecting all influences affecting or threatening to affect injuriously the public health within the district of which he is medical officer.

2. The medical officer shall inquire into and ascertain, by such means as are at his disposal, the causes, origin and distribution of diseases within the district; and ascertain to what extent the same have resulted from or may depend on insanitary conditions capable of removal or mitigation.

3. The medical officer shall, by inspection of the district, both systematically at certain periods, and at intervals as occasion may require, keep himself informed of the conditions injurious to health existing therein.

4. The medical officer shall be prepared to advise the local authority on all matters affecting the health of the district, and on all sanitary points involved in the action of the

local authority; and, in cases requiring it, he shall certify, for the guidance of the local authority, or of the sheriff, or any magistrate or justice, as to any matter in respect of which the certificate of a medical officer or a medical practitioner is required as the basis or in aid of sanitary action.

5. On receiving information of the occurrence of any contagious, infectious, or epidemic disease within the district, the medical officer shall visit the spot without delay, and inquire into the causes and circumstances thereof, and advise the persons competent to act as to the measures which may appear to him to be required to prevent the extension of the disease, and, so far as he may be able, assist in the execution of the same.

6. On receiving information from the sanitary inspector that his intervention is required in consequence of the existence of any nuisance injurious to health, or of any overcrowding in a house, the medical officer shall, as early as practicable, take such steps authorised by the statute in that behalf as the circumstances of the case may justify and require.

7. The medical officer shall inquire into any offensive process of trade carried on within the district, and report on the appropriate means for the prevention of any nuisance or injury to health therefrom.

8. The medical officer shall make the necessary inspections and otherwise perform the duties devolving on him under the Factory and Workshop Act, 1883, in regard to bake-houses.

9. The medical officer shall from time to time, and once at least in every year, visit and inspect every hospital belonging to the local authority or to which the local authority are entitled to send patients, and shall forthwith report to the local authority any circumstance which may impair the fitness of such hospital for the reception and treatment of infectious disease.

10. The medical officer shall perform all the duties imposed upon him by any Bye-Laws and Regulations of the local authority duly approved in respect of any matter affecting the public health, and he shall further observe and execute, so far as the circumstances of the district may require, the instructions of the Board of Supervision and all the lawful orders and directions of the local authority applicable to his office.

11. The medical officer shall attend at the office of the local authority, or at some other appointed place, at such stated times as they may direct.

12. The medical officer shall keep a book or books, to be provided by the local authority, in which he shall make an entry of his visits, and notes of his observations and instructions thereon, and also the date and nature of applications made to him, the date and result of the action taken thereon, and of any action taken on previous reports; and shall produce such book or books whenever required to the local authority, the Board of Supervision, and the inspecting officer of the Board.

13. The medical officer shall from time to time make such special reports and returns as may be called for by the local authority or the Board of Supervision.

14. The medical officer shall at all reasonable times afford assistance and furnish information to the county council and the officers appointed by them; and, when he deems it necessary, or when so instructed by the local authority, he shall take the advice of the medical officer of the county.

[The above Bye-Laws, with the necessary verbal alterations, may be adopted by the local authorities of burghs.]

The Board of Supervision state in their 47th Annual Report that it was their intention that the reports to be furnished by medical officers should comprise mortality tables arranged under the heads of various classes of diseases; but that a number of officers were unable to furnish them with that information, owing to the refusal of some of the registrars to supply extracts from their books, save at a cost that the local authorities were unwilling to incur. In Scotland there is no provision for the supply of those extracts such as exists in England, and the returns desired by the medical officers can only be obtained by private arrangement with the registrars. The Board express the opinion that, failing agreement, there seems no resource but legislation, whereby Scotland would be placed in the same position as England in that respect.

In their 46th Annual Report the Board of Supervision say that the

services of a large number of medical officers and sanitary inspectors of parochial boards have been dispensed with, giving local authorities freedom to establish a suitable and efficient sanitary organisation in their several districts.

The powers and duties of the medical officer under the Public Health (Scotland) Act, 1867, are neither numerous nor well-defined.

The description of nuisances in the 16th section is more comprehensive than in the English Acts ; but the medical officer has no power of entry to any premises for inspection under the 17th section, unless the local authority or sanitary inspector have reasonable grounds for believing that a nuisance exists, and demand admission for him between 9 A.M. and 6 P.M. or at any hour when the operations suspected to cause the nuisance are in progress or are usually carried on ; and they can apply for a magistrate's order for his admission if it is refused. Under the 18th section he may give a written certificate of a nuisance to a local authority, who may take proceedings for its abatement. Under the 26th section, the sanitary inspector, but not the medical officer, has power of entry to inspect any meat or other specified articles of food, and to deal with any that are unfit for human food. When an Order for prevention of disease by the Privy Council is in force in any place in his district he may give a certificate of overcrowding in any house to the local authority, so that it may be brought under the common lodging-houses provisions. He is not mentioned in sections 40, 42, 43, 50 or 66, but being a legally qualified practitioner he may give certificates to the local authority for the cleansing and disinfecting of infected houses, &c. ; for the removal of persons sick of infectious diseases without proper lodging, or on board any ship ; or for a magistrate's order for the removal of dead bodies, or for the disinfection to his satisfaction of any infected house ; or for the removal to a hospital of any person ill with infectious disease in a common lodging-house. Under section 51 he might give notice for the periodical removal of manure from mews, &c. He has to receive notice, under the 67th section, of all cases of infectious disease occurring in common lodging-houses, and must forthwith visit and report on them. Under section 68 the keepers of common lodging-houses must give him free access into them at all times. It will probably be part of his duty to assist the local authority in the inspection of their district required by the 99th section. Under section 102 he might appear for the local authority in legal proceedings. Section 118 requires that when he is acting in the *bona-fide* execution of the Act the local authority shall indemnify him in respect of all costs, liabilities and charges to which he may be subjected.

Under the Privy Council Order of July 12, 1883, as to cholera, which is still in force, the medical officer of health of any district in which there is a port or harbour may have important duties to perform with respect to ships infected, or suspected to be infected, with cholera ; but as they are so nearly allied to those imposed by the English Cholera Regulations, which have been fully described, they need not be given in full.

That officer might have to do with the destruction of rags brought from cholera-infected places, as required by such Directions and Regulations of the Board of Supervision as they issued on July 15, 1892, with regard to the importation of rags from France, ports on the Black Sea, &c. He might also be concerned with Directions and Regulations of the Board under Part III. of the Public Health (Scotland) Act, 1867, like those they issued on July 21, 1892, requiring all local authorities to remove nuisances, cleanse dwellings, check overcrowding, attend to the purity of water-supplies, provide hospital accommodation, &c.

There is no power of forming combined port sanitary authorities in Scotland, so that the sanitary jurisdiction over rivers may be divided between several local authorities working independently of one another; and even Glasgow is dependent upon the vigilance of an authority at the entrance to the Clyde for protection against shipborne infection.

Until the passing of the Scotch Local Government Act, the Public Health Act was to a great extent a dead letter. There are so many shortcomings in that Act that it is time there should be a consolidating and amending Public Health Act for Scotland.

In places to which the Burgh Police (Scotland) Act, 1892, applies some advances have been made; and there the commissioners are obliged to appoint a medical officer of health who must be a qualified medical practitioner, and after May 15, 1894, must be registered as a holder of a diploma in sanitary science, public health, or State medicine under section 21 of the Medical Act, 1886. He has to ascertain the existence of disease, especially infectious disease, and point out any likely local causes of it, and the best means of checking infectious diseases; and has to report to the commissioners and perform all other like duties which may be required of him, as well as those under the Public Health Acts. The commissioners have to make bye-laws to regulate his duties, and he holds his office during their pleasure, but cannot be removed from it without the sanction of the Board of Supervision. He has several powers and duties under the Act, and for the purposes of it may enter any premises at all reasonable hours of the day, but not without the consent of the occupier until after forty-eight hours' notice.

Ireland

The Public Health (Ireland) Act, 1878, divides Ireland into urban and rural sanitary districts. The 11th section constitutes the medical officers of dispensary districts medical officers of health, with an additional salary, to be approved by the Local Government Board for Ireland; and requires every urban and rural sanitary authority to appoint such other sanitary officers, including a medical superintendent officer of health, as the Board may direct, with a salary to be approved by them. That Board must assign to the medical officers of health and superintendent officers their respective duties, and functions in the discovery or inspection or removal of nuisances, in the supply of pure water, in the making or repairing of sewers and drains, or in generally aiding the administration of the sanitary laws. The Board have the same powers with regard to their qualification, appointment, duties, regulation of salary, and tenure of office as they have in the case of the medical officer of a dispensary district.

The Board have issued General Sanitary Orders to all rural sanitary authorities, and the majority of the urban sanitary authorities in Ireland, Nos. I., II., and III., dated August 8, 1879, in which the duties of the officers are set forth.

In rural sanitary districts, the relieving officers of unions and the collectors of poor rates, or any other person approved by the Board, are eligible for the office of sanitary sub-officer; every medical officer of a union, including the workhouse medical officers, and any other duly qualified medical practitioner, subject to the approval of the Board, are eligible for the office of consulting sanitary officer or medical superintendent officer of health; and clerks of unions and their assistants appointed by the guardians, or any other person who may be approved by the Board, are eligible for the office of executive sanitary officer. In urban sanitary districts consulting sanitary officers and medical superintendent officers of health must be duly qualified medical prac-

titioners ; and executive sanitary officers must be qualified as the sanitary authorities shall, with the consent of the Board, determine.

Every sanitary authority must appoint an executive sanitary officer, and so many sanitary sub-officers as they shall, with the consent of the Board, determine ; and when directed by that Board they must appoint a consulting sanitary officer or a medical superintendent officer of health.

Every officer appointed by a sanitary authority is to continue to hold office for such period as they may, with the approval of the Board, determine, or until he die, or resign, or be removed by the authority, with the assent of the Board, or by the Board.

Every sanitary sub-officer must, by inspection of his district, keep himself informed in respect of any nuisances existing in it that require abatement under the Sanitary Acts, and if he receives notice of any nuisance in the district he must visit the place and inquire into the alleged nuisance ; and when he finds any matter he thinks demands attention from the medical officer of health of the dispensary district, he must forthwith notify it to him on a prescribed form annexed to the orders ; he must submit to the sanitary authority at each weekly meeting duplicates of the reports which he has made to the medical officer during the preceding week, and must report to the authority any other matter affecting or threatening to affect injuriously the public health within his district.

Every medical officer of health who has been apprised officially by the sanitary sub-officer or otherwise becomes cognisant of any matter demanding his attention must visit the place, and if he finds such matter to involve danger to public health he must report thereon to the sanitary authority on a prescribed form, recommending a remedy to be adopted.

Every medical officer of health must inform himself respecting all influences affecting or threatening to affect injuriously the public health within his district, and must report thereon to the sanitary authority and recommend measures to be adopted for the protection or improvement of the public health.

Every consulting sanitary officer who is appointed must attend meetings of the sanitary authority whenever required to do so, and must advise them on all matters and proceedings requiring medical knowledge and advice in the administration of the sanitary laws.

Every medical superintendent officer of health when appointed must discharge all the duties imposed by the Orders on the consulting sanitary officer, and in addition to them must perform the following duties : he must report monthly to the sanitary authority on the general sanitary condition of the district, and on the discharge of their duties by the medical officers of health and sanitary sub-officers of the district.

Every medical officer of health and sanitary sub-officer must attend meetings of the sanitary authority whenever required to do so, and must assist in all proceedings in which his assistance may be required.

Every medical officer of health, and every other officer appointed under the Orders, must, in matters not specifically provided for in them, observe and execute the instructions of the Local Government Board for Ireland and all the lawful orders and directions of the sanitary authority applicable to his office.

The medical officers of health, and the consulting sanitary officer or medical superintendent officer of health, if such an officer be appointed to a sanitary district, must furnish to the Local Government Board such statistical returns of sickness and disease as shall from time to time be required from them respectively.

Every executive sanitary officer must attend the meetings of the sanitary authority ; take their directions on the sanitary business of the district, on the reports of the sanitary officers, and all proceedings arising thereon ; give instructions for the prompt and correct execution of all such orders and directions, and report on such execution, or on any neglect or failure therein which may come to his knowledge ; and must record the proceedings of the sanitary authority, and transmit a copy of such record to the Local Government Board.

The proceedings of medical officers in Ireland are ruled chiefly by the Public Health (Ireland) Act, 1878, which is so very similar to the English Act of 1875 that it would be mere repetition to enumerate all its provisions concerning them, and it must be referred to for detailed information. Section 258 enacts that every officer of a sanitary authority must attend and assist in any prosecution instituted by the authority ; but in the case of a medical officer he is entitled to remuneration from the authority at a rate approved by the Local Government Board, unless it has been agreed that this duty is to be included in his salary, or that his whole time is to be occupied in the discharge of the duties of his office. Section 266 does not empower him to sign notices under the Act.

Medical officers of urban and rural sanitary districts on the coast of Ireland may have to take action under the Cholera Regulations of the Local Government Board, dated December 6, 1890, which are similar to the English Cholera Regulations. Article 19 stipulates that every medical officer of health employed by a sanitary authority under this Order shall be paid by them such remuneration for his services as the Board may direct or approve. Those officers may also have duties to perform under Orders of the Board prescribing Regulations with reference to ships having on board rags from cholera-infected places.

In rural districts the medical officer of health may be concerned with inspecting and reporting on the sanitary condition of cottages for the purposes of the Labourers (Ireland) Acts, so long as they are continued in force.

INDEX

ABB

- Abbott, Dr., on modification of small-pox by vaccination, 445
- Actinomyces, in section through tongue of ox, plate xvii. figs. 115-17; plate xxv. figs. 150-51
- mycelium of, culture, plate xvii. fig. 118
- Actinomycosis, club-shaped bodies of, 227
- cultivation of fungus of, 228
- habitat of fungus of, 227
- pathology of, 225
- tumours and abscesses in, 227
- Acute exanthemata, diseases classified as, 188
- Age, as affecting fatality of small-pox, 897
- — — prevalence of small-pox, 896
- Age and sex, as affecting mortality, 500
- — — — influencing liability to infectious disease, 255
- Age-incidence of small-pox and vaccination, 435
- Air, examination of bacteria in, 68
- as affecting growth of bacilli, 19
- of Oxford Street, plate cultivation of, plate xxvi. fig. 2
- — Wandsworth Common, plate cultivation of, plate xxvi. fig. 1
- Arkkon, Sir W., on physique of recruits, 602
- Albuminous materials and multiplication of bacilli, 17
- 'Alexines' (germicide substances), in blood-plasma, 53
- Alkali Acts, duties of Medical Officer of Health with regard to, 778
- Ambulances for conveyance of infectious cases, 796
- America, cremation in, 717
- Amaba coli* in cases of dysentery, 186
- — — mucus of intestine, plate xli. fig. 96
- Aniline dyes, effects of, on bacteria, 12
- — — manufacture of, 29
- Animal tissues, relation of bacteria to, 46
- Animals, quantity of water requisite for, 628
- Anthrax, attenuation of bacilli of, 68, 120
- bacilli of, 110
- — — in soil, 112
- — — — method of staining, 81
- bacillus, plate cultivation of, plate xxxi. fig. 47

ATM

- Anthrax bacillus, stab-culture of, in gelatine, plate xxviii. fig. 22; plate xxxvi. figs. 78, 79; plate xxxvii. fig. 80. (*See also* 'Bacilli')
- changes in bacilli of, in cultivation, 118
- cultivation of bacilli of, 115
- degeneration of bacilli of, 117
- difficulties in protective inoculation against, 121
- infection with, by cutaneous inoculation, 112
- — — through alimentary canal and lungs, 118
- — — — flies, 112
- inoculation with blood of cattle dead of, 114
- method of distinguishing from malignant cedema, etc., 119
- mode of infection of animals by, 111
- protection against, by injection of alexines, 57
- results of protective inoculation against, 122
- spores of bacilli of, 28, 117
- symptoms and appearances of, 110
- transmitted by the mother to the foetus, 76
- Anti-compulsion, feeling of, in connection with vaccination, 455
- Anti-vaccination, allegations in support of, 455
- Arabia, spread of small-pox from, 886
- Arloing, Dr., on bacilli of *charbon symptomatique*, 109
- Army, results of re-vaccination in, 440
- statistics, 642
- Arnold, Dr., on inoculation with leprosy, 225
- Arthro-spores, question as to formation of, 26
- Ashanti expedition, malarial fevers during, 659
- — — rations issued during, 622
- Aspergillus*, species of, in animal body, 288
- *glaucus*, plate xl. figs. 104, 105
- spores, section through kidney of rabbit, after injection of, into jugular vein, plate xl. fig. 106
- Assyrians, burial customs among, 688
- Atmospheric changes as influencing mortality, 504

BAC

- BACILLI**, aerobic, of malignant cedema, plate vi. figs. 41, 42
- — — section of tissue of guinea-pig after inoculation with, plate vii. fig. 43
 - — — stab-culture of, in gelatine, plate xxviii. fig. 23
 - air as affecting growth of, 19
 - albuminous material influencing multiplication of, 17
 - alterations in forms of, with growth, 10
 - anthracis, colony of, on gelatine, plate x. figs. 66, 67
 - from blood of frog, plate x. fig. 68
 - — human malignant pustule, plate x. figs. 64, 65
 - gelatine culture of, plate iii. fig. 15
 - in blood of guinea-pig, plate ix. fig. 59
 - kidney of rabbit, plate ix. fig. 62; plate xx. fig. 131
 - liver of guinea-pig, plate ix. fig. 63
 - spleen of guinea-pig, plate ix. fig. 61
 - — subcutaneous tissue of rabbit, plate ix. fig. 60
 - — potato culture of, plate xviii. fig. 120
 - spindle-shaped elements with vacuoles shown, plate iii. figs. 16, 17
 - threads of, from gelatine culture, plate x. fig. 70
 - with spores, plate x. fig. 69
 - attenuation of powers of, 67
 - Brownian molecular movements of, 14
 - chain formation by, 11
 - cilia or flagella of, 15
 - comma, 175
 - conditions and mode of multiplication of, 16
 - development of, from spores, 27
 - differentiation of species of, 28
 - distinguishable from cocci, 11
 - effects of aniline dyes upon, 12
 - formation of pellicle by, 16
 - forms of, resembling cocci, 10
 - from beef-pie that had caused diarrhoea, plate xv. fig. 99
 - larynx, in case of diphtheritic croup, plate xxii. figs. 132, 135
 - importance of presence or absence of spores of, 24
 - in croupous pneumonia, plate v. figs. 32, 85
 - liver of Rhea, plate xxiv. figs. 148, 149
 - Malpighian corpuscle in case of meat-poisoning, plate xxv. fig. 153
 - method of staining spores of, 31
 - modes of division of, 21
 - moisture as affecting growth of, 18
 - motility of, 14
 - multiplication of, 16
 - of acute mouse septicæmia in liver of mouse, plate xxi. fig. 127
 - — lung of mouse, plate xx. fig. 128; plate xxi. fig. 129
 - — anthrax, 110
 - — spore-formation by, 23
 - — staining of, 31
 - — catarrhal ophthalmia, plate xiv. figs. 97, 98; plate xviii. fig. 123

BAC

- Bacilli of charbon symptomatique**, 108
- — — in blood of guinea-pig, plate xi. fig. 71
 - — cholera, cultures, plate xiii. figs. 87, 89-91; plate xiv. fig. 92
 - — in mucus-flakes, plate xiii. fig. 88. (See also 'Cholera' and 'Comma')
 - — chronic necrotic deposit in membrane of fauces of fowl, plate xxviii. fig. 88
 - — diphtheria, 155; plate xi. figs. 72-76
 - — from corneal ulcer of inoculated cat, plate xii. fig. 78
 - — milk of infected cow, plate xii. fig. 79
 - — mucous membrane of pharynx of child, plate xix. fig. 133
 - — necrotic tumour of inoculated cow, plate xi. fig. 77; plate xii. fig. 82
 - — udder of cow inoculated on shoulder, plate xxi. fig. 130
 - — in kidney of cat, plate xix. figs. 187, 138
 - — membrane of tonsil of child, plate xxii. fig. 134
 - — fowl-cholera, 103; plate viii. figs. 53-55
 - — rapidity of growth of, 21
 - — fowl-enteritis, 105; plate viii. figs. 50-52
 - — glanders, plate xiv. figs. 100, 101; plate xxii. fig. 139; plate xxiii. fig. 140
 - — grouse-disease, 105
 - — among blood-corpuscles, plate vii. fig. 47
 - — from infected mouse, plate ii. fig. 8. (See also 'Grouse-disease')
 - — human septicæmia, 101
 - — influenza, 130
 - — leprosy, plate xvii. figs. 113, 114; plate xxiv. figs. 145-47
 - — malignant cedema, 99
 - — cedema fluid, spore formation in, culture in agar, plate vii. fig. 46. (See also 'Malignant Cedema')
 - — pneumonia (FRIEDLÄNDER), 124
 - — poisonous foods, 73
 - — pseudo-diphtheria, culture, plate xii. fig. 80
 - — rag-sorters' disease, 101
 - — septicæmia in mice, 100
 - — — rabbits, 97
 - — swine erysipelas and fever, 106
 - — — in blood of pigeon, plate ix. fig. 57
 - — — liver of pigeon, plate ix. fig. 58
 - — — kidney of pigeon, plate xxi. fig. 180
 - — fever, in spleen of mouse, plate viii. fig. 56
 - — tetanus, plate xv. fig. 103
 - — cultivation of, in grape sugar gelatine, plate xxix. fig. 87
 - — tubercle, 217
 - — — cultivation of, 219
 - — — ecogenic character of, 222
 - — — forms of, 218
 - — — method of staining, 31, 218

BAC

- Bacilli of tubercle*, plate xv. figs. 108-5;
 plate xv. figs. 106, 107
 — in liver of rabbit, plate xvi. fig. 110
 — — lung of cow, plate xvi. figs. 108, 112; plate xxiv. fig. 144
 — of rabbit, plate xvi. fig. 109;
 plate xxiii. fig. 141
 — — spleen of fowl, plate xvi. fig. 111; plate xxiii. figs. 142, 143
 — — prolonged vitality of, 222
 — — question as to spores of, 26
 — — spores of, 221
 — — surface cultivation of, on glycerine agar, plate xxv. figs. 68, 69
 — — typhoid, cultivated on glycerine, plate xiii. fig. 85
 — — EBERTH-GAFFKY, colonies of, on gelatine, plate xxxiii. fig. 59
 — — gelatine cultivation of, showing flagella, plate iii. fig. 20
 — — streak-culture of, plate xxvii. fig. 9
 — oxygen necessary for motility of, 16
 — proteid medium for growth of, 17
 — protoplasm of, 5, 17
 — putrefactive, spores of, 28
 — rapidity of growth of, 19
 — salts necessary for growth of, 17
 — shape of, 7, 10
 — signs of spore-formation by, 24
 — species of, in mouth and pharynx, 46
 — spores of, 22
 — staining of, with aniline dyes, 12, 29
 — 'swarming' of, 15
 — temperature as affecting growth of, 18
 — vacuoles in, 12
 — variations in modes of division of, 21
 — with blood, in case of influenza, plate xiv. fig. 95
Bacillus, capsulated, of FRIEDLÄNDER, in pneumonic sputum, plate vi. fig. 87
 — filamentous, culture, plate iii. fig. 19
 — from gelatine culture of sewage, plate ii. fig. 14
 — — with spores, plate xviii. fig. 119
 — fluorescens liquescens, stab-culture of, in gelatine, plate xxvii. fig. 20; plate xxxvi. fig. 74
 — mesentericus, plate-culture on gelatine, plate ii. fig. 13
 — minute, from dust of room, plate iii. fig. 18
 — of pneumonia, in blood of mouse, plate vi. fig. 86
 — oval, from veal-pie which had caused choleraic diarrhoea, plate i. fig. 7
 — prodigiosus, culture of, on potato, plate xxxviii. fig. 89
 — — streak-culture of, on agar, plate xxxiv. fig. 67
 — proteus-like, from pork-pie which had caused choleraic diarrhoea, plate ii. fig. 9
 — pyocyaneus, 89
 — — culture, plate iv. fig. 28
 — — foetidus, 89
 — — stab-culture of, in gelatine, plate xxxv. fig. 73
 — — streak-culture of, on agar, plate xxxiv. fig. 64

BAC

- Bacteria*, acidity of medium as affecting growth of, 45
 — action of blood-plasma upon, 49
 — action of, increased by growth of other species, 60
 — — on leucocytes, 54
 — affinity of protoplasm of, for aniline dyes, 6
 — animal tissues as a soil for, 46
 — antagonism amongst, 58
 — attenuation of power of, 68
 — attraction for, of cells and tissues, 55
 — biological characters of, 41
 — causes of attenuation of function of, 70
 — characters of, 5
 — chemical function of, 64
 — — products of, as causing disease, 71
 — chemiotactic action of, 54
 — — substances upon, 54
 — classification of, 8, 42
 — cultivation of, in artificial media, 35
 — — in blood-serum, 40
 — — in nutrient agar, 40
 — — on potato, 40
 — — destruction of, by chemical substances, 81
 — — — different forms of, 59
 — — — drying, 81
 — — — heat, 80
 — — — normal lymph and blood, 49, 51
 — — — white corpuscles, 50
 — differences between, in plate-cultivation, 39
 — ecogenic and endogenic, 75
 — effects of boiling water upon spores of, 23
 — — gastric juice upon, 77
 — — inhibited by blood plasma, 53
 — examination of, in fresh state, 28
 — — — suspended drop, 28
 — fermentative action of, 65
 — formation of spores or seeds by, 22
 — general morphology of, 5
 — growth of, checked by carbolic acid and mercury perchloride, 79
 — heat as influencing growth and life of, 48
 — — — restraining growth of, 79
 — — 'impression' preparations of, 40
 — in air, examination of, 63
 — — pus, species of, 90
 — — sections of tissue, methods of staining, 82
 — — soil and water, examination of, 62
 — incubation period after invasion of, 74
 — infective processes caused by, 74
 — influences restraining growth and multiplication of, 79
 — insusceptibility to action of, 50
 — light as influencing growth and life of, 48, 79
 — liquefaction of gelatine by, 86
 — loss of pathogenic power of, 67
 — manner of infection by, 74
 — modification of effects of, in various tissues, 71
 — moisture as restraining growth of, 79
 — multiplication and movements of, in body after death, 48
 — mutual antagonism of various species of, 59

BAC

- Bacteria, nitrifying power of, 15
 — non-specific, resistance of normal tissue to, 49
 — number of, as influencing severity of disease, 74
 — of suppuration, 84
 — passage of, through maternal circulation, 77
 — — — through mucous membranes, 76
 — — — walls of intestine and bronchia, 77
 — pathogenic action of, 66
 — — groups of, 66
 — phosphorescent, 44
 — pigment-forming species of, 44, 65
 — plate-cultivation of, 87
 — presence of, in epidermis, 47
 — — — mucous membranes, 47
 — — — ulcers, 48
 — production of poisons by, 65
 — — — ptomaines by, 72
 — protoplasm of, 5
 — putrid decomposition of albumen by, 66
 — reaction of media suitable for growth of, 44
 — relation of animal tissues to, 46
 — — — to purulent processes, 90
 — roll-plate cultivation of, 39
 — saprophytic, invasion of diseased tissue by, 48
 — secondary access of, to diseased parts, 48
 — shape of, 8
 — size of, 7
 — soil as restraining growth of, 79
 — species of, found in mouth and pharynx, 46
 — — — — nose and lungs, 47
 — specific or pathogenic, 83
 — spores of, with reference to infectious diseases, 23
 — stab-culture of, 86
 — staining of, 29
 — — — by GRAM's method, 38
 — — — — Warsova method, 32
 — — — — WEIGERT's dyes, 30
 — streak-culture of, 37
 — study of biological characters of, 34
 — susceptibility to, increased by fatigue, 61
 — tube-plate cultivation of, 38
 — variations in power of, over different animals, 67
 — — — size of, due to character of medium, 8
 — — — — due to phases of growth, 7
 — virulence of, modified by growth of other species, 59
 BADCOCK, Mr., on inoculation of cows with small-pox virus, 423
 Bake-houses, inspection of, by Medical Officer of Health, 753
 BALLARD, Dr., on choleraic diarrhoea due to food, 189
 — — — epidemic diarrhoea, 339
 — — — infantile diarrhoea, 187
 — — — microbes of pneumonia, 127
 Barracks, cavalry, 606
 — construction of, 604
 — cooling of, 608

BUR

- Barracks for married soldiers, 606
 — in hot climates, 607
 — Indian Army Regulations for, 608
 — sanitary arrangements in, 608
 — sites for, 608
 — size of, 604
 — ventilation of, 606
 — water-supply of, 607, 627
 BARTOWS or mounds, burials in, 677
 BARRY, Dr., on age-incidence of small-pox in Sheffield, 439
 BASTIAN, Dr., on evolution and origin of life, 245
 BAUMGARTEN on heredity of tuberculosis, 215
 Birth-rate, statistics of, 493
Blastomycetes or *Torula*, 235
 — — — alcohol produced by species of, 235
 — — — thrush produced by species of, 235
 BLAXALL, Dr., on epidemic of typhoid at Sherborne, 321
 — — — inspection of ships from foreign ports, 584
 Blood, caution as to transference of, in vaccination, 428
 Blood-plasma, germicidal substances in, 58
 Blood-serum, cultivation of bacteria in, 49
 — germicidal powers of, 49
 Boiling, as a means of purifying water, 629
 'Bolagh' and 'galar breac,' Irish names for small-pox, 387
 BOLLINGER on *actinomycosis*, 225
 — — loss of infectivity after dilution of milk from tuberculous cows, 214
 BOUSQUET, M., on cow-pox as a source of vaccine lymph, 421
 BOYLE's system of ventilating ships, 540
 BRASHEY, Lord, on alleged deterioration of British seamen, 557
 BRETONNEAU on diphtheria, 294
 Bronze, sepulture in age of, 678
 Broth, cultivation of bacteria in, 35
 BRUCE, Dr., on Malta fever, 171
 BRYDEN, Dr., on susceptibility to cholera of newly arrived troops, 649
 BUCHANAN, Sir G., on epidemic of small-pox in Sheffield, 451
 — — — risk to neighbourhood from small-pox hospitals, 795
 — — — — typhoid at Caius College, 322
 BUCHNER on changes in bacillus anthracis by successive cultivations, 119
 — — germicidal power of blood-plasma and blood-serum, 49
 Buildings, necessity for space around, 761
 BURDETT, Col., system of messing for soldiers, 620
 Burial customs among ancient Persians, 683
 — — — Assyrians, 683
 — — — Christians in Rome, 690
 — — — Jews, 685
 — — — the Egyptians, 681
 — — — Romans, 688
 — — in Greece, 687
 — — — India, 685
 — earth-to-earth system of, 710
 — grounds, contamination of air and water by, 728

BUR

- Burial grounds, duties of Medical Officers of Health with regard to, 809
 — — — sufficiency of space in, 729
 — — — suitable soil and position for, 728
 — in earth, defence of, 720
 — intramural, dangers of, 724
 — laws regarding (1842, 1868) 695
 — modern, accumulation of bodies in, 698
 — — bad management and insufficiency of, 704
 — — conditions necessary for safety of, 705
 — — diseases caused by methods of, 700, 711
 — — evils of, 697
 — — imperfections of laws regarding, 706
 — — water-pollution resulting from, 701
 — of still-born children, laxity as to, 707
 — survival of old customs of, 674
 — urban, as contrasted with extramural, 724
 — without medical certificate, 707

CARUS COLLEGE, epidemic of typhoid at, 822
 Calf-lymph, advantages of, 480
 — use of, 429

Camps, arrangements in, 611
 — regulations and instructions for, 610
 — sites for, 609

— — — in malarious districts, 660

— water-supply for, 627

Canal boats, accommodation in, 548

— barges, &c., 534
 — inspection of, by Medical Officers of Health, 767

Cancer, presence of coccidia in, 234

— results of inoculation with, 234

Carbolic acid as a disinfectant, 88

Cargoes, infectious, on board ship, 581

CARNELLY on examination of bacteria in air, 68

CARTER, Dr. V., on spirilla of relapsing fever, 172

Caterham, epidemic of typhoid at, 823

Cats, diphtheria communicated from, to human beings, 159

— inoculation of, with diphtheritic membrane and bacilli, 159, 161

— symptoms of diphtheria in, 159

Cavalry, dress and accoutrements of, 684

Caves, burial in, 675

CEHLX, Mr., on cause of 'topical severity' of vaccination, 422

— — — inoculation of cows with variolous matter, 428, 426

— — — 'spurious' pocks, 420

Cellar dwellings, inspection of, by Medical Officer of Health, 765

Cemeteries, accumulation of bodies in, 698, 705

— allowance of space in, 729

— at Certosa, 679

— drainage of, 727, 729

— extramural, 724

— formation of, in England, 694

— in Italy, first appearance of, 679

— memorandum on sanitary requirements of, 728

— modern, difficulties of isolating, 725

OHO

Cemeteries, modern, improvements required in, 725

— soil and position of, 728

— suggestions for improved management of, 727

Centres of population in Great Britain, 467

— — — defects in statistics of, 468

Cerebro-spinal fever, cause and mode of dissemination of, 309

— fatality of, 308

— history and distribution of, 305

— influence of climate and season on spread of, 309

— — — race, age, and sex, on liability to, 309

— — — mortality from, 308

— — — recent epidemics of, 307

'Challenger,' ocean temperatures taken during voyage of, 515

CHANDLER, Mr., on inoculation for small-pox, 411

Charbon symptomatique, 108

— — — bacilli in blood of guinea-pig dead after inoculation with, plate xi. fig. 71

— — — of, 109

— — — culture of bacilli of, in grape sugar gelatine, plate xxix. fig. 38

— — — germs of, over graves of animals, 702

— — — inoculation for, 109

CHAUVEAU, M., on inoculation of cows with variolous matter, 426

Chemiotaxis, a power of certain substances over bacteria, 54

CHRYNE, W., on mutual antagonism of certain bacteria, 60

Chicken-pox, distinguished from modified small-pox, 462

— mortality of, 462

— nature and symptoms of, 461

— period of incubation of, 462

Cholera, Asiatic, aerobic and anaerobic growth of organisms of, 386

— — — a filth disease, 174

— — — among soldiers, 648

— — — artificial production of, in guinea-pigs, 182

— — — bacillus (so-called), 18

— — — bacteria in intestinal discharges in, 176

— — — cause and modes of dissemination of, 338

— — — comma-bacilli in cases of, 176

— — — conditions affecting prevalence of, 384, 649

— — — requisite for spread of, 174

— — — danger of reappearance of, in England, 338

— — — dejections of, administered to animals, 181

— — — disinfectants for *materies morbi* of, 651

— — — duties of Medical Officers of Health with regard to, 784

— — — elevation as affecting frequency of, 649

— — — facts connected with etiology of, 173

— — — growth of organism of, in soil, 385

— — — history and distribution of, 326

— — — human intercourse in communication of, 648

CHO

- Cholera, Asiatic, hypotheses (Mr. POWERS) as to, 335
- in camps and cantonments, 650
 - India the original home of, 332
 - individual predisposition to, 649
 - influence of climate and season on spread of, 333
 - — — gastric juice upon bacilli of, 182, 650
 - — — race, sex, and age on liability to, 333
 - — — introduction of, into new locality, 173
 - — — kinship of, to diarrhoea, 337
 - — — KOCH on production of, in animals, 181
 - — — KOCH's theory of causation of, 179
 - — — localisation of, 648
 - — — locality as influencing spread of, 174
 - — — mortality and fatality of, 332
 - — — on ship-board, and duties of health-officer, 585
 - — — period of incubation of, 337
 - — — phases of organism of, 335
 - — — possible spread of, by emanations from soil, 338
 - — — *post-mortem* appearances of, 174
 - — — precautions as to, at ports, 587
 - — — — against, among soldiers, 650
 - — — protection from, 387
 - — — rapidity of growth of spirilla of, 20
 - — — recent epidemics of, 328
 - — — regulations of Local Government Board as to, 784
 - — — relation of, to other diseases, 337
 - — — route of, in 1892, 331
 - — — seasonal prevalence of, 178
 - — — soil as a factor in epidemics of, 648
 - — — spirilla of, 18
 - — — — producing acidity in medium, 45
 - — — water carriage of virus of, 335, 649
 - — — bacilli of, cultures, plate xiii. figs. 87, 89-91; plate xiv. fig. 92
 - — — — in mucus-flakes, plate xiii. fig. 88
 - — — nostras (*see* 'Epidemic Diarrhoea'), 339
 - — — spirilla, KOCH's, plate-culture of, plate xxix. fig. 85
 - — — stab-cultivation in gelatine of different varieties of, plate xxxvii. figs. 81-85
 - — — varieties of, cultivated on potato, plate xxxix. figs. 92-95
 - — — variety of, belonging to series in plate xxxvii., plate xxxviii. fig. 86
 - Choleraic diarrhoea, oval bacillus from veal-pie causing, plate i. fig. 7
 - — — proteus-like bacillus from pork-pie causing, plate ii. fig. 9
 - Cilia of bacilli, method of staining, 15
 - Cladotrix dichotoma*, varying forms of, 11
 - Climate and soil, as affecting prevalence of small-pox, 394
 - Clothing, military, 630
 - Cocci from abscess of rabbit, plate iv. fig. 23
 - Coccidia*, diseases associated with, 231
 - in cancer cells, 234
 - — — *epithelioma contagiosum* of fowls, 233
 - — — tubes, MIESCHER's, 233

COW

- Coccidium oviforme* from liver of rabbit, plate xli. fig. 97
- — — in bile ducts of rabbits, 231
 - COHN, F., classification of bacteria, 8
 - COHNHEIM on specific virus of tubercle, 216
 - Comma bacilli from mucus-flakes of ape dead from diarrhoea, plate xiv. fig. 93
 - — — stab-culture of different varieties of, in gelatine, plate xxx. figs. 39-46
 - — — bacillus, DENKER's, 179
 - — — FIEBNER's, 179
 - — — cultivation in gelatine, plate iv. fig. 22
 - — — plate-cultivation of, plate xxvi. fig. 3
 - — — production of acidity in medium by, 45
 - — — stab-cultivation of, in gelatine, plate xxxv. fig. 72
 - — — KOCH's, cases of cholera without, 185
 - — — chemical products formed by, 179
 - — — degeneration of, under culture, 178
 - — — demonstration of, by cultivation, 176
 - — — destruction of, by gastric juice, 182
 - — — experiments with, 181
 - — — explanation of action of, in intestines, 184
 - — — found only in intestines, 179
 - — — in cholera evacuations, 175
 - — — production of septicæmia by injection of, 180
 - — — relation of, to Asiatic cholera, 184
 - — — results of injection of, into small intestines, 181
 - — — variations in forms of, 178
 - — — various species of, 185
 - — — spirilla from intestine of ape, after injection of sulphate of sodium into bowel, plate xiv. fig. 94
 - Communities, comparison of deaths with births in, 492
 - — — standards of comparison of health of, 491
 - Conjunctivitis, catarrhal, microbes of, 133
 - Contagia*, fixed and miasmatic, 76
 - Contagious Diseases Acts, 667
 - — — benefits arising from, 668
 - — — effects of repeal of, 668
 - — — — suspension of, in India, 669
 - Contagium vivum*, theory of, 5
 - Convection, aerial, of small-pox, 400
 - Copenhagen, epidemics of small-pox in, 395
 - COBY, Dr., on calf-lymph, 429
 - — — influence of good vaccination, 448
 - — — inoculation of cows with variolous matter, 423
 - Cotton, linen, and wool, as materials for soldiers' clothing, 631
 - Counties, Administrative, Medical Officers of Health of, 738
 - County Councils, appointment of Medical Officers of Health by, 735
 - Courts and alleys, sanitary defects in, 762
 - Cow-pox as a source of vaccine-lymph, inquiries regarding, 421
 - — — false and true, 140

COW

- Cow-pox, lymph, experiments with, by JENNER, PEARSON, and WOODVILLE, 418
 — relation of, to horse-pox, 417
 — spontaneous, appearances in, 189
 — 'spurious,' JENNER's cases of, 420
 Cows at Hendon, cutaneous and visceral diseases in, 148
 — diseased, milk of, causing scarlet fever, 148
 — diseases among, at Edinburgh, 153
 — of, and transmission of scarlet fever, 150
 — examination of eruptions of, 148
 — question as to nature of eruptions on udders of, 151
 — results of inoculation of, with variolous matter, 423
 — *streptococci scarlatina* found in ulcers of, 152
 COXWELL, Dr., on susceptibility of frogs and rats to anthrax after chloroform narcosis, 58
 CREIGHTON, Dr., on syphilis and vaccination, 458
 Cremation, absence of religious objections to, 718
 — alleged annihilation of exhumation evidence by, 718
 — apparatus for, in France, 718
 — arrangements for, at Woking, 716
 — as a mode of disposal of the dead, 712
 — conditions under which practised at Woking, 715
 — construction of apparatus for, 714
 — in foreign countries, 716
 — the Colonies, 718
 — not forbidden by English law, 714
 — objections to, 720
 — revival of, in England, 718
 — Society for Promotion of, 718
 CROOKSHANK, Dr., on eruptive diseases in cows, 141
 — — — variolation of cows, 423
 Croup, mortality from, 367
 — nature of, 366, 368
 — relation of, to diphtheria, 366
 — seasonal mortality from, 368
 CULLINGWORTH, Dr., on decline in mortality from puerperal fever, 359
 Cultivation of bacteria in artificial media, 35
 CUNNINGHAM, Dr., on cases of cholera without comma bacilli, 185
 — — — production of septicæmia by injections of comma bacilli, 180
 DAIRIES, cow-sheds, and milk-shops, duties of Medical Officers of Health with regard to, 810
 DARRISHIRE, Dr., on epidemic of scarlet fever at Oxford, 147
 DAVAINNE on septicæmia in rabbits, 97
 Dead bodies, decomposition of, in earth, 721
 — — — vaults, 722
 — — — desiccation of, 711
 — — — pit burial of, 723
 — — — removal and re-interment of, 722
 — — — cave-burial of the, 675

DIP

- Dead, disposal of the, 674
 — — — apathy of public with regard to, 674
 — — — by cremation, 712
 — — — in Great Britain, 691
 — — — otherwise than by burial, 709
 — sepulture of, in Bronze Age, 678
 — — — Iron Age, 679
 — — — Stone Age, 675. (*See also* 'Burial,' 'Cremation' and 'Graveyards')
 Death and disease, distribution of, 498
 — mean age at, 490
 — regulations in foreign countries for certificates of, 707
 — rate, infantile, statistics of, 494
 — — zymotic, statistics of, 498
 Death-rates of males in various occupations, 507
 — sources of fallacy in, 495
 Deaths, defective registration of, in England, 709
 — in England, Lowe's returns of, 476
 Dental caries, microbes occurring in, 92
 Desiccation of dead bodies, 711
 Diarrhœa, choleraic, due to toxic effects of food, 189
 — — — microbes connected with, and morbid appearances in, 189
 — — — infantile, bacteria connected with, 188
 — — — causation and pathology of, 187
 — — — kinship of, to cholera, 337
 — — — mucus-flakes and comma bacilli of ape dead from, plate xiv. fig. 98
 DRMEDALE, Baron, inoculation as practised by, 411
 Diphtheria, average death-rates of, plate xliii.
 — — — bacilli of, plate xi. figs. 71-76
 — — — colonies of, on agar, plate xxxiii. fig. 55
 — — — — — gelatine, plate xxxii. figs. 52, 53
 — — — — — surface of agar, plate xxxii. fig. 56
 — — — — — gelatine, plate xxviii. fig. 29
 — — — from corneal ulcer of inoculated cat, plate xii. fig. 78
 — — — — — milk of infected cow, plate xii. fig. 79
 — — — — — necrotic tumour of inoculated cow, plate xi. figs. 77, 82
 — — — in kidney of cat, plate xix. figs. 187, 188
 — — — — — section of larynx of cat, plate xii. fig. 84
 — — — — — child, plate xxii. figs. 182, 185
 — — — — — lung of cat, plate xii. fig. 83
 — — — — — through membrane of tonsil of child, plate xxii. fig. 184
 — — — — — mucous membrane of pharynx of child, plate xix. fig. 183
 — — — — — udder of cow, plate xxi. fig. 180
 — — — — — stab-culture of, in gelatine, plate xxix. figs. 83, 84
 — — — — — streak-cultures of, plate xxviii. fig. 27; plate xxxiv. fig. 66

DIP

- Diphtheria, bacilli of, tube-plate cultivation of, plate xxviii. fig. 28
- bacillus of, 155
- cause of, 299
- chemical poisoning in, 157
- climate and season as affecting prevalence of, 297
- communication of, from cats to human beings, 159
- — — from cows through milk, 162, 302
- contagious nature of, 161
- cultivation of bacillus of, 156
- distribution of, compared with that of scarlet fever, 296
- epidemics of, due to milk contamination, 161, 301
- fatality of, 297
- history of, 298
- human, distinguishable from similar disease in pigeons, 164
- in animals, evidence as to, 158, 302
- increased frequency of, 295, 296
- influence of race, sex, and age on liability to, 298
- inoculation of cows with bacillus of, 161
- — with cultures of bacillus of, 156
- minor throat illnesses with epidemics of, 300
- modes of dissemination of, 301
- mortality of, 297
- not necessarily connected with defective drainage, 303
- passage of virus of, through cow to milk, 163
- periodicity of, 296
- periods of incubation and infectiveness of, 304
- prevalence of, in England, 295
- protection from, 304
- relation of, to other diseases, 303
- school attendance and spread of, 300
- species of bacilli found in, 155
- symptoms and appearances of, 154
- — of, in cats, 158
- Diplococci, forms of, 8
- Diplococcus pneumoniae*, 126
- in various acute infective diseases, 129
- Diseases among soldiers, prevention of, 647
- connection between, and atmospheric changes, 504
- nomenclature and classification of, 475
- on shipboard, prevention of, 583
- Disinfection, 78
- chemical, 81
- of cholera dejecta, &c., 651
- of houses, to check infectious diseases, 782
- of typhoid dejecta, &c., 655
- Dissemination of small-pox, 400
- Dolmens, burial in, 677
- Drainage arrangements in houses, examination of, by Medical Officer of Health, 775
- Duck cholera, bacilli of, 104
- DUNNIE, Mr., on effects of a second inoculation, 415
- Dust in factories, &c., as a cause of phthisis, 364

ERY

- Dust of room, minute bacilli from, plate iii. fig. 18
- Dysentery, *amaba coli* in, 186
- appearances in, 186
- bacteria connected with, 187
- and diarrhoea among troops, 663
- — — — causes of, 664
- — — — changes of temperature as producing, 665
- — — — protective measures against, 664
- — — — relation of, to malaria, 664
- EARTH-TO-EARTH system of burial, dangers of, 710
- EBERTH-GAFFKY, bacillus of typhoid, 168
- Ectogenic and endogenic bacteria, 75
- Edinburgh disease (among cows), streak-culture of streptococcus of, plate xxvii. fig. 12
- eruptive disease among cows at, 153
- EDWARDS, Dr., on deaths from chicken-pox, 463
- Egyptians, burial customs among early, 681
- Embalming, art of, in Egypt, 682
- Endospores, formation of, 25
- England and Wales, measles mortality in, 260
- — — practice of inoculation in, 410
- — — small-pox, death-rate per million in, 393
- — — in, during Middle Ages, 388
- English cholera (see 'Epidemic Diarrhoea'), 339
- Epidemic diarrhoea, age and sex as affecting incidence of, 342
- — cause and dissemination of, 343
- — conditions of locality favouring frequency of, 344
- — — spread of, relating to population, 345
- — general conditions favouring frequency of, 343
- — leading phenomena of, 340
- — mortality from, 341
- — nature of, 338
- — period of incubation of, 346
- — relation of, to other diseases, 346
- — season as affecting mortality from, 343
- roseola (see 'Rötheln'), 263
- Epidemics of small-pox, periodicity of, 395
- Epidermis, bacteria present in, 47
- ERICHSEN, Mr., on hospital erysipelas, 353
- Erysipelas, cause and mode of dissemination of, 352
- history and geographical distribution of, 350
- micrococci of, 98
- mortality from, 351
- period of incubation of, 357
- produced by vaccine lymph, 144
- race, sex, and age as influencing liability to, 352
- relation of, to other diseases, 354
- — — — puerperal fever, 355
- resulting from vaccination, 460

ERY

- Erysipelas, scarlatina, and other diseases, death-rates from, with rain at Greenwich, plate xlv.
 — season as influencing spread of, 351
 — spread of, in hospitals, 353
 ESTLIN, Mr., on cow-pox as a source of vaccine lymph, 421
 Europe, small-pox in, during Middle Ages, 386
 Evolution, doctrine of, and theory of spontaneous generation, 245
 Exhumation, rarely necessary for legal purposes, 713
- Factories and workshops, inspection of, by Medical Officers of Health, 755
 Fallacies in statistics, sources of, 485
 Faroe Islands, outbreak of measles in, 263
 FARR, Dr., on causes of high mortality in towns, 504
 — on fallacies in Malthusian theory, 472
 — on inaccurate statistics, 469
 Fatality of small-pox in new countries, 397
 FEHLEISEN on micrococci of erysipelas, 93
 Fever, intermittent, appearances in, 190
 — etiology of, 191
 — *plasmodium malarie* as causes of, 191
 Fever hospitals (other than small-pox), absence of risk to neighbourhood from, 793
 Filters, forms and use of, 629
 FINKLER's comma bacillus, nature of, 184
 FISCHER on inoculation of calves with small-pox matter, 143
 Fishermen, diseases prevalent among, 580
Flacherte (disease of silk-worms), nature and microbes of, 122
 Floating hospitals for Port districts, 588
 FLÜGEL on leucocytes as destroyers of bacteria, 51
 FOA on microbe of croupous pneumonia, 129
 Food of the soldier, 619
 — in India, 621
 — on active service, 622
 — sailors', quality and examination of, 561
 Foods, choleraic diarrhoea as caused by, 189
 Foot-and-mouth disease, section through eruption of, plate v. fig. 31
 — streptococci of, 136
 — streptococcus of, plate iv. fig. 27
 — symptoms and appearances of, 134
 — transmission of, from cows to men by milk, 135
 Foreign armies, dress and accoutrements of, 637
 — food of, 624
 Foulbrood (disease of bees), 122
 Fowl cholera, bacilli of, 103; plate viii. figs. 53, 55
 — streak-culture of, on surface of slanting gelatine, plate xxvii. fig. 7
 — enteritis, bacilli of, 104; plate viii. figs. 50-52

GRA

- Fowl enteritis, bacilli of, colonies on gelatine, plate xxxii. fig. 48; plate xxxiii. fig. 58
 — streak-culture on surface of slanting gelatine, plate xxvii. fig. 8
 FOX, WILSON, on specific virus of tubercle, 217
 FRAENKEL and SIMMONDS on production of typhoid in rabbits, 184
 France, graveyards and cremation in, 718
 FRANKLAND, Dr. P., examination of bacteria in air, 63
 FRÄNKLER and WEICHELBAUM, on diplococcus of pneumonia, 126
 French Navy, regulation kit for, 573
 FRIEDLÄNDER's bacillus of pneumonia, 124
 Fulham Small-pox Hospital, effects of, on neighbourhood, 789, 794
- GAFFKY on bacillus of typhoid, 168
 GAMALEIA on vibrio identical with comma bacillus, 185
 Garden-earth, œdema fluid of guineapig dead after inoculation with, plate vi. fig. 40
 — tissue-juice of guineapig after infection with, plate vii. fig. 45
 Gastric juice, action of, on microbes, 78
 GAYTON, Dr., on influence of multiple marks of vaccination, 447
 Gelatine, cultivation of bacteria in, 35
 — liquefaction of, by bacteria, 36
 — staining of, by aniline dyes, 37
 General Medical Council and diplomas in State medicine, 737
 German measles (see 'Rötheln'), 263
 Germany, cremation in, 717
 — vaccination laws in, 404
 GILBERT ANGELICUS on small-pox, 388
 Glanders and farcy, appearances in, 203
 — — — bacilli of, 204
 — — — communicability of, to animals and man, 204
 — — — cultures of bacilli of, 204
 — — — literature on subject of bacilli of, 205
 — — — method of staining bacilli of, 33
 — — — symptoms of, in man, 204
 — bacilli of, plate xiv. figs. 100, 101; plate xxii. fig. 139; plate xxiii. fig. 140
 — colonies of, on agar, plate xxxi. fig. 50; plate xxxiii. fig. 54
 — culture of, on potato, plate xxxviii. fig. 90; plate xxxix. fig. 91
 — stab culture of, in broth gelatine, plate xxxvi. figs. 75, 76
 GOLAI on *plasmodium malarie*, 191
 Gonococci in gonorrhœal pus, plate xiv. fig. 96; plate xviii. figs. 121, 122
 Gonorrhœa, gonococcus of, 132
 GOODHART, Dr., on diagnosis of German measles, 265
 GRAM's method of staining sections, 33
 Graves, plot-system of, 725
 — public, improved construction of, 726
 — suggestions for improvement of, 726
 Graveyards, accumulation of bodies in, 698
 — as sources of disease, 700

GRA

- Graveyards, germs of disease retained in, 702
 — in Liverpool, removal of bodies from, for re-interment, 721
 — in London, 693
 — noxious emanations emitted by, 701
 — offensiveness of, 697
 — pollution of water by, 701
 — scarlet fever epidemic after breaking up, 702
 Great-pox, distinguished from small-pox, 390
 Greece, ancient, burial customs in, 687
 Greece and Rome, small-pox in, 385
 GREEN's method of ventilating ships, 540
 GREENHOW, Dr., on distribution of death and disease, 498
 GREGORY OF TOURS on small-pox in sixth century, 386
 GRESSWELL, Dr., on mortality from scarlet fever, 271
 Grouse disease, bacilli of, 105
 — — — among blood corpuscles, plate vii. fig. 47
 — — — colonies on gelatine, plate xxxiii. fig. 60
 — — — from infected mouse, plate ii. fig. 8
 — — — in blood of ammer, plate vii. fig. 48
 — — — streak-culture, plate xxvii. fig. 11
 — — — blood of guineapig infected with bacilli of, plate vii. fig. 49
 Guineapigs, experiments upon, with cholera bacilli, 182
 Gulf Stream, temperature of air in, 518
 GUY, Dr., on averages in statistics, 487
 Gymnastic training of soldiers, 638
 HAUSER's *proteus* in putrid meat, 101
 Heat, as influencing growth and life of bacteria, 43
 — destruction of bacteria by, 80
 Heat-stroke, causes of, 662
 — early signs of, 663
 — in camps and barracks, 663
 — liability to, increased by alcohol, 663
 — measures of protection against, 662
 HEIDENHAIN, Dr. M., on changes in nuclei and cells, 235
 Hendon farm, malady in cows at, 141
 — — scarlet fever by milk from, 148
 Herpes fungus, mycelium and spores, plate xli. fig. 101
 Hide-sorters, anthrax of, 113
 Hindoos, burial customs among, 684
 Hindostan, method of inoculation in, 409
 HIRSCH, Dr., on connection between erysipelas and puerperal fever, 355
 — — — epidemic pneumonia, 372
 — — — geographical distribution of cerebro-spinal fever, 305
 — — — — diphtheria, 295
 — — — — influenza, 283
 — — — — measles, 258
 — — — — scarlet fever, 266
 — — — — typhoid, 315
 — — — — typhus, 275
 — — — — yellow fever, 374

INF

- HIRSCH, Dr., on recurrence of small-pox epidemics, 396
 HIRSCHBERGER on milk of tubercular cows, 214
 HOLINSHED's 'Chronicles,' small-pox referred to in, 389
 Horn-pox, a variety of small-pox, 405
 Horse-pox, relation of, to cow-pox, 417
 Hospital fatality of small-pox, 399
 Hospitals, dissemination of small-pox from, 791
 — — — military, in India, 615
 — — — the field, 616
 — — — organisation of, 615
 — — — points to be secured in, 613
 — — — stationary field, 617
 — — — wards of, 614
 — — — public, for infectious diseases, 786
 — — — — influence of, on surrounding neighbourhoods, 788
 — — — ships as, 618
 House-refuse, duties of Medical Officer of Health with regard to, 771
 Houses, defects in, dangerous to health, 760
 — examination of, by Medical Officers of Health, 774
 Housing of Working Classes Act (1890), 756
 HUEPPE on changes in *bacilli anthracis*, 120
 — — — spore-formation in spirilla, 25
 HUTCHINSON, Mr., on vaccinal syphilis, 457
 Huts as barracks, 608
 — temporary, on service, 609
 HUXLEY, Prof., on fungus of salmon-disease, 239
 Hydrophobia, 195
 — attenuation of virus of, 198
 — in man, pathology of, 195
 — nature of virus of, 202
 — PASTEUR's method of treatment of, 198
 — preventive and protective inoculation against, 198
 — Report of English Commission on, 196
 — results of PASTEUR's treatment of, 201
 — uncertain occurrence of, in persons bitten, 200. (*See also* 'Rabies')
- ICELAND, ravages of small-pox in, 387
 Immunity, production of, by injection of 'toxins,' 56
 India, burial customs in, 685
 Infantile diarrhoea, food as a cause of, 345
 Infantry, dress and accoutrements of, 636
 Infants, suggested rules for burial of, 796
 Infection, dissemination of, from hospitals, 791
 — manner of, 74
 — nature and meaning of, 71
 Infectious diseases, ambulances for cases of, 796
 — — among scholars, means of checking, 802
 — — communicated by ships' cargoes, 581
 — — compulsory notification of, 798
 — — conditions requisite for development of, 244

INF

- Infectious diseases, *de novo* theory of, 245
 — — descent of, from antecedent cases, 247
 — — determination of separate natures of, 251
 — — difficulty of tracing previous cases of, 251
 — — dual notification of, 799
 — — due to microphytic life-processes, 247
 — — duties of Medical Officers of Health in regard to, 782
 — — epidemic, pandemic, and endemic classes of, 252
 — — essence of, 3
 — — evolution in connection with, 250
 — — germ theory of, 243
 — — hospital accommodation for, 786
 — — illustrations of new beginnings of, 249
 — — immunity from, of certain individuals, 255
 — — influence of season on spread of, 256
 — — investigation of outbreaks of, by Medical Officers of Health, 806
 — — liability to, as influenced by age and sex, 255
 — — list of, 243
 — — micro-organisms as causes of, 4
 — — modes of spread of, 252
 — — natural history of, 243
 — — notification of, 797
 — — — by Guardians of Poor, 797
 — — pathology and etiology of, 3
 — — period of incubation of, 253
 — — periodicity in, 256
 — — protection from, 252
 — — question as to continuity of series of, 248
 — — recent increase of knowledge of, 3
 — — relative fixity of type of, 251
 — — removal of patients suffering from, 801
 — — rules regarding notification of, 799
 — — special seats of invasion of, 253
 — — 'superadded waves' of, 257
 — — variations between epidemics of, 248
 — — Notification Act, diseases specified in, 799
 — — — measures for disarming opposition to, 800
 — — — powers and duties of Medical Officers of Health under, 800
 — — Prevention Act (1890), powers of Medical Officers of Health under, 813
 Inflammation and suppuration, relation of microbes to, 90
 Influenza, bacteria found in, 130
 — blood from case of, with bacilli, plate xiv. fig. 95
 — case mortality of, 286
 — cause and dissemination of, 287
 — chains of short rods from bronchial sputum of, plate i. fig. 6
 — geographical distribution of, 283
 — history of, 282
 — influence of age and sex on mortality from, 286
 — — — race, climate, and season on, 283

KIT

- Influenza, mortality from, 284
 — periods of incubation and infectiveness of, 289
 — propagation of, by human intercourse, 288
 — protection from, 287
 — symptoms resembling, in animals, 288
 Inhumation first practised in Europe, 679
 Inoculation, alleged evil results of, 413
 — as a protection from small-pox, 408
 — cases of small-pox after, 414
 — fatality rate of, 416
 — insusceptibility to, 413
 — invention of, in China, 385
 — made illegal, 416
 — method of testing efficacy of, 414
 — methods of, 408
 — opinions regarding value of, 412
 — phenomena of, 409
 — practice of, in England, 410
 — profit and loss by, 415
 — protective power of, 414
 — repeated, effects of, 415
 — Suttonian method of, 411
 Inspector of Nuisances, duties of, under Infectious Diseases Prevention Act (1890), 813
 — — — — Notification of Infectious Diseases Act, 801
 — — — — with regard to dairies, cowsheds, and milkshops, 811
 — — — relations of, to Medical Officer of Health, 744
 Instructions to public vaccinators, 427
 Ireland, appointment and duties of Medical Officers of Health in, 821
 Iron, age of, sepulture in, 679
 Italy, cremation in, 717
 JENNER, early experiments with vaccination by, 416
 — on constitutional symptoms produced by vaccination, 422
 — — modified forms of small-pox, 407
 — — relationship of small-pox to cow-pox, 142, 423
 — — spurious small-pox and cow-pox, 420
 JENNER, Sir W., on differences between typhus and typhoid, 815
 JEVONS, Prof., on correction of results in statistics, 487
 Jews, burial customs among, 685
 JOHN OF GADDESSEN on small-pox, 888
 JONSON, Ben, allusion to small-pox by, 890
 JURIN's statistics as to fatality of small-pox, 899
 KARLINSKI on microbes in pus, 90
 KARTULIS, on *amœba coli* in dysentery, 186
 — on bacilli of catarrhal conjunctivitis, 133
 Keswick, epidemic of scarlet fever at, 146
 Kilmarnock, lessons from epidemic of small-pox at, 436
 Kitchens attached to barracks, 606
 KITISATO on bacilli of influenza, 131
 — — production of tetanus in animals, 193

KIT

- KITT on production of tetanus in horses, 195
 KLEBS' 'antidote theory' of acquired immunity, 56
 — on microbe in blood in influenza, 130
 KOLB on microbes of purpura hæmorrhagica, 102
 KOCH on anthrax bacilli, 110
 — — bacilli of cholera, 18, 175
 — — — tuberculosis, 217
 — — — in scrofula and lupus, 221
 — — chemical disinfection, 81
 — — Egyptian ophthalmia, 133, 229
 — — imperfect protection by vaccine against anthrax 121
 — — influence of gastric juice on cholera bacilli, 182
 — — malignant œdema, 99
 — — mouse-septicæmia, 100
 — — results of feeding animals with cholera dejections, 181
 — — septicæmia in rabbits, 98
 — — spirilla of relapsing fever, 172

LANCASHIRE COUNTY COUNCIL, rules for Medical Officers of Health of, 740

Lancets used for vaccination, cautions as to, 429

LAVERAN on *plasmodium malariae*, 191

LAVERAN's corpuscles, from atypical case of malaria, plate xlii. figs. 132-3

Lead coffins, dangers attending use of, 723

Leicester, default of vaccination in, 431

— epidemics of diarrhoea at, 340

— experience of isolation at, 460

Leprosy a communicable disease, 224

— bacilli of, 224; plate xvii. figs. 113, 114; plate xxiv. figs. 145, 146

— inoculation experiments with, 225

— pathology of, 228

— question of conveyance of, by vaccination, 459

Leptothrix, formation of, 11

LEUCKART on coccidia, 232

Leucocytes as a nidus for certain microbes, 52

— objections to METSCHNIKOFF's theory as to action of, 51

— question as to germicidal power of, 50

Leucocytosis an inconstant phenomenon, 52

LEWIS, T., on flagellate monadina in blood of horses, 231

Libraries, public, books from, in infected houses, 801

Life, expectation of, 490

— mean and probable duration of, 490

— insurance, tables of, 490

— tables, construction of, 488

Light as influencing growth and life of bacteria, 43

Lime as a disinfectant, 83

LINGARD on inoculation of guineapigs with lupus and scrofula, 221

— transmission of anthrax to the fœtus, 77

LISTER, Sir J., on doctrine of spontaneous generation, 245

MAL

Liverpool, experience of burial-grounds in, 720

— extramural cemeteries near, 724

— rules as to space round houses in, 762

Local authorities, provision of isolation hospitals by, 787

— Government Board and Medical Officers of Health, 734

— — — rules (1891) for appointment and duties of Medical Officers of Health, 741, 747

Lodging-houses, inspection of, by Medical Officers of Health, 765

LÖFFLER on bacillus of diphtheria, 155

— — — glanders, 204

— — — method of staining of, 33

London bills of mortality with regard to small-pox, 433

— decline in mortality from small-pox in, 434

— Fever Hospital, question as to influence of, on neighbourhood, 789

— in 1665, churchyards in, 693

— multiplication of graveyards in, 693

— number of graves in, 705

LONGSTAFF, Dr., on causes of diphtheria 303

— — — relation between diphtheria and croup, 367

— — — — erysipelas and puerperal fever, 355

LUBARSCH on germicidal action of living tissues, 50

Lupus, relation of, to tuberculosis, 221

LUSTGARTEN on method of staining syphilis bacillus, 33

Lymph and blood, normal, power of, to kill bacteria, 49

— calf, use of, 429

— vaccine, cautions as to taking, 428

— — storage of, 428

MACNAMARA, Mr., on locality as affecting spread of cholera, 334

Malaria plasmodium, plate xlii. figs. 106-131

Malarial disease among soldiers, 658

— conditions predisposing to, 658

— during Ashanti war, 659

— forms of, 658

— history and distribution of, 346

— influence of age and sex on liability to, 349

— — — seasons on spread of, 349, 659

— measures of protection against, 659

— nature of poison of, 658

— protection or acquired immunity in, 349

— — quinine as a prophylactic in, 661

— spread of, by air-currents, 349

— — — water, 350, 660

— — transportation of organisms of, 347

Malignant œdema, cultivation of the aerobic bacillus of, in broth, plate vi. fig. 42

— — — anaerobic bacillus of, in grape-sugar gelatine, plate xxix. fig. 36

— — fluid of guineapig, dead of, after inoculation with garden-earth, plate vi. fig. 40

MAL

- Malignant oedema, fluid of rabbit, dead of, after inoculation with the aerobic bacillus of, plate vi. fig. 41
 — — plate-cultivation in gelatine of aerobic bacillus of, plate xxvi. fig. 4
 — — production of, 99
 — — shake-cultures in gelatine of the aerobic bacillus of, plate xxviii. figs. 24-26
 — — stab-culture in gelatine of the aerobic bacillus of, plate xxviii. fig. 28
 Malta fever, micrococci found in, 171
 Malthusian theory of population, 472
 MARCHIAFAVA and CELLI, on plasmodium malaris, 192
 Marching, amount of work done in, 640
 — in foreign armies, 640
 — pace and steps in, 639
 — precautions as to, 641
 Marine hygiene, 513
 — — and port sanitation, 583
 — — prevention of disease on shipboard, 588
 — — bibliography of, 596
 — — cargoes and other contents of ships in relation to, 581
 — — rules for, 580. (*See also* 'Sailors,' 'Sea,' 'Shipping,' and 'Ships')
 Marquess, hospital, 612
 Marriage-rate, statistics of, 498
 Marriages, births, and deaths, returns of, 474
 MARSON, Mr., on value of multiple marks of vaccination, 446
 MARTIN, Dr. S., on chemical nature of diphtheria poison, 157
 Mastitis, purulent matter in abscess of, plate iv. fig. 24
 McLEOD and MILLS on production of cholera in guineapigs, 180
 McVAIL on statistics of syphilis mortality, 459
 Measles, cause and mode of dissemination of, 262
 — fatality of, 260
 — history and geographical distribution of, 257
 — influence of age, sex, and race, on liability to, 261
 — — climate and season on prevalence of, 258
 — mortality, actual, from, 259
 — periodicity of, 258
 — periods of incubation and infectiveness of, 262
 — protection from, 263
 — resemblance between, and R6theln, 265
 Meat, putrid, bacillus in, 101
 Meat-poisoning, bacilli in kidney from case of, plate xxv. fig. 152
 Medical Officers of Health, analyses of water and air not the duty of, 769
 — — and Act of 1872, 734
 — — appointment of, under Act of 1875, 735
 — — correspondence of, with Local Government Board, 762
 — — creation of, 733
 — — drawbacks of uncertain tenure of office by, 746

MED

- Medical Officers of Health, duties of (Local Government Board, 1891), 747
 — — — under Housing of Working Classes Act (1890), 756
 — — — with regard to alkali works, 778
 — — — — — burial-grounds, 809
 — — — — — cholera out-breaks, 784
 — — — — — closure of schools, 808
 — — — — — construction of mortuaries, 809
 — — — — — disinfection, 788, 814
 — — — — — epidemics, 805
 — — — — — hop-pickers, 766
 — — — — — hospital accommodation for infectious diseases, 786
 — — — — — house refuse, 771
 — — — — — infectious diseases, 782
 — — — — — investigating outbreaks of infectious diseases, 806
 — — — — — new streets and buildings, 809
 — — — — — nuisances, 771
 — — — — — offensive trades, 777
 — — — — — pollution of rivers, 771
 — — — — — re-vaccination, 805
 — — — — — Sale of Food and Drugs Act, 780
 — — — — — horseflesh, &c., 779
 — — — — — sewerage and drainage, 770
 — — — — — slaughter-houses, 780
 — — — — — taking legal proceedings, 810
 — — — — — unsound meat, 778
 — — — — — water - supply, 767
 — — — — for City of London (1892), 786
 — — — — general rules for guidance of, 743
 — — — — in Ireland, appointment and duties of, 821
 — — — — Metropolis, duties of, 740
 — — — — Scotland, appointment and duties of, 815
 — — — — inspection of bakehouses by, 753
 — — — — canal-boats used as dwellings, 767
 — — — — cellar dwellings and lodging-houses by, 765
 — — — — dairies, cowsheds, and milkshops by, 810
 — — — — factories and workshops by, 755
 — — — — infected ships by, 785
 — — — — overcrowded buildings by, 764

MED

Medical Officers of Health, inspection of premises by, method of, 774
 ————— seamen's lodging-houses by, 766
 ————— tents, vans, and sheds by, 766
 ————— 'unhealthy areas' by, 766
 ————— unhealthy dwelling-houses, 757
 ————— water-supply by, 767, 777, 807
 ————— working-class lodging-houses by, 759
 ————— knowledge of subjects to be possessed by, 742
 ————— length of tenure of office by, 745
 ————— liabilities of, 752
 ————— notes of proceedings of, 750
 ————— notification of infectious diseases to, 797
 ————— of Administrative Counties, 738
 ————— Port Sanitary Authorities, duties of, 749
 ————— penalties incurred by, for malpractices, 752
 ————— personal qualifications of, essential to success, 741
 ————— powers and duties of, under Notification Act, 800
 ————— of, to enter premises, 772
 ————— qualifications and duties of, 736
 ————— relations of, to Inspectors of Nuisances, 744
 ————— reports made by, 751
 ————— rules (1891) for appointment and duties of, 741
 ————— salaries and pensions of, 747
 Medium, influence of, on activity of bacilli, 70
 — reaction of, as affecting growth of bacteria, 45
 Meningitis, diplococcus pneumoniae in, 129
 Mercury perchloride as a disinfectant, 82
 Metropolis, duties of Medical Officers of Health in, 740
 MIRSCHNIKOFF on destruction of bacteria by white corpuscles, 50, 172
 Mexico, ravages of small-pox in, 391
 Microbes, manner of infection by, 74
 Micrococci, a group of bacteria, 8
 — absence of motility in, 15
 — differentiation of species of, 28
 — distinguished from bacilli, 10
 — forms of, 8
 — in erysipelas, 93
 — of suppuration, 84
 — vaccine lymph, 144
 — pyogenic, chemical substances produced by, 91
 — in ulcerative endocarditis, 92
 — mode of action of, 91
 — question as to spores of, 26
 — species of, in mouth and pharynx, 46
 — variations in forms of, 9
 Micrococcus aquatilis, alleged existence of, in pure water, 17

MUR

Micrococcus aurantiacus, streak-culture of, on gelatine, plate xxxii. fig. 61
 — prodigiosus, shown to be a bacillus, 9
 — scarlatinae, culture on gelatine, plate i. fig. 5
 — tetragonus, 9, 89; plate i. fig. 4
 Micro-organisms as causes of disease, 4
 — classes of, 4
 — varieties in pathogenic properties of, 248
 Microphytes, saprophytic, modifications of functions of, 249
 Middlesbrough, bacilli in pneumonia epidemic at, 127
 — pneumonia epidemic at, 372
 MIRSCHER's coccidia tubes, 233
 MIKULICZ's cells of rhinoscleroma, 207
 Military Hygiene, 601
 — service, effects of, 645
 Milk-diphtheria, epidemics of, 161, 301
 — dissemination of typhoid by, 167, 324
 — transmission of scarlet fever by, 141, 146, 273
 ————— tubercle by, 215
 Moisture as affecting growth of bacteria, 18
 Monads in connection with disease, 230
 MONTI on virulence of bacteria increased by other species, 60
 Montreal, lessons from small-pox epidemic in, 450
 MOORE, history of small-pox by, 386
 Mortality as influenced by age, 500
 ————— birth-rate, 501
 ————— occupation, 506
 — bills of, 473
 — from small-pox, decline in, 433
 — in various countries, 392
 — preventable, statistics of, 499
 — rate of, and atmospheric changes, 504
 — density of population, 502
 Mortuaries, duties of Medical Officers of Health with regard to, 809
 MOSLER on epidemic of croupous pneumonia, 129
 MOTT, Mr., on vaccination and inoculation, 426
 Moulds or mycelial fungi, 237
 Mouth and pharynx, bacteria found in, 46
 Mucor a mould-fungus, 239
 — corymbifer, hyphae and sporangia, plate xli. fig. 103
 Mucous membrane, passage of bacteria through, 76
 — presence of bacteria in, 47
 MUDGE, Mr. J., on inoculated small-pox, 435
 Mumps, cause and mode of dissemination of, 293
 — fatality of, 292
 — history and distribution of, 292
 — influence of age, sex, climate, &c., on prevalence of, 292
 — period of incubation of, 293
 — protection from, 293
 — relation of, to other diseases, 293
 MURCHISON, Dr., on non-infectious character of typhoid fever, 319
 — relapsing fever, 281
 — simple continued fever, 312
 — typhoid fever, 314

MUR

- MURPHY, Mr. S., on influence of London Fever Hospital on neighbourhood, 789
 — — — inoculation of cows with viruluous matter, 423
 MUSSULMANS, burial customs among, 685
Mycosis mucorina in man, 239

- NASAL cavities, bacteria present in, 47
 Navies, sickness and mortality of, 574
 Navy, results of re-vaccination in, 443
 NEISSER on bacillus of leprosy, 224
 — — gonococcus of gonorrhoea, 132
 New England, practice of inoculation in, 410
 NICOLAÏER on production of tetanus in animals, 193
 Notification of Infectious Diseases, 798
 Nuisances, duties of Medical Officer of Health with regard to, 771
 Nurses in small-pox hospitals, protection of, by re-vaccination, 444
 Nutrient agar, cultivation of bacteria in, 40

- OAKLEY, Suffolk, epidemic of cerebro-spinal fever at, 307
 OBERMEYER on spirilla of relapsing fever, 171
 Occupations, influence of, on mortality, 506
 Odours, foul, in houses, investigation of, 766
 Oedema fluid of guineapig, after inoculation with anaerobic oedema bacillus, plate vii. fig. 44
 Offensive trades, duties of Medical Officers of Health with regard to, 777
Odium lactis, 237
 — — mycelium and spores, plate xl. fig. 99
 — — of favus, 237
 Ophthalmia among troops, causes and forms of, 666
 — — precautions against, 667
 — — catarrhal or Egyptian, pus with minute bacilli, plate xiv. figs. 97, 98; plate xviii. fig. 123
 — Egyptian, causes of, 133, 229
 Otitis media and rhinitis, caused by *diplococcus pneumoniae*, 130
 Overcrowded buildings, duties of Medical Officers of Health with regard to, 764
 Oxford, epidemic of scarlet fever at, 147

- PAGET, Mr. C. E., on mortality in unhealthy areas in Salford, 760
 Parsees, burial customs among, 684
 PARSONS, Dr., on disinfection by heat, 80
 — — epidemics of influenza, 284, 287
 — — relation of diphtheria to other diseases, 303
 PASTEUR on attenuation of anthrax bacilli, 120
 — — bacilli of anthrax, 112
 — — — fowl-cholera, 104
 — — charbon germs in soil, 702
 — — nature of virus of rabies, 202
 — — preventive inoculation against rabies, 198

POC

- PASTEUR on rabies, 196
 PEARSON, Dr. G., on history of cow-pox, 417
Pébrine (of silkworms), nature and microbes of, 122
Penicillium a mould-fungus, 238
 — *glaucomum*, plate xli. fig. 102
 Periodicity of small-pox, 395
 PERKINS's automatic ventilator for ships, 541
 Persians, ancient burial customs of, 683
 PETRUSCHKY on infection of frogs with anthrax, 53
 PETTENKOFER on influence of ground-water on dissemination of typhoid, 267
 PFLEFFER on chemiotactic power of various substances over bacteria, 54
 PFLEIFFER on bacilli of influenza, 131
 — — coccidia in epithelium in variola, &c., 283
 — — septicæmic effect of vibrio METSCHNIKOFF on pigeons, 185
 Phagocytosis an inconstant phenomenon, 52
 — theory of, 50
 Phthisis, prevalence of, connected with occupations, 364
 — — due to overcrowding, 363
 PIERCE, Dr. B., on torula in diabetic urine, 236
 Pigment formed by some species of bacteria, 44
Plasmodium malariae as a cause of intermittent fever, 191; plate xlii. figs. 108-131
 Plate-culture of bacteria, 37
 Pneumonia, association of, with other diseases, 372
 — bacilli, attenuation of power of, 69
 — — movements of, 14
 — croupous, bacilli of, culture, plate v. fig. 35
 — — — culture in broth, plate xxvii. fig. 19
 — — — stab-culture in gelatine, plate xxxv. fig. 71
 — — — streak-culture, plate xxvii. fig. 10; plate xxxiv. fig. 65
 — colonies of bacilli of, on gelatine, plate xxxi. fig. 51; plate xxxiii. fig. 57
 — lung-juice in, showing blood-discs and bacilli, plate v. fig. 32
 — section through lung in, plate v. figs. 33, 34
 — epidemic, diplococcus of FRÄNKEL and WEICHSELBAUM in, 126
 — — an infectious disease, 123
 — — FINKLER on, 126
 — — microbe in sputum in, 124
 — — symptoms of, 124
 — epidemics of, 373
 — history and distribution of, 369
 — infectiousness of, 371
 — influence of cold in causation of, 370
 — lobar, forms of, 371
 — mortality and fatality of, 372
 — nature and forms of, 368
 — predisposing causes of, 372
 — race, age, and sex as influencing liability to, 371
 Pock, derivation of term, 390

POC

- Pock, of sheep, section through, plate v. fig. 30
- Pollution of rivers, duties of Medical Officers of Health with regard to, 771
- Population, density of, and rate of mortality, 502
- theories of, 472
- Pork and ham, choleraic diarrhoea caused by, 189
- Pork-pie poisoning, streak-culture of bacillus of, on gelatine, plate xxxiv. fig. 62
- Port Sanitary Authorities, duties of Medical Officers of Health of, 749
- Port sanitation, 583
- Ports, hospitals at, 588
- sanitary staffs, and appliances at, 588
- Portsmouth pie-poisoning, stab-culture in gelatine of bacillus of, plate xxviii. fig. 32
- — — streak-culture on gelatine of bacillus of, plate xxvii. figs. 16, 17
- Potassium salts and phosphates necessary for growth of bacteria, 17
- Potato as a medium for cultivating bacteria, 40
- POWER, Mr., on aerial convection of small-pox, 401
- — — diseases in Hendon cows, 148
- — — hypotheses as to cholera, 335
- — — influence of a small-pox hospital on neighbourhood, 789, 794
- Premises, method of examination of, by Medical Officers of Health, 774
- PRESTON, Mr. T., on statistics of syphilis mortality, 458
- Prisons, mortality from phthisis in, 364
- Protection from attacks of small-pox, 408
- Proteid medium for growth of bacilli, 17
- Proteus hominis* in rag-sorters' disease, 102
- *vulgaris*, a bacillus in putrid meat, 101
- colonies of, and 'swarmers,' plate ii. fig. 10
- — — — — $\times 1,000$, plate ii. figs. 11, 12
- Protozoa, flagellate, diseases due to, 230
- Provinces, decline in mortality from small-pox in, 434
- Prussia, mortality from small-pox in, 442
- results of re-vaccination in, 440
- Pseudo-diphtheria, bacilli of, plate xii. fig. 80
- — — — from milk of cow, plate xii. fig. 81
- — — — — colonies on agar, plate xxviii. fig. 30
- — — — — streak-culture on agar, plate xxviii. fig. 31
- Ptomaines, production of, by bacteria, 64, 72
- results of injection of, 72
- Public Health Act (1848), 733
- — — (1872), 734
- Puerperal fever, causes and modes of dissemination of, 359
- — — history of, 357
- — — mortality from, 358
- — — recent decline in mortality from, 359
- — — relation of, to erysipelas, 355
- Purpura hemorrhagica, microbes of, 102

ROM

- Purulent processes, relations of, to bacteria, 90
- Pyæmia, mode of production of, 91
- QUARANTINE and Quarantine Act, 584, 587
- Quarter ill, or black leg in cattle, 108
- RABBITS, septicæmia in (DAVAINE and KOCH), 97
- Rabies, animals susceptible to, 195
- artificial production of, 196
- attenuation of virus of, 198
- dumb and furious forms of, 197
- inoculation of rabbits with virus of, 196
- PASTEUR on production of, 196
- *post-mortem* appearances in animals suffering from, 197
- transmissibility of, 196
- Race as affecting prevalence of small-pox, 394
- Rag-sorters' disease, 101
- RANSOME, Dr. A., on seasonal variation of epidemic diseases, 256
- Rations for sailors, scale of, 563
- of British soldiers, 619
- foreign armies, 624
- Ray-fungus, or actinomycetes, 225
- Recruits, Army, age of, 602
- chest capacity of, 602
- examination of, 601
- height and weight of, 601
- Reformation, disposal of dead at period of, 692
- Relapsing fever, cause and modes of dissemination of, 280
- — — history and distribution of, 278
- — — influence of season on prevalence of, 281
- — — — sex and age on liability to, 279
- — — mortality and fatality of, 279
- — — period of incubation of, 281
- — — protection from, 281
- — — relation of, to other diseases, 281
- — — spirilla of (OBERMEYER), plate xiii. fig. 86
- — — spirillum found in blood in, 171, 280
- Respiratory system, bacteria present in, 47
- Re-vaccination, effects of, 440
- gradual return of susceptibility to, 453
- phenomena of, 431
- results of, in Army and Navy, 441
- Revival of learning, small-pox during time of, 389
- RHAZES, treatise on small-pox by, 385
- Rhea, bacilli in liver of, plate xxiv. figs. 148, 149
- Rhinoscleroma, appearances and nature of, 207
- bacilli found in, 207
- MIKULICZ, cells of, in, 207
- Rinderseuche, bacillus of, 108
- Ringworm, oidium of, 237
- ROBERTS, Mr. C., on physical requirements of factory children, 479
- ROBERTSON, Dr., of Keswick, on epidemic of milk scarlatina, 146
- Roll-plate cultivation of bacteria, 39
- Romans, burial customs among the, 688

RÖT

- Rötheln or German measles, a distinct disease, 263
- — — epidemics of, 264
 - — — incubation and spread of, 264
 - — — mistakes in diagnosis of, 265
 - — — protection against, 265
 - — — resemblance between, and other diseases, 265
- Royal Navy, disqualifications for service in, 558
- — rations for sailors in, 563
 - — regulation kit for, 578
 - — re-vaccination in, 581
 - — sickness and mortality in, 575
 - — tests for fitness for service in, 559
- RUMSEY, Dr., on fallacies of statistics, 470
- Rural Sanitary Districts, formation of, 734
- SACCHAROMYCES MYCODERMA** and acetic acid, 236
- — from an artificial cultivation, plate xl. fig. 100
 - — or torula, plate xli. fig. 98
- Sailors, alleged deterioration of, 557
- biscuits in rations of, 562
 - causes of death among, 576
 - clothing supplied to, 571
 - cooking appliances for, 563
 - diseases prevalent among, 578
 - food of, 560
 - kits and outfits for, 571
 - number of, 556
 - preservation of food of, 562
 - physical qualifications of, 559
 - physique of, 558
 - quality and examination of food of, 560, 568
 - rations of, 563
 - regulation kit for, in Royal Navy, 573
 - sanitary regulations and supervision of, 560
 - scarcity of, 557
 - sickness and mortality among, 574
 - status of, 557
 - tests for fitness for service as, 559
- Sale of Food and Drugs Act, duties of Medical Officers of Health with regard to, 780
- Salmon disease, fungus of, 239
- Sapremia, pathology of, 95
- Saprolegnia, fungus of salmon disease, 239
- of salmon disease, plate xl. fig. 107
- Sarcina, a form of coccus, 9
- lutea, plate i. fig. 3
 - culture of, on potato, plate xxxviii. fig. 87
- SATTLER on trachoma conjunctivæ, 229
- Scarlattina, micrococcus of, plate i. fig. 5
- streptococcus of, plate iv. figs. 25, 26
- Scarlet fever, cause and dissemination of, 272
- — contagious character of, 146
 - — difference between distribution of, and of diphtheria, 267
 - — epidemics of, due to diseased milk, 146
 - — fatality or case-mortality of, 268
 - — history and geographical distribution of, 266

SEP

- Scarlet fever, influence of age and sex with regard to, 271
- — — climate and season on prevalence of, 269
 - — inoculation with virus of, 273
 - — kidney changes in, 145
 - — milk as the vehicle of contagion of, 141, 146, 273
 - — mortality from, 267
 - — period of incubation and infectiveness of, 273
 - — periodicity of, 267
 - — probable transmission of, through diseased cows, 148
 - — protection from, 274
 - — relation of, to other diseases, 274
 - — skin-eruption in, 145
 - — symptoms resembling, in cows, 148
 - — tissues implicated in, 145
- School-teachers, duties of, with regard to infectious diseases, 803
- Schools, closure of, during prevalence of infectious diseases, 803
- SCHOTTLEUS on prolonged vitality of tubercle bacilli, 222
- Scotland, age-incidence of small-pox in, 440
- legislation with regard to burials in, 695
 - lessened mortality from small-pox in, 435
 - Medical Officers of Health in, 815
 - regulations for Medical Officers of Health in, 817
- Scrofula, tubercle bacilli in, 221
- Scurvy among sailors, 578
- — troops, causes of, and precautions against, 665
- Sea, air of, 517
- extent of, 518
 - health preservation and hygiene at, 580
 - influence of, on climate, 517
 - life at, 519
 - movements of, 516
 - voyages, dangers to health in, 520
 - water, analyses of, 514
 - — specific gravity and temperature of, 515
- Season as affecting prevalence of small-pox, 394
- — influencing spread of infectious diseases, 256
- SEARON, Dr., on hospital fatality from small-pox, 399
- — inoculation of cows with variolous matter, 426
- SEMMELWEISS on cause of puerperal fever, 359
- Septic intoxication from injection of ptomaines, 72
- Septicæmia as a term applied to a group of diseases, 96
- human, microbes of, 101
 - in guinea-pigs and mice, microbe causing, 108
 - — mice (KOCH), 100
 - — rabbits (DAVAINE), 97
 - — — (KOCH), 98
 - KOCH's, blood of mouse dead of, plate vi. fig. 39

SEP

- Septicæmia, Koch's, lung of mouse dead of, plate xx. fig. 128
 — mode of production of, 91
 — produced in guineapigs by injection of comma bacilli, 180
 — puerperal, streptococci in, 102
 — specific microbes capable of causing, 97
 — symptoms and appearances of, 95
 Sewage, bacillus filamentosus from culture of, plate ii. fig. 14
 Sewerage and drainage, inspection of, by Medical Officers of Health, 770
 Sex as affecting prevalence of small-pox, 395
 SHAKESPEARE, small-pox referred to by, 391
 Sheffield, lessons drawn from small-pox epidemic in, 451
 — variations in age-incidence of small-pox in, 438
 Sherborne, typhoid epidemic at, 321
 Shipping of principal maritime countries, 533
 — Merchant, statistics of, 532
 — Royal Navy, 531
 — statistics of, 531
 Ships, accommodation and space for crews in, 542
 — air and ventilation of, 534
 — and questions of quarantine, 537
 — apprentices in, 555
 — arrangements for cleansing, 552
 — — drainage of, 549
 — bilge-water as affecting air of, 534
 — cargoes, as affecting air of, 534
 — cholera on board and duties of Medical Officer of Health, 585
 — classification of, 521
 — closets in, 550
 — clothing on board, 571
 — crews of, 554
 — dangerous goods on board, 582
 — decay of wood in, 524
 — disinfection of, 595
 — from foreign stations, inspection of, 584
 — infectious cargoes on board, 581
 — iron, construction of, 530
 — — steel, and composite, 526
 — manning of, 555
 — materials used in construction of, 523
 — merchant, food allowance in, 567
 — — numbers employed in, 555
 — — ordinary condition of forecabin in, 552
 — metals used in building, 526
 — methods of ventilating, 536
 — of Royal Navy, 523
 — passenger, 531
 — — rules for accommodation in, 549
 — preservation and storage of water on board, 570
 — — of wood in, 525
 — prevention of disease in, 583
 — principal defects of sailors' quarters in, 548
 — purposes served by, 522
 — sailing, and steamers, 522
 — sanitary supervision of, 583
 — spirit ration on board, 571
 — structural sanitary defects in, 595

SMA

- Ships, temperature and lighting of, 553
 — transport of patients from, 593
 — various parts of, 528
 — ventilation of, plans for, 539
 — water and beverages on board, 568
 — wind-sails for ventilation of, 537
 — wooden, construction of, 527
 Sickness, statistics of, and their defects, 477
 SIMON, Sir J., on preventable mortality, 499
 — — — on relation between filth and spread of cholera, 334
 — — — — indoor occupations and phthisis, 364
 Simple continued fever, 312
 — — — doubts as to existence of, 313
 Slaughter-houses, duties of Medical Officers of Health with regard to, 780
 Small-pox, aerial convection of, 400
 — after vaccination, 420
 — age as affecting fatality of, 397
 — — — prevalence of, 396
 — age-incidence of, as showing value of vaccination, 435
 — alterations in age-incidence of, 437
 — and vaccination, 385
 — — — future of, 461
 — and vaccinia, relations between, 427
 — cause and mode of dissemination of, 400
 — climate and soil as affecting prevalence of, 394
 — decline in mortality from, since vaccination, 433
 — degree of susceptibility to, after vaccination, 453
 — derivation of term, 390
 — distinguished from syphilis, 389, 390
 — effect of sanitary measures on spread of, 456
 — epidemics of modified forms of, 407
 — eruption of, modified by vaccination, 448
 — experience of protection from, during present century, 404
 — factors determining epidemic recurrence of, 396
 — fatality of, as affected by local conditions, 400
 — — — in hospitals, 399
 — — — new countries, 397
 — frequency of second attacks of, 407
 — geographical distribution of, 393
 — history of, 385
 — — — during revival of learning, 389
 — hospitals, influence of, upon neighbourhood, 789, 794
 — — selections of sites for, 796
 — immunity from, in Tasmania and New Zealand, 393
 — in Africa, 394
 — — England in Middle Ages, 388
 — — Europe in Middle Ages, 386
 — — Greece and Rome, 385
 — — India and China, 393
 — — Ireland during Middle Ages, 387
 — — the New World, 391, 394
 — incubation of, 402
 — infectivity of, 400
 — insusceptibility to contagion of, 403

SMA

- Small-pox, JURIN's statistics as to fatality of, 398
 — lessened frequency of, in present century, 405
 — lessons to be drawn from epidemic, 1870-1873, 450
 — memorandum on steps to be taken when prevalent, 805
 — mitigation of, by vaccination, 421
 — modification of, by vaccination, 445
 — modifications of, 405, 407
 — — distinguished from chicken-pox, 462
 — natural, as a protection from subsequent attacks, 403
 — nomenclature of, 390
 — nurses, protection of, by re-vaccination, 444
 — old writers upon, 385
 — period of infectiveness of, 402
 — periodicity of epidemics of, 395
 — post-vaccinal, 405
 — prevalence of, from early times, 391
 — prevention of, 460
 — — by vaccination, 419
 — protection from attacks of, 402
 — — by inoculation, 408
 — — — natural small-pox, 406
 — — — during present century, 404
 — — — influenced by age, 404
 — — — loss of, by children, 404
 — — — permanence of, 404
 — race as affecting prevalence of, 394
 — ravages of, in Iceland, 387
 — referred to by HOLINSHED, 389
 — — by SHAKESPEARE, 390
 — regarded as an all-prevailing scourge, 391
 — relationship of, to cow-pox, 423
 — results of inoculation of, on the cow, 423
 — season as affecting prevalence of, 394
 — second attacks of, 406
 — — — fatality from, 407
 — sex as affecting mortality from, 395
 — share of, in total mortality, 392
 — spread of, through Europe from Arabia, 386
 — spurious, JENNER's cases of, 420
 — table showing mortality from, in Army and Navy, 443
 — table showing mortality from, in Prussia, 442
 — value of vaccination shown during epidemics of, 448
 SMITH, Dr. W. R., on streptococci of puerperal septicaemia, 102
 Soil as influencing properties of microbes, 248
 — examination of bacteria in, 62
 Soldiers, boots for, 638
 — causes of mortality among, 646
 — cholera among, 648
 — clothing for, 630
 — diarrhoea and dysentery among, 668
 — effects of military service upon mortality of, 645
 — food of, 619
 — gymnastic training of, 638
 — head-dress of, 638

STA

- Soldiers, heat-stroke among, 662
 — hospital admissions and invaliding among, 647
 — issue of spirit ration to, 623
 — malarious fevers among, 658
 — marches of, 639
 — measurements of (Dr. SEGGEL), 608
 — ophthalmia among, 666
 — prevention of disease among, 647
 — regulation kits for, 630
 — — — in cold and tropical climates, 631
 — scale of messing for (Col. BURDETT's), 620
 — scurvy among, 665
 — selection of, 601
 — specific diseases prevalent among, 648
 — statistics of disease and mortality of, 642
 — typhoid fever among, 652
 — venereal diseases among, 667
 — water-supply for, 627
 — work of, 638
 — yellow fever among, 656
 SPEAR, Mr., on anthrax of hide-sorters, 113
 SPENCER, H., on evolution and 'first organism,' 246
 Spirilla, absence of spore formation in, 26
 — cilia and flagella of, 15
 — differentiation of species of, 28
 — distinguished from bacilli, 14
 — forms of, 13
 — KOCH's (*see* 'Cholera')
 — movements of, 15
 — of relapsing fever, plate xiii. fig. 86
 — with two flagella at one end, plate iii. fig. 21
 Spore-formation confined to certain species of bacilli, 26
 Spores of bacteria, 22
 — — — first appearance of, 24
 — — — germination of, 27
 — — — influence of nutritive material on formation of, 25
 — — — — oxygen on formation of, 25
 — — — methods of staining, 31
 — — — — discovering, 25
 — — — question as to division of, 27
 — — — temperature necessary for formation of, 25
 SQUIRE, Dr., on German measles, 264
 Stab-culture of bacilli, 36
 Staining dyes for bacilli, 29
 Staphylococci, 8
 — species of, in pus, 84
 Staphylococcus albus liquescens, cover-glass specimen of, plate i. fig. 1
 — — stab-culture of, in gelatine, plate xxxv. fig. 71
 — aureus, broth-culture of, plate xxvii. fig. 18
 — — in renal vessels of a rabbit, after injection of a culture, plate xix. fig. 126
 — — in valve, in case of ulcerative endocarditis, plate xviii. fig. 125
 — — streak-culture of, on agar, plate xxxiv. fig. 63
 — pyogenes, 87
 — — albus, 86
 — — aureus liquescens, 84

STA

- Staphylococcus pyogenes aureus*, rapidity of growth of, 20
 — sarcina-like, plate i. fig. 2
 State-medicine, resolution of General Medical Council as to diplomas in, 737
 Steam, action of, on bacilli, 80
 STEPHENS, Mr. Justice, on legality of cremation, 715
 STERNBERG on immunity from yellow fever, 376
 Stone Age, sepulture in the, 675
 Streak-culture of bacteria, 37
 Streets and buildings, duties of Medical Officers of Health with regard to, 809
 Streets, new, in London, rules for width, &c., of, 762
 Streptococci, 9
 — in valve in ulcerative endocarditis, plate xviii. fig. 124
 — modifications of pathogenic powers of, 69
 — of erysipelas, 94
 — variations in modes of division of, 21
 Streptococcus of erysipelas, streak-culture of, plate xxvii. fig. 15
 — of foot-and-mouth disease, culture, plate iv. fig. 27
 — pyogenes, in chronic abscess, plate vi. fig. 38
 — — streak-culture of, plate xxvii. fig. 14
 — — scarlatinae, from agar culture, plate iv. fig. 25
 — — from culture, plate iv. fig. 26
 — — streak-culture of, plate xxvii. fig. 13
 Suakim Expedition, 1885, rations during, 622
 Suppuration and acute phlegmon, causes of, 84
 — — — occurrence of, in absence of microbes, 91
 — — — processes in, 83
 SUTTON, Daniel, inoculation as practised by, 411
 Sweden, lessened mortality from small-pox in, 435
 Swine erysipelas, bacilli of, 105
 — — bacillus of, in blood of pigeon, plate ix. fig. 57
 — — — in liver of pigeon, plate ix. fig. 58
 — — — stab-culture in gelatine, plate xxviii. fig. 21; plate xxxvi. fig. 77
 Swine fever, 106
 — — bacillus of, in spleen of mouse, plate viii. fig. 56
 — — — plate cultivation, plate xxi. fig. 49
 SYDENHAM on modified forms of small-pox, 407
 Syphilis, appearances and symptoms of, 205
 — as propagated by vaccination, 457
 — bacilli of, method of staining, 33
 — etiology of, 206
 — LUSTGARTEN's bacillus of, 206
 — statistics of mortality from, 458
 TASMANIA and New Zealand, immunity from small-pox in, 393

TUB

- TAYLOR, Dr. M. W., on milk-epidemic of scarlet fever, 273
 Temperature as affecting growth of bacilli, 18
 Tents used in foreign armies, 612, 613
 — — — India, 612
 — vans, and sheds, inspection of, by Medical Officers of Health, 766
 — various kinds of, 612
 Tetanus a communicable disease, 193
 — bacilli of, 193; plate xv. fig. 102
 — — — cultivation in grape-sugar gelatine, plate xxix. fig. 37
 — cultures of bacilli of, 194
 — immunity produced by injections of certain albumens, 57
 — in horses, 195
 — methods of producing insusceptibility to, 195
 — pathology of, 194
 — production of, in animals, 193
 THREES' plan of ventilating ships, 541
 THOMAS, Prof., on epidemics of rubella, 264
 — — — inoculation of scarlet fever virus, 273
 THOMPSON, Prof., on varieties of small-pox, 406
 — Sir H., on cremation, 713
 — — — dangers of modern burial-practice, 711
 THORNE, Dr., on dissemination of typhoid by water, 167
 — — — epidemic of typhoid at Caterham, 823
 — — — influence of multiple marks of vaccination, 446
 — — — mortality of scarlet fever, 267
 — — — protection of nurses by re-vaccination, 444
 — — — provision of isolation hospitals, 787
 — — — school attendance, and spread of diphtheria, 300
 — — — transmission of foot-and-mouth disease by milk, 135
 Timber of ships, durability of, 524
 — — — preservation of, 525
 — — — strength of, 526
 Tissue, method of staining sections of, 33
 Tissues, normal, resistance of, to invasion of bacteria, 49
 TIZZONI and CATTANI on production of insusceptibility to tetanus, 195
 Torula or saccharomyces, plate xli. fig. 98
 Towns Improvement Clauses Act (1847), 773
 Toxins, injection of, producing immunity against virulent attacks, 56
 — production of, by bacteria, 64
 Trachoma conjunctivae, pathology of, 229
 Troop-ships, scale of rations for, 565
 TROUSSEAU on attenuation of variolous virus, 418
 — on phenomena of inoculation, 409
 Tube-plate cultivation of bacteria, 38
 Tubercle, appearances and nature of, 208
 — bacilli of (Koch's), 217
 — — — attenuation of power of, 68

TUB

- Tubercle, bacillus of, plate xv. figs. 108-5; plate xvi. figs. 106, 107
- — — in liver of rabbit, plate, xvi. fig. 110
 - — — — lung of cow, plate xvi. figs. 108 112
 - — — — — rabbit after injection of culture, plate xvi. fig. 109
 - — — — spleen of fowl, plate xvi. fig. 111
 - — — — surface cultivation of, on glycerine agar, plate xxxv. figs. 68, 69
 - communicability of, 210
 - communication of, from person to person, 215
 - cultivation of bacillus of, 219
 - feeding animals with, 211
 - giant-cells in, 209
 - inoculation of guineapigs with, 210
 - — — with cultures of bacilli of, 220
 - method of staining bacilli of, 81
 - necrosis and caseation of, 209
 - production of, in animals by inhalation, 212
 - specific virus of, 216
 - structure of, 208
- Tuberculin as a means of diagnosing tubercle, 223
- Tuberculosis, acute miliary, cause and dissemination of, 362
- caused by specific bacilli, 217
 - climate and season as influencing spread of, 361
 - communicability of, 215
 - defective ventilation as influencing, 368
 - effects of injection of tuberculin upon, 222
 - evidences of infectiousness of, 365
 - forms of, 360
 - heredity of, 215, 368
 - history and distribution of, 360
 - infection with, through milk, 365
 - mortality from, 360
 - of intestine in children, due to diseased milk, 215
 - — the skin, different from lupus, 221
 - overcrowding as influencing, 368
 - propagation of, through food, 365
 - race, sex, and age as influencing liability to, 362
 - relation between, and various occupations, 364
 - — of, to other diseases, 366
 - bovine, 212
 - — appearances in, 218
 - — as related to tubercle in man, 214
 - — frequency of, 214
 - — great virulence of, 214, 222
 - — inoculation of animals with matter of, 218
 - — — man with matter of, 215
 - — question as to dangerous character of carcass in, 216
 - — transmission of, to calves through milk, 218
 - — in fowls, 214
- Tyne Floating Hospital, description of, 589
- Typhoid, bacillus of, cultivation on gelatine, plate xiii. fig. 85
- — — — gelatine, showing flagella, plate iii. fig. 20

TYP

- Typhoid, bacillus of (EBERTH - GAFKY), colonies on gelatine, plate xxxiii. fig. 59
- — — streak-culture, plate xxvii. fig. 9
 - or enteric fever, air of sewers and drains as cause of, 320
 - alleged microbe of, 168, 652
 - appearances in, 165
 - cause and dissemination of, 318
 - death-rate of, among troops in India, 653
 - defective sewers in causation of, 320
 - *de novo* origin of, 325
 - disinfection of contaminated articles in, 655
 - dissemination of, by water, 167, 321, 655
 - distinction of, from typhus, 314
 - etiology of, 166
 - fatality of, 317
 - ground-water as affecting spread of, 167
 - growth of organism of, in soil, 320
 - history and distribution of, 313
 - immunity from, acquired by residence, 654
 - in tropical countries, predisposing causes of, 653
 - influence of race, sex, and age on liability to, 317
 - influence of season on prevalence of, 315
 - intermittent water-supply in causation of, 323
 - investigation of outbreak of, by Medical Officers of Health, 806
 - measures for prevention of, 654
 - mesenteric glands in, 165
 - milk as a vehicle of dissemination of, 167, 324
 - — of diseased cows as a possible cause of, 324
 - mortality of, 316
 - nature of virus of, 167
 - not communicable to lower animals, 168
 - period of incubation of, 318
 - Peyer's glands in, 165
 - possible modes of dissemination of, 320, 655
 - precautions against, in camps in tropics, 655
 - prevalence of, in hot season, in India, 654
 - protection from, 318
 - question as to importance of certain bacilli in, 169
 - — — infectiousness of, 319
 - recent epidemics of, traced to water, 321
 - reduction of mortality from, 316
 - relation of, to malaria, 325
 - spleen in, 166
 - water in dissemination of, 167, 321, 655
- Typhus, cause and dissemination of, 277
- fatality and mortality of, 275
 - history and distribution of, 274
 - influence of age, race, and sex on liability to, 276
 - — — climate and season on prevalence of, 276
 - overcrowding and poverty as factors in, 277

TYP

- Typhus, periods of incubation and infectiveness of, 278
- protection from, 278

ULCERATIVE ENDOCARDITIS, micrococci in, 92
 — — section through valve, plate xviii. figs. 124, 125

Ulcers of skin and mucous membrane, bacteria in, 48

Unhealthy areas and houses, appearances of dwellers in, 759

— — — excessive mortality of, 760
 — — — inspection of, by Medical Officers of Health, 756

United States Navy, regulation kit for, 573

Unsound meat, duties of Medical Officers of Health with regard to, 778

Unvaccinated, age-incidence of small-pox in, 436

Urban sanitary districts, formation of, 784

VACCINAL PROTECTION, conclusions as to duration of, 453

Vaccinated, age-incidence of small-pox in the, 435

— percentages of, in England and Scotland, 432

— resistance of the, to small-pox, 454

Vaccination, alleged risks of, 457

— answers to objections against, 455

— arm to arm, desirability of, 428

— cautions as to lymph used in, 428

— early testimony as to effects of, 419

— effects of Acts of Parliament (1871) regarding, 431

— — — as compared with variolation, 419

— efficacy of, in preventing small-pox, 419

— erysipelas as a result of, 460

— evidence as to value of, 433

— experiments with, by Drs. PEARSON and WOODVILLE, 418

— explanation of severe symptoms following, 422

— followed by variola, 418

— good and bad, tables showing results of, 454

— grounds of opposition to, 455

— illustrations of influence of, 452, 456

— in Germany, laws regulating, 402

— infantile, permanent results of, 455

— inflamed sores of, in early experiments, 422

— influence of multiple marks of, 446

— JENNER's first experiments with, 416

— lessening influence of, in course of time, 453

— marks of, in confluent small-pox, 456

— mitigation of small-pox by, 421

— modification of small-pox by, 445

— operation of, 427

— prevalence of, in England, 431

— primary, phenomena of, 430

— protection by, 416

— question of conveyance of leprosy by, 459

— registration of, 428

— small-pox after, 420

— storage of lymph for, 428

VOI

Vaccination, syphilis propagated by, 457

— use of calf-lymph for, 429

— value of, 432

— — — as shown by age-incidence of small-pox, 435

— — — — — decline in mortality, 433

Vaccinators, public, instructions to, 427

Vaccine lymph, erysipelas produced by, 144

Vaccinia and variola, relation between, 426

— human, by inoculation from calf, 140

— microbes of, 143

Variola, derivation of, 390

— human, crust stage of, 138

— — — papular and vesicular stages of, 137

— — — pathology of, 136

— — — section through, showing loculi, plate v. fig. 29

— ovina, 139

Variole vaccinæ, 139

Variolation, effects of, as compared with vaccination, 419

— local, 414

Variolous virus, artificial attenuation of, 413

— — — inoculation of cows with, 423

Vaults and brick graves, dangers of, 722

— under churches, massing of bodies in, 699

Veal, choleraic diarrhoea caused by, 189

Veneral diseases among troops, 677

— — — in Calcutta, frequency of, 670

— — — increased frequency of, in India, 669

— — — recent diminished frequency of, 668

Ventilation, defective, relation of, to phthisis, 363

— of ships, 535

Vesicles, vaccine, proper number of, 427

VILLEMIN on communicability of tuberculosis, 210

VIRCHOW on heredity of tubercle, 215

— on tuberculosis, 208

Vital statistics, 467

— — — construction of tables of, 468

— — — defects in the, furnished by census, 468

— — — — — Registrar-General's, 476

— — — — — graphic representations of, 480

— — — — — methods of using, 488

— — — — — of density of population and rate of mortality, 502

— — — — — distribution of death and disease, 498

— — — — — influence of atmospheric changes on mortality, 504

— — — — — birth-rate on mortality, 501

— — — — — occupation on mortality, 506

— — — — — measurements, 479

— — — — — mortality and reproduction, 473

— — — — — as affected by age, 500

— — — — — number of population, 467

— — — — — preventable mortality, 499

— — — — — sickness, 477

— — — — — practical applications of, by Health Officers, 508

— — — — — Registrar-General's methods of, 492

— — — — — returns of, 474

— — — — — sources of fallacy in, 485

— — — — — standards of comparison of, 491

VOIGT on inoculation of calves with small-pox matter, 143

VON

- VON EMMERICH** on inconstancy of phagocytosis, 53
- Voyages**, sanitary questions with regard to, 519
- WARSAWA** method of staining bacilli, 32
- Water**, analysis of, not the duty of Medical Officers of Health, 769
- conveyance of cholera by, 335
 - — — malaria by, 350
 - — — typhoid by, 167, 321, 653
 - examination of bacteria in, 62
 - filtration of, 629
 - from Grand Junction Water Company, plate-cultivation showing colonies, plate xxvi. fig. 6
 - West Middlesex Water Company, plate-cultivation showing colonies, plate xxvi. fig. 5
 - investigation of, by Medical Officer of Health in outbreak of typhoid, 807
 - multiplication of bacteria in, 58
 - pollution of, by modern method of burial, 701
 - purification of, 628
 - supplied on board ship, 568
 - supply, duties of Medical Officers of Health with regard to, 767
- Waterproofing** of boots, 634
- WEBB'S** plan of ventilating ships, 539
- WEIGERT-KOCH'S** method of staining bacilli, 29
- WEIGERT'S** method of staining sections of tissue, 33
- Wells**, pollution of, by graveyards, 701
- WELSH, Dr.**, on ophthalmia among troops at Malta, 666
- Western Hemisphere**, ravages of small-pox in, 391
- WHITELEGGE, Dr.**, on croup and diphtheria, 368
- — — mortality from scarlet fever, 270
 - — — 'superadded waves' of epidemic diseases, 257
- Whooping-cough**, case-mortality of, 289
- cause and dissemination of, 291
 - history and distribution of, 289
 - influence of season, age, and sex on, 290
 - mortality of, 289
 - periods of incubation and infectiveness of, 291
 - relations of, to other diseases, 291
- WILKS, Dr.**, on value of vaccination, 482
- WILLAN** on antiquity of small-pox, 385
- — vaccine inoculation, 414
- WILTSCHOND** on bacilli of typhoid, 170

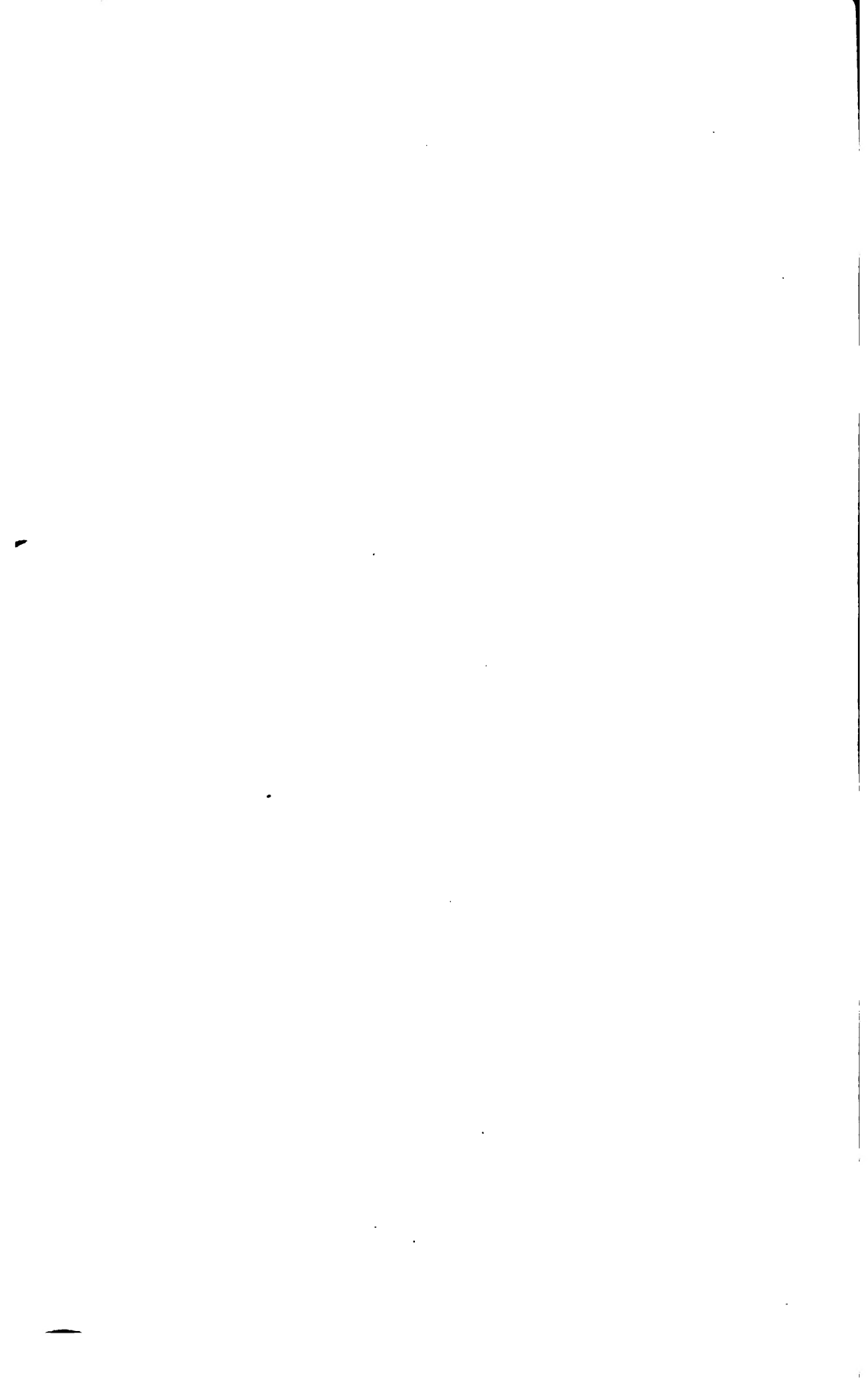
YOR

- Woking**, cremation at, 715
- description of burial arrangements at, 726
- Wood** used in shipbuilding, decay and preservation of, 524
- WOODHEAD, Dr. Sims**, on anaërobic and aerobic growth of cholera organisms, 336
- — — modification of functions of microphytes, 249
 - — — — tabes mesenterica caused by diseased milk, 215
- WOODVILLE, Dr.**, experiments by, with cow-pox lymph, 418
- WOOLDRIDGE, Dr.**, on production of immunity by injection of toxins, 56
- — — protection by vaccines against anthrax, 121
- Woolsorters**, anthrax of, 113
- Working-class** lodging houses, inspection of, by Medical Officer of Health, 759
- Worms** in mould, diseased germs conveyed by, 703
- WYSSOKOVITCH** on behaviour of bacteria introduced into blood, 48
- XEROBIS CONJUNCTIVÆ**, bacillus of, 133
- YELLOW FEVER** among soldiers, 856
- — and plague, precautions against invasion of, 587
 - — cause and mode of dissemination of, 377
 - — climate and season as controlling, 375
 - — communicability of, 378
 - — conditions favouring outbreaks of 657
 - — — — prevalence of, 380
 - — distribution of, influenced by acclimatization and race, 656
 - — endemic centres of, 373
 - — epidemics of, on shipboard, 379
 - — fatality of, 376
 - — history and distribution of, 373
 - — infection of localities by virus of, 378
 - — measures for prevention of, 657
 - — origin of, 658
 - — peculiarities in spread of, 379
 - — period of incubation of, 381
 - — protection from, 376
 - — race, sex, and age as influencing liability to, 376
- Yorktown and Camberley**, epidemic of diphtheria at, 161

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